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# DAMAGE BOOK

# ENCYCLOPEDIA AND DICTIONARY

OF

# MEDICINE AND SURGERY

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FOR

WILLIAM GREEN & SONS.

AUGUST 1907.

# GREEN'S ENCYCLOPEDIA AND DICTIONARY OF MEDICINE AND SURGERY

VOL: V

INULIN TO LUMBAR-PUNCTURE

WILLIAM GREEN & SONS
EDINBURGH & LONDON

# EDITORIAL NOTE

THIS, the fifth, volume of the *Encyclopedia and Dictionary of Medicine and Surgery* carries the work from INULIN to LUMBAR PUNCTURE, and contains 908 subject-headings. It also completes the first half of the whole work. The outstanding article, containing seventeen subdivisions by some twelve or thirteen authors, is that on LABOUR; and there is also a series of papers dealing with the LARYNX and its Diseases.

Of articles of more than 1000 words there are forty-seven. These include the various subdivisions of the two large groups headed *Labour* and *Larynx* respectively, as well as contributions on *Invalid Feeding*, *Iris and Ciliary Body*, *Jaundice*, *Diseases of Joints*, the *Physiology and Surgical Affections of the Kidney*, *Diseases and Injuries of the Knee-Joint*, *Diseases of the Lacrimal Apparatus*, *Lardaceous Degeneration*, the *Crystalline Lens*, *Leprosy*, *Leucocythæmia*, *Leucocytosis*, *Lichen*, *Life Insurance*, the *Physiology and Diseases of the Liver*, and *Lumbago*. New articles have been added dealing with *Cleidotomy*, *Iodine and the Iodides*, and the *X-Rays in Leukæmia*.

There are sixty-nine articles of less than 1000 words but of at least ten lines in length. These include contributions on such subjects as *Inulin*, *Inunction*, *Inversion*, *Iodism*, *Iodoform*, *Ionic Action*, *Ipecacuanha*, *Iritis*, *Iron*, *Irrigation*, *Irritants*, *Isomer and Isomeric*, *Isotonic*, *Jaborandi*, *Jacksonian Epilepsy*, *Jalap*, *Jaws*, *Juniper*, *Kaladana*, *Kava*, *Kephir*, *Keratin*, *Keratitis*, *Keratosis*, *Kino*, *Kjeldahl's Method*, *Knee-Joint*, *Krameriæ Radix*, *Labia*, *Lactic Acid*, *Lagophthalmos*, *Lanoline*, *Laparotomy*, *Laryngismus Stridulus*, *Latah*, *Latent*, *Laundries*, *Laurocerasi Folia*, *Lavage*, *Lavender*, *Lead*, *Lecithin*, *Leeches*, *Lembert Suture*, *Lemon*, *Leontiasis Ossea*, *Leptothrix*, *Lesion*, *Leucine*, *L.G.B.*, *Ligaments*, *Linseed*, *Lipoma*, *Liquor*, *Liquorice*, *Lithium*, *Lixiviation*, *Lochia*, *Lodging-Houses*, and *Lopez Root*.

There are 792 subject-headings and short paragraphs dealing with a great variety of matters. In order to show their diverse character, I may name a few. There are definitions or short explanations of *Inversion*, *Invertins*, *Involution*, *Ioderma*, *Iodophilia*, *Iodothyrin*, *Iridectomy*, *Irideremia*, *Iridocinesis*, *Iridodesis*, *Iridodialysis*, *Iridodonesis*, *Iridoplegia*, *Iridotomy*, *Irrespirable Gases*, *Isadelphia*, *Ischæmia*, *Ischiadelphia*, *Ischialgia*, *Ischiocele*, *Ischuria*, *Isobars*, *Isogens*, *Isotherms*, *Isotropy*, *Ispaghula*, *Jacquemier's Test*, *Jactitation*, *Janiceps*, *Jaw-Clonus*, *Jecorin*, *Jejunostomy*, *Jensen's Pump*, *Jensen's Tumour*, *Jigger*, *Jiu-Jitsu*, *Jumpers*, *Justo-Major*, *Justo-Minor*, *Kahler's Disease*, *Kala-azar*, *Kalmuck Idiocy*, *Kaposi's Disease*, *Kataphoresis*, *Kehrer's Operation*, *Keratolysis*, *Keratoma*, *Kernig's Sign*, *Ketones*, *Kiestine*, *King's Evil*, *Kirrhonosis*, *Klikuschi*, *Knacker*, *Kneippism*, *Kocher's Operation*, *Koplik's Spots*, *Korsakoff's Syndrome*, *Kraurosis*, *Kromskop*, *Kutubuth*, *Labitome*,

*Labyrinth*, *Lac*, *Lacing-Liver*, *Lacs*, *Lactams*, *Lacteals*, *Lactuca*, *Læhme*, *Lagnesis*, *Lahs' Theory*, *Laloo*, *Lambdacism*, *Laminaria Tents*, *Langhans' Layer*, *Lanugo*, *Lapis*, *Larval*, *Laterad*, *Lathyrism*, *Latrines*, *Laudable*, *Laveran's Bodies*, *Layman*, *Leather Dressing*, *Legal's Disease*, *Legitimacy*, *Leiter's Coil*, *Lemniscus*, *Lentigo*, *Leontiasis Hystrix*, *Le Pita*, *Leptocephaly*, *Lerema*, *Leschenoma*, *Lesser's Triangle*, *Leste*, *Leucoblasts*, *Leucoma*, *Leucomaines*, *Levurine*, *Lie*, *Liernur System*, *Ligar's Line*, *Liman Cure*, *Lime Test*, *Limosis*, *Liouville's Icterus*, *Lipase*, *Lipuria*, *Lisfranc's Amputation*, *Listerism*, *Little's Disease*, "*Living Skeletons*," *Lobengulism*, *Loco Disease*, *Loreta's Operation*, and *Lues*.

It will be evident, therefore, from these details, that the *Encyclopedia and Dictionary* continues to show in this volume the characteristics of the earlier ones. The purely Dictionarial part endeavours, without striving after the degree of copiousness attained by some works which are Dictionaries only, to give the meaning of every medical term which is at all likely to puzzle the general practitioner in his reading of current medical and surgical literature; while the part which constitutes the Encyclopedia consists of articles which are authoritative, coming as they do from the pens of men who are experts in their various departments.

J. W. BALLANTYNE.

*July* 8, 1907.

# CONTENTS

## THE CHIEF ARTICLES IN VOLUME V., WITH THEIR AUTHORS


| | PAGE |
|---|---|
| Invalid Feeding. Mrs Chalmers Watson, M.D. | 1 |
| Iris and Ciliary Body. H. E. Juler, F.R.C.S. | 36 |
| Jaundice. H. D. Rolleston, M.D., F.R.C.P. | 51 |
| Joints, Diseases of. Alexis Thomson, F.R.C.S.E. | 62 |
| Kidney | 89 |
| Physiology of. T. H. Milroy, M.D. | 89 |
| Surgical Affections of. E. Hurry Fenwick, F.R.C.S. | 93 |
| Knee-Joint | 127 |
| Diseases of. Alexis Thomson, F.R.C.S.E. | 127 |
| Injuries of. A. E. J. Barker, F.R.C.S. Eng., F.R.C.S.I.; and E. G. Leopold Goffé, F.R.C.S. | 142 |
| Labour | 154 |
| A. Physiology of. F. W. N. Haultain, M.D., F.R.C.P.E. | 154 |
| Duration and Progress. H. Jellett, M.D., F.R.C.P.I. | 163 |
| Diagnosis and Mechanism. W. R. Dakin, M.D., F.R.C.P. | 168 |
| Management of. Mrs Chalmers Watson, M.D. | 189 |
| Labour in Multiple Pregnancy. W. Stephenson, M.D. | 204 |
| B. Precipitate and Prolonged Labour. G. E. Herman, M.D., F.R.C.P. | 207 |
| Faults in the Passenger. W. E. Fothergill, M.D., B.Sc. | 239 |
| Accidental Complications. W. W. H. Tate, M.D., F.R.C.P. | 246 |
| Retained Placenta, etc. T. W. Eden, M.D., F.R.C.P. | 252 |
| Post-partum Hæmorrhage. H. Jellett, M.D., F.R.C.P.I. | 256 |
| Injuries during Labour. Amand Routh, M.D., F.R.C.P. | 264 |
| C. Operations | 276 |
| Induction of Premature Labour. W. E. Fothergill, M.D., B.Sc. | 276 |
| Forceps. Sir Alex. R. Simpson, M.D., F.R.C.P.E. | 281 |
| Version. F. W. N. Haultain, M.D., F.R.C.P.E. | 287 |
| Embryotomy, etc. Sir Alex. R. Simpson, M.D., F.R.C.P.E. | 290 |
| Cleidotomy. J. W. Ballantyne, M.D., F.R.C.P.E. | 295 |
| Cæsarean Section. Murdoch Cameron, M.D. | 299 |

PAGE

LACRIMAL APPARATUS. W. Hill Griffiths, F.R.C.S. . . . . 306

LARDACEOUS DEGENERATION. Montagu Murray, M.D., F.R.C.P.; and W. C. Bosanquet, M.D., F.R.C.P. . . . . 315

LARYNX . . . . . 320

EXAMINATION OF. J. M. Hunt, M.B. . . . . 320

ACUTE AND CHRONIC INFLAMMATION, ETC. St. Clair Thomson, M.D., F.R.C.S., F.R.C.P. 324

CHRONIC INFECTIVE DISEASES. P. M'Bride, M.D., F.R.C.P.E. . . . 341

NEOPLASMS, SIMPLE. J. M. Hunt, M.B. . . . . 347

NEOPLASMS, MALIGNANT. Sir Felix Semon, K.C.V.O., M.D., F.R.C.P. . . 351

NEUROSES OF. P. Watson Williams, M.D. . . . . 362

AFFECTIONS OF CARTILAGE, ETC. Logan Turner, M.D., F.R.C.S.E. . . . 369

CONGENITAL LARYNGEAL STRIDOR AND LARYNGISMUS STRIDULUS. John Thomson, M.D., F.R.C.P.E. . . . . 372

LENS (CRYSTALLINE). G. A. Berry, M.B., F.R.C.S.E. . . . . 370

LEPROSY. G. Thin, M.D. . . . . 382

LEUCOCYTHÆMIA. G. Lovell Gulland, M.D., F.R.C.P.E. . . . . 392

LEUCOCYTOSIS. R. Muir, M.D., F.R.C.P.E. . . . . 400

LICHEN. W. Allan Jamieson, M.D., F.R.C.P.E. . . . . 406

LIFE INSURANCE. W. Elder, M.D., F.R.C.P.E. . . . . 411

LIVER . . . . . 427

PHYSIOLOGY OF. D. Noel Paton, M.D., F.R.C.P.E. . . . . 427

DISEASES OF (other than "Tropical" disorders). H. D. Rolleston, M.D., F.R.C.P. . 431

TROPICAL AFFECTIONS, ETC. J. Cantlie, F.R.C.S. . . . . 488

LUMBAGO. D. Chalmers Watson, M.D., F.R.C.P.E. . . . . 527

# LIST OF ILLUSTRATIONS


PAGE

Iritis (*Plate*) . . . . . *Facing* 40
Coloboma.
Iritis with adhesions.
Syphilitic Iritis.

Iris and Ciliary Body—
Fig. 1. The Incision with Keratome . . . . . 45
„ 2. The Iridectomy . . . . . 45
„ 3. Optical Iridectomy . . . . . 45

Fracture of Patella (*Plate*) . . . . . *Facing* 146

Labour (Physiology of)—
Fig. 1. Diagram of Full-time Gravid Uterus, showing main disposition of Fibres . 156
„ 2. Full-time Gravid Uterus of Primipara, showing cervix closed . . . 157
„ 3. Commencing dilatation of Cervix before the actual onset of labour pains . 157
„ 4. Commencement of First Stage with early dilatation of Os externum . . 157
„ 5. Gravid Uterus towards end of the first stage . . . . . 157
„ 6. Commencing Labour, showing complete attitude of fœtal flexion . . 158
„ 7. Labour, Second Stage, showing commencing straightening of fœtus . . 159
„ 8. Birth of Head, showing extension of fœtal ovoid . . . . 159
„ 9. Vertical Mesial Section of Bony Pelvis, showing planes at which measurements are taken . . . . . 160
„ 10. Diagrams of Fœtal Head, showing sutures and diameters . . . 161
„ 11. Diagrammatic representation of the manner in which the cervix is taken up in the case of a *primipara* . . . . . 165
„ 12. Diagrammatic representation of the manner in which the cervix is taken up in the case of a *multipara* . . . . . 165
„ 13. Before the expulsion of the placenta (diagrammatic) . . . . . 167
„ 14. After „ „ „ . . . . . 168
„ 15. Graphic representation of parts felt on abdominal palpation in the case of cephalic lie . . . . . 169
„ 16, 17. Inspection and Palpation . . . . . 170
„ 18, 19. „ „ . . . . . 171
„ 20. Uterus at term . . . . . 174
„ 21. Effect of Inward Pressure of walls of birth-canal on moderately flexed head . 174
„ 22. Effect of Obliquity of Uterine Axis on head at brim . . . . 175
„ 23. Fœtal-axis-pressure on slightly flexed head . . . . . 175
„ 24. Relation of Head to Finger when occiput lies to the left . . . 176
„ 25. Relations of Child to Pelvis during delivery of shoulders . . . 177
„ 26. First Vertex. Pelvis of woman lying on her left side . . . . 179
„ 27. Third Vertex of same pelvis . . . . . 179
„ 28. Persistent Occipito-posterior position . . . . . 179
„ 29. Rarer form of Mechanism in persistent Occipito-posterior cases . . . 179
„ 30. Relation of Head to Trunk in Face Presentation . . . . 180
„ 31. Mechanism in First Face Presentation . . . . . 181
„ 32. Vertex. Brow and Face . . . . . 183
„ 33. Relations of Head and Pelvis in Brow Mechanism . . . . 184
„ 34. Schatz's Method of converting Face Presentations into Vertex Presentations . 199
„ 35. The Prague Method of extracting the head . . . . . 201
„ 36. Method of Jaw and Shoulder Traction for delivery of the head . . . 202

PAGE

Labour (Pathological Section—Precipitate and Prolonged Labour)—
Fig. 1. Diagram showing thickening of the upper part of the Uterus . . . 211
„ 2. Showing thinning of lower Uterine Segment . . . . . 211
„ 3. Ruptured Uterus, showing retraction ring at level of firm attachment of peritoneum . . . . . 212
„ 4. Pelvis of Fœtus at term . . . . . 213
„ 5. Pelvis of Adult . . . . . 213
„ 6. Sagittal Section of normal pelvis . . . . . 213
„ 7. „ flat „ . . . . . 214
„ 8. Diagram of the generally contracted flat non-rickety pelvis . . . 214
„ 9. Diagram of Pelvic Cavity of generally contracted flat non-rickety pelvis . 214
„ 10. Sagittal Section of small round pelvis . . . . . 215
„ 11. Diagram of the Brim of the same round pelvis . . . . . 215
„ 12. Diagram of the Cavity of the small round pelvis . . . . 215
„ 13. Sagittal Section of flat rickety pelvis . . . . . 215
„ 14. Diagram of rickety flat pelvis . . . . . 215
„ 15. Rickety flat pelvis . . . . . 216
„ 16. Diagram of Brim of small flat rickety pelvis . . . . . 216
„ 17. Diagram of Pelvic Cavity in small flat rickety pelvis . . . . 216
„ 18. Scolio-rachitic Pelvis . . . . . 216
„ 19. Diagram of Brim of scolio-rachitic pelvis . . . . . 216
„ 20. „ Cavity „ „ . . . . . 217
„ 21. Position in which the Head enters the Brim of the flat pelvis . . . 218
„ 22. Obliquity of Naegelé; the sagittal suture near the sacral promontory . 218
„ 23. Showing Mark made by Promontory in delivery of the after-coming head . 219
„ 24. Showing Change in Shape of Head produced by traction with base in advance . 219
„ 25. Pressure Marks on Head after a labour with flat pelvis . . . 219
„ 26. „ „ „ small round pelvis . . 220
„ 27. Diagram of Cavity of Funnel-shaped Pelvis in sagittal plane . . . 221
„ 28. „ „ „ „ coronal plane . . . 221
„ 29. Obliquely contracted Pelvis of Naegelé . . . . . 221
„ 30. Diagram of the brim of fig. 29 . . . . . 221
„ 31. Transversely contracted Pelvis of Robert . . . . . 222
„ 32. Diagram illustrating the production of Kyphotic Pelvis . . . 222
„ 33. Kyphotic Pelvis . . . . . 223
„ 34. Diagram of Cavity of Kyphotic Pelvis in sagittal plane . . . 223
„ 35. „ Brim „ „ „ . . . 223
„ 36. Kypho-scolio-rachitic Pelvis . . . . . 224
„ 37. Diagram of brim of fig. 34 . . . . . 224
„ 38. „ cavity of fig. 36 . . . . . 224
„ 39. Osteomalacic Pelvis . . . . . 225
„ 40. Pseudo-osteomalacic Rickety Pelvis . . . . . 225
„ 41. A Lumbar Vertebra showing the defect in ossification upon which Spondylolisthesis depends . . . . . 226
„ 42. Spondylolisthesis . . . . . 226
„ 43. Early Stages of Spondylolisthesis . . . . . 227
„ 44. Split Pelvis . . . . . 228
„ 45. Duncan's Callipers . . . . . 229
„ 46. Direct Pelvimetry, measurement 4 inches . . . . . 230
„ 47. „ „ 3½ „ . . . . . 230
„ 48. „ „ 3¼ „ . . . . . 230
„ 49. „ „ 3 „ . . . . . 230
„ 50. „ „ 2½ „ . . . . . 231
„ 51. Mode of measuring the Diagonal Conjugate . . . . . 231
„ 52. Change in Shape of Head produced by downward pressure with vertex in advance . . . . . 235
„ 53. Ovarian Tumour obstructing delivery . . . . . 237
„ 54. Sacral Exostosis . . . . . 238
„ 55. Cancerous Growths from pelvic bones . . . . . 238
„ 56. Morbid adhesion of the Membranes . . . . . 255
„ 57. Method of Detachment . . . . . 255
„ 58. Rectangular Needle . . . . . 268

PAGE

Labour—*continued.*
Fig. 59. Laceration of the Pelvic Floor extending half-way to the rectum . . 269
„ 60. Sagittal Section of the Perineal Body showing its component structures . 270
„ 61. Rectangular Needle . . . . . 272
„ 62, 63. Diagrams showing the faulty and the correct methods of inserting sutures for the repair of incomplete ruptures . . . . . 273

Labour: Forceps—
Fig. 1. Chamberlen's Forceps . . . . . 282
„ 2. Chapman's Forceps with handles ending in hooks . . . . 282
„ 3. Smellie's Wooden Forceps . . . . . 283
„ 4. Diagram showing Forceps with pelvic curve . . . . . 284
„ 5. Tarnier's Forceps with curved rods . . . . . 284
„ 6. Diagram illustrating Tarnier's Method . . . . . 284

Labour: Version—
Fig. 1. External Version (Schäffer) . . . . . 287
„ 2. Bipolar Cephalic Version (Schäffer modified) . . . . . 288
„ 3. „ Podalic „ (after Schäffer) . . . . . 288
„ 4. „ „ „ (Schäffer modified) . . . . . 288
„ 5. Internal Version, 1st stage (after Schäffer) . . . . . 289
„ 6. „ 2nd stage „ . . . . . 289
„ 7. „ in impacted case assisted by loop (after Schäffer) . . 290

Labour: Embryotomy—
Fig. 1. Smellie's Scissors for perforating the skull . . . . . 291
„ 2. J. Y. Simpson's Perforator . . . . . 291
„ 3. Lyon's Craniotomy Forceps . . . . . 292
„ 4. Carl Braun's Cranioclast . . . . . 292
„ 5. The Basilyst—partially opened and traction blade separated . . 293
„ 6. „ with traction blade adapted . . . . . 293

Labour: Cæsarean Section—
Fig. 1, 2. Length of Abdominal Incisions . . . . . 302
„ 3, 4. Method of placing flat vulcanite Pessary upon the Uterine Wall . . 303
„ 5, 6. Method of grasping Uterus and insertion of Silk Ligatures . . . 303

# ENCYCLOPEDIA AND DICTIONARY OF MEDICINE AND SURGERY

**Inulin.**—A carbohydrate ($6C_6H_{10}O_5,H_2O$) closely allied to dextrin; differing from it, however, in containing lævulose instead of dextrose molecules; it is a white powder and is contained in elecampane (*Inula helenium*), colchicum, arnica, dandelion, etc.; iodine turns it yellow, and it is converted into sugar by its own special ferment and not by diastase. *See* PHYSIOLOGY; FOOD AND DIGESTION (*Food, Carbohydrates*).

**Inunction.** *See also* PHARMACOLOGY; SYPHILIS (*Treatment of Secondary Syphilis by Mercury*).—Inunction consists in the application of a medicinal substance to the skin in such a manner as will lead to its absorption. Various diseases, both of a local and general nature, may be treated by this method. Of these, perhaps the best illustrations may be found in the use of mercury for syphilis, cod-liver oil or other fatty substance in cases of markedly impaired nutrition, and olive or other oil in certain cases of chronic rheumatism. The beneficial effects accruing from such use of oils are not sufficiently appreciated. In the cases that are materially improved by this treatment the benefit is probably explained by the influence of the oil on the local, and it may be general derangement of fat metabolism in the body. No matter what the nature of the case may be there are certain elementary points to be attended to. These can be stated shortly:—(i.) A preliminary stimulation and softening of the skin by heat in the form of hot water immersion is of great service in furthering the efficient absorption of the drug.

(ii.) The oily fluid or the ointment should be thoroughly rubbed into the selected part at bedtime, the friction being mainly from below upwards, and continued for ten to twenty minutes.

(iii.) Thereafter the limb or other part of the body should be covered with a layer of flannel more or less impregnated with the substance, and left in contact over night. The parts are thoroughly washed in the morning, and the treatment continued at the discretion of the physician.

Lately additional interest has been given to this subject by the strong expression of opinion by various writers to the effect, that, so far as mercury is concerned, the system becomes impregnated with it, not by a process of cutaneous absorption, as has long been held, but by a process of volatilisation. Recent clinical experience, it may be added, lends a good deal in support of this belief. The methods recently introduced consist in applying to the surface of the chest, as a kind of under vest, a specially prepared cloth containing a mercurial preparation in its interior. The advantages of this manner of administration from a point of view of cleanliness are apparent, and if a more extended experience confirms its efficiency, the older system of inunction will probably give place entirely to it.

**Invagination.** *See* INTESTINES, SURGICAL AFFECTIONS OF (*Intestinal Obstruction, Intussusception*).

## Invalid Feeding.


INTRODUCTORY . . . . . 2
GENERAL PREPARATION OF FOOD . . 2
(*a*) *When Juices are Retained—Roasting, Grilling, Baking, Boiling, and Frying* . . . 3
(*b*) *When Juices are Extracted—Stewing, Braising, and Soup-making* . . . . . 4
GENERAL SERVING OF FOODS—*Including Nasal and Rectal Feeding* . 5
MILK AND ITS PRODUCTS—*Whey, Koumiss, etc.* . . . 6, 7, 8
EGGS . . . . . 8

PREPARED AND PREDIGESTED FOODS . . 9
PEPTONISED FOODS . . . . . 10
FOOD IN PYREXIAL STATES . . . 12
*Beverages, Meat Teas, Extracts, Juices* . . . . . 12, 13, 14
FOOD IN CONVALESCENCE . . . 14
*Milk Puddings, Fish, Soups, Meats, Vegetables* . . . . . 15–23
FOOD IN OLD AGE . . . . . 23
*Soups, Fish, Vegetables, Fruits* . 23–26
SPECIAL COOKERY FOR DIABETES . . 26
SALISBURY DIET . . . . . 31

*See also* DIET; DIGESTION; DIABETES MELLITUS; FOOD; GASTRO-INTESTINAL DISORDERS OF INFANCY; GOUT; INFANT FEEDING; etc.

INTRODUCTORY.—The nutritive constituents of food, their relative values, and the factors which modify individual requirements in health, have already been considered in the article on "Diet" (vol. ii.). In disease, as in health, we must have regard to the functions of food, as *building material*, *fuel*, and *suppliers of energy*; and we have here also to consider the modifying influence of age and sex, height and build, and the amount of work or rest to be indulged in, and any personal peculiarities in the requirements of each individual case. But, above all, we must remember that if the palate, or the sense of sight or smell be offended, no matter what the nutritive value of a food may be, the food is valueless; and as the most frequent cause of offence lies in a want of knowledge of certain practical details in the manner and preparation of foods, the scope of this article will be mainly limited to noting these points. Every practitioner should possess some knowledge of cookery. It is idle for the practitioner to recommend certain food-stuffs without taking all necessary precautions to ensure that the manner of their preparation is suited to the palate as well as to the physiological capacity of the patient. Yet such recommendations are by no means rarely made. While in many houses where there is a thoroughly competent housewife or a trained nurse, any directions regarding the manner of preparation of egg flip, sweetbread, or the cooking of an oyster, may be superfluous, other cases are only too numerous where, if the practitioner has not the practical knowledge to enable him to give his instructions in detail, the patient suffers in consequence. To illustrate: The following instructions for an egg flip are frequently given:—"Beat up an egg; add a little milk, a little sugar, and a stated quantity of brandy." What is the result? For lack of the requisite straining of the mixture the second or third mouthful taken by the patient may include the streaky, tenacious portion known as the trend, which is to many people so nauseating that no more egg flip will be taken at that time or for many a day thereafter. And in the same way with many other dishes.

GENERAL PREPARATION OF FOOD.—Cooking increases the digestibility of food-stuffs by exercising a mechanical, chemical, and anti-parasitic effect on the food, and it is of great importance in ordering a diet for an invalid that the physician should understand at least the main principles by which the food is prepared.

The mechanical action is a twofold one: the prolonged heat acts by softening hard tissues and by preparing tough tissues for mastication. Also, as the functions of digestion and assimilation are best carried on at a blood heat, one object of cooking is to raise the food to a suitable temperature, and thus spare the bodily energies as much as possible.

The chemical changes taking place act on the meat substances by transforming the connective tissues into gelatine, the proteids of the meat at the same time developing odorous substances which impart flavour and stimulate the appetite. The starch granules are broken up and prepared for the action of the saliva, and the cooking converts some of the starch into dextrine and even into maltose. These chemical changes can be further carried on by the addition of some of the pancreatic ferments, which transform the albuminous substances into peptones, and also act on the starches, and carry on the further transformation of the starches into soluble sugars. These more complicated changes are further considered under the Predigested and Prepared Food section (see p. 9).

Not the least important action of cooking is the destruction of the parasites and pathogenic bacteria of disease that may occur in the food.

The effect of different degrees of heat on albumin is the keynote to all the various culinary methods, which are variations of one another depending on whether the meat juices are to be retained or extracted.

(*a*) When the meat juices are to be *retained* there is, first, a preliminary case hardening of the albumin by intense heat; after which, cooking proceeds at a lower temperature, rising in the interior of the meat just to the coagulating point of albumin. This object may be carried out by any of the following methods:—

(i.) Roasting, Grilling, Baking.—The heat being radiant heat and hot gases.

(ii.) Steaming and Boiling.—In this the heat is hot water.

(iii.) Frying, Wet or Dry.—This process is carried on by hot oil or hot oil and conduction.

By following these methods we retain all the nutritive, sapid qualities within the portion, leaving none of them, or very little, in the medium used.

(*b*) If the juices are to be *extracted* from the meat the result is a stew or a soup.

Stewing first extracts the juice and then cooks the meat in it, and is thus conducted slowly and over a long time.

Soup-making aims at extracting as much as possible, and therefore begins in the cold, never being above about 160° F.

These various processes will now be referred to in some detail.

A. *When the Meat Juices are Retained*

*Roasting.*—In this method of cookery the meat is cooked by the radiant heat of a fire from a large glowing surface free from smoke. The meat is exposed to this heat, within a few inches, for five to ten minutes, and is basted with melted dripping, the surface albumin being coagulated to the thickness of a sixpence. After this has taken place the meat is removed ½ to 1 ft. farther from the fire, and the cooking process is continued slowly at a greatly reduced temperature, the meat being slowly cooked in its own juices.

If the heat is too great the hardened case is apt to crack, allowing the juices to escape. To prevent this the meat is basted with hot dripping, which prevents charring and cracking of the surface. If properly done this fat does not soak into the meat. Although the surface heat is very great, the heat in the interior of the joint should not be much above the coagulating point of albumin, and any rise above this renders the meat less digestible, the albuminous matters becoming hard and horny.

The inside parts of a *properly* roasted joint are the best for a weak digestion.

This process requires a quarter of an hour per pound for beef and mutton, but longer for veal and poultry. The meat loses weight from expulsion of water.

*Baking.*—Joints of meat can be well cooked in the oven of an ordinary kitchen range or in a gas cookery, and the results are very similar to roasting. If the heat of the oven is not properly regulated the results are different, the meat being richer and more indigestible, and it has a different flavour. This is owing to the process being conducted at too high a temperature after the preliminary case hardening.

*Broiling, Grilling, or Brandering.*—This is a similar process to roasting, and is admirably adapted for quickly cooking a chop, steak, and kidney, fish, fowl, or mushroom, etc.

A clear and smokeless fire is required, and is carried on by holding the article to be cooked over the fire.

The object is the same—the formation of a surface skin of coagulated albumin, and the retention within this of all the juices of the meat. To avoid breaking this coagulated layer steak tongs should be used instead of forks for turning the meat.

**In this form of cooking the process is so rapidly carried on that there is not much time allowed for softening the food, so that, unless the meat is very tender, this process is not to be recommended.**

*Boiling and steaming* as described here consists in cooking meat, fish, or vegetables either with hot water or steam, with a view to retain in the meat all the flavours, and is the opposite of soup-making, which aims at withdrawing the meat juices. Here, again, the first step is to coagulate a surface layer of albumin by plunging the joint into a large pot of boiling water, to which has been added a handful of salt. This is done on account of the boiling-point of salt water being higher than that of fresh water. The formation of this hardened case takes about seven minutes; the pot should then be withdrawn and kept at the side of the fire, not even simmering. This method is not only much more economical, but the meat is very much more tender if the cooking is slowly conducted over a period of five or six hours. The best method of slow cooking is by steaming the article of food in its own juice; this can be done most satisfactorily in a Warren cooking pot. This is a pot consisting of three compartments connected by a steam chimney. Water is put into the lowest pot, in the second the meat and its flavourings, and in the top the vegetables. The food is thus cooked by steam; all the juices of the meat are retained; and the meat will be found to be much more succulent, tender, and digestible than if simply boiled in water. If the boiling is carried to excess the meat is "boiled to rags," but this only means that all the connective tissue has become gelatinised, and the muscle fibre remains as tough as leather, and may often be recognised unchanged in the fæces.

The boiling of vegetables aims at softening the cellulose and rendering it tender and digestible; it also opens up the starch granules, so that the saliva can get at them, and converts some of the starch into dextrine. Vegetable proteids, when in the form of legumin, are not coagulable by heat, so overcooking is not so injurious, but merely entails a waste of fuel.

Green vegetables and rice are eaten on account of the salts they contain. Therefore, unless they are steamed, or the water in which they are boiled is kept as stock for soup, the best part of their substance is thrown away. Steaming has the further advantage of preserving the shape of the vegetable or fish, and of therefore presenting it in a more appetising form.

*Frying* is boiling in oil, but as practised in this country is a dirty and wasteful method, being usually a combination of broiling and scorching. There are two forms. In *dry frying*—which is our national form of frying—a shallow frying-pan is used, and the food is either cooked in its own fat or with the aid of sufficient fat to prevent burning. **It is only suitable for fatty food like herrings and sausages, but the products of this form of frying are often greasy, and for many persons exceedingly indigestible.**

*Wet frying* or sautéing is properly boiling in oil. For this there is required a deep pan of clarified dripping or of olive oil. If the process is properly carried out the food is deliciously cooked and absolutely free from grease. The temperature of the fat is important; when fat is placed on the fire and reaches 212° F. (the boiling-point of water), it bubbles and makes a hissing sound due to a small portion of the water in the fat becoming steam and being got rid off. Fat, however, does not boil until it has reached a much higher temperature. At a temperature of 340° F. a slight bluish vapour is given off, and this is the time when if a piece of bread be dropped in, it becomes brown in a second or two, and on being taken out of the fat, almost instantly loses all its greasy appearance. If the fat gets hotter and begins to boil, a state of affairs to be avoided, it begins to smoke and decompose.

The principle on which the success of frying depends is that at the moment of contact with the almost boiling fat a thin film is formed over every part of the surface of the fish or other object to be fried, and all the juices and flavours are retained. The food is kept in until it has a golden brown colour, when it is removed from the oil bath. It may look greasy for a moment, but this drains off very quickly if put on a piece of paper near the fire, and it is absolutely free from grease when it appears at table. If the object to be fried is coated with egg and bread crumbs the crumbs should be *very* fine, and firmly pressed down to prevent the grease adhering to the surface and by being absorbed by it.

The best substance to use is second quality of olive oil, or roast beef dripping; clarified lard should never be used, as the other media for frying are as easily obtained and are much better.

### B. *When the Juices are Extracted*

*Stewing and braising* are processes by which meat and poultry can be prepared and served in the most tender condition possible.

*Stewing* aims at making an extract of the nutritive juices of the food, which are then used for cooking. A low steady heat is wanted, and since nothing is lost in this process it is one of the most economical ways of preparing food. The meat is chopped into convenient sized portions, seasoned (if there are bones they also are broken up and put into the pot), the whole is covered with cold water, and a tight-fitting lid put on to the pot, which is placed near the fire, but never allowed to boil. The old saying, "A stew boiled is a stew spoiled," is a true one. If vegetables are to be added they are best put into the pot after the meat is half done. The flavours of the vegetables are lost with too continuous cooking. The more slowly the stew is cooked the better it is done. Tough meats are rendered more digestible by the addition of a little vinegar to loosen their fibres and to convert them into acid albumoses.

*Braising* is slow cooking in a closed vessel, the meat being just covered with an extract of animal and vegetable juices instead of water, and so the flavour of the meat is improved. This method is best adapted for foods somewhat insipid in themselves, like veal and poultry. At the end of the process the meat should be browned, and this is carried out by having a concave lid in which hot cinders can be placed, or it may be done by radiation before the fire.

*Soup-making.*—This, like stewing, proceeds upon the principle of extracting as much as possible from the materials, the extraction being effected by the use of a considerable quantity of water at a very moderate heat extending over a long period.

The simplest form of soup-making is that of making beef tea (p. 13).

The meat is cut up into several pieces to expose as large a surface to the water as possible; the solvent power of the water is increased by the addition of a little vinegar, and the temperature must never rise over 160° F.

As vegetables require a very much greater heat than this to soften them, they should either be first boiled, and then the meat should be added; or the steamed vegetables should be added when the soup is almost ready. Flavouring herbs should be put in at the last moment. In all soups made from meat great care should be exercised in the removal of fat. This is best done by the soup being made the day before it is required, and poured into a basin and carefully skimmed when cold.

Varieties of Soup.—*Stock.*—This is the simplest form of soup, and is the product of the mysterious stock-pot. This vessel should receive all scraps of meat and vegetables that are over from the preparation of other dishes. It stands constantly at the side of the stove, gradually concentrating its contents, and it requires to be brought to the boil every day for five minutes to prevent fermentation. It should also be cleaned out every third or fourth day in case of the stock going sour. This stock can be used for many soups, for the basis of gravy, and, instead of water, in stewing and boiling meat.

The next variety of soup is that which is made on the lines of a good Scotch broth of meat and vegetables either boiled in weak stock or with water; this is known in France as pot-au-feu, and here the boiled meat is intended to be eaten either with or immediately after the soup.

A stronger extract than pot-au-feu is grand bouillon, in which to the meat and vegetables are added bones and connective tissue for the sake of the gelatine. This, when cold and

strained, forms a slightly firm jelly, which can be cleared and flavoured in many ways.

A still richer form of soup is one which to the grand bouillon, extract or purée of roast meat, fowls and vegetables have been added, and this is known as a consommé. This is, of course, most albuminous.

In framing a diet, due weight must always be given to the composition of the particular soup indicated.

### General Serving of Food for Invalids

If the patient is always objecting to the food, it is quite possible that the manner in which the food is served is responsible for the patient's fastidiousness.

Everything should be served as daintily as possible, the dishes, glasses, and traycloth being thoroughly clean. We must remember that to most invalids the meals are the events of the day, and too much care cannot be taken to ensure that everything regarding the food should be done in as attractive a way as possible. Patients should not as a rule be consulted as to their meals, but there should be an endeavour to discover beforehand what the patient likes. Any particular fancy as to sweetness or otherwise should be remembered. Untouched food should never be left in an invalid's room, but should be put aside in a cool place, and no food should be cooked or prepared in the invalid's presence if it can be done elsewhere. Food should never be tasted, or cooled by being blown upon, as these disgust the patient.

In the case of *helpless patients* who cannot feed themselves nourishment may be given by the spoon, or the feeding cup. When the patient is very helpless a useful device is to pull out the cheek, by inserting the finger between the gum and the cheek, and then introduce the fluid nourishment slowly at one side. A tea-spoon is the most convenient size of spoon for child feeding, and a dessert-spoon for the adult. As to drinking cups, the shape made with three handles is very convenient for the patient to use when feeding himself; but when the services of a nurse are required, a small boat-shaped feeding cup with a curved spout and about three inches of rubber tubing attached is the most useful. In the latter case great care must be taken to maintain thorough cleanliness of the apparatus in use.

In feeding with a spoon or feeding cup it is certainly easier for the patient to have the head raised if it is permissible; the nurse in these cases passes the left arm under the pillow on which the patient is lying, and gently raises the head.

*Nasal feeding* is called for in some cases of insensibility, melancholia, or other variety of insanity, and after tracheotomy. This method of supplying nourishment is very important. Half a pint to a pint of fluid food can be given at one time. The apparatus is simple; the small glass barrel of a urethral syringe has about 10 inches of narrowish rubber tubing attached. This is well oiled, and is passed gently along the floor of the nose in a backward and slightly outward direction; it slips over the posterior surface of the velum palati, and from thence into the pharynx and gullet. Patients soon become tolerant of this method. The nutriment is slowly poured from a jug in the glass receiver held directly over the patient's face.

*Rectal Feeding.*—Nutrient enemata are most valuable in certain circumstances, enabling the patient to hold out and to maintain his strength until the crisis that called for their use is over. Life may be maintained by rectal feeding alone for several weeks if all due precautions are taken with regard to it. As the large bowel possesses powers of absorption, but very slight if any power of digestion, the food given in this way must have the ingredients transformed into that state which is most easily absorbed. If this is not attended to, the nutrition will be defective, and a very troublesome irritative state of the bowel is induced which may become an insuperable obstacle to further successful treatment of the case.

The foods selected for rectal feeding must, therefore, be bland and easily absorbed. Predigested albumin and starch, and also fat in the form of an emulsion, are all absorbed in the rectum. Therefore peptonised beef tea, peptonised milk gruel, or maltine and peptonised milk, are all most useful forms of food. If peptonised food is not at hand, two tea-spoonfuls of liquor pancreaticus should be added to a 3-ounce enema of beef tea or of milk gruel shortly before its injection. Wine or spirit can also be added, and are to a certain extent absorbed. If the patient is suffering from pyrexia, grape sugar may be added to the enemata to supply the fuel food required.

A good enema for occasional use is—1 egg, 2 table-spoonfuls of milk, and a table-spoonful of brandy, well beaten up together.

The following special rules should be observed in the administration of a nutrient enema. (See also "Enemata," vol. iii. p. 119.)

1. Two to two and a half ounces is the average size of an enema for an adult. More is apt to set up peristalsis.

2. The enema should be injected slowly and gently.

3. The temperature of the enema should be lukewarm, about 105° F.

4. The nozzle of the syringe should be lubricated with oil or vaseline, and care must be taken in the introduction of the syringe that the same spot of mucous membrane is not pressed upon each time. After injection there should be gentle pressure over the anus with a soft pad.

5. Care must be taken that the rectum does not become loaded with fæces. A preliminary washing out the bowel with tepid normal saline solution (75 per cent. NaCl) at least once in twenty-four hours is very desirable.

6. A few drops (5 to 20 mins.) of tinct. opii given in every second enema is useful when the enema is retained with difficulty, and also as a prophylactic measure.

7. Not more than one enema every six hours should be given if possible.

Recourse may be had to the use of various forms of nutrient suppositories. Of these there are many excellent forms of prepared nutrient suppositories now in the market, which can be used to alternate with the home-made peptonised materials. It is advisable to bear in mind that the sediment from some of these preparations may possess rather more irritating properties than home-made preparations. Hence the great value of the occasional douching of the bowel above referred to.

### Milk

A thorough understanding of the chief points to be attended to in the administration of milk is very important, as it is so often the chief and only agent we have to fall back upon in cases of illness.

Good milk should contain 3 to 4 per cent. of fat. Its freshness and purity are essentials.

Milk should be obtained fresh twice daily, and should never be kept standing in the sick-room. It very rapidly becomes contaminated by bad air and emanations of decomposing substances, and thus soon loses its freshness. It should be brought into the sick-room in very small quantities, and should be kept covered. Unless the source of the milk is beyond suspicion the milk should be boiled or scalded.

Boiling most readily kills the germs, but unless a patient likes boiled milk it is almost impossible to get him to take it.

*Boiled Milk.*—If the milk is to keep some time it must be boiled when quite fresh. This is best carried out in a lined saucepan that has been rinsed out with water, or in a milk-can standing in a saucepan of boiling water.

The fire must be clear to avoid smoking. The milk must not be brought to the boil too slowly or it will curdle; and for the same reason it must only boil for a few minutes. As boiled milk that has been kept for several hours will not stand being boiled again, and hot boiled milk is more palatable than cold, therefore one has to fall back on sterilising or Pasteurisation of the milk to avoid waste. This consists in keeping the milk at a temperature of about 158° F. for twenty minutes; this is sufficient to kill most germs, but not the spores nor the lactic acid producing bacteria. This form of cooking does not alter the flavour of milk.

There are many sterilisers in the market, and the process is fully described under "Infant Feeding" (vol. iv. p. 389); but it can be roughly done for occasional use by placing the milk in a deep jug and standing it in a pan of water, the water standing at a higher level than the milk. Boil the water for twenty minutes, but do not let the milk boil. This does not alter the flavour of the milk.

The amount of milk that should be taken per diem by a sick adult person in bed, when milk is the sole diet, is about four to five pints.

The possible disadvantages attendant on milk administration are, as a rule, easily overcome. Of these the more important are difficulties in connection with its curdling. In health, the normal curd usually readily redissolves in the alimentary canal; in illness, either from the want of digestive agents, or over-acidity of the stomach, a hard firm curd may form, which is an irritant to the whole intestinal surface, producing pain, flatulence, diarrhœa, or constipation, and may be discharged unchanged in the stools in some cases. This hard curd may be very dangerous in intestinal ulceration, as after typhoid. To avoid this the milk may be simply diluted to a third or a half by some mineral water, such as potash, Seltzer, soda, or an aerated plain water; or it may be diluted, and the formation of anything but a fine granular curd prevented, by mixing the milk with barley or toast water.

*Clear Barley Water*

2 oz. pearl barley (Robinson's).
1 pint boiling water.
Rind of half lemon.
Sugar to taste.

*Method.*—Wash barley well and put into a jug with thinly peeled rind of a lemon, and a little sugar. Pour freshly-boiled boiling water on barley. Cover jug and stand till cold. Strain, and it is ready for use.

*Thick Barley Water*

(Barley can be used twice.)

2 oz. pearl barley.
2 pints cold water.
Rind and juice half lemon.
Sugar to taste.

*Method.*—Wash the barley well, put it into a saucepan with cold water to cover it. Bring the water to the boil and then pour it away. Rinse the barley with cold water and rinse out the saucepan. (This is to blanch the barley, and if it were not done, the colour of the water would be dark.) Return the barley to the saucepan with two pints of cold water and the thinly peeled rind of half a lemon. Simmer slowly for two hours, strain, sweeten, and strain in lemon juice. This is served hot or cold. Time, two hours.

*Note.*—Omit the lemon juice if it is to be mixed with milk. It is a refreshing drink and

a favourite with most invalids: a useful diluent for milk.

*Toast Water*

1 slice crust of bread.
1 pint cold water.

*Method.*—The crust of bread is better than slice, as it does not turn sour so soon.

Toast well on both sides until dry and nicely browned—not burnt. Have the fresh cold water in a jug, and break the toast into pieces and put into it. (If the water is poured on to the toast, it would be thick and not clear.)

Cover the jug, and let the toast remain soaking until the water is the colour of sherry wine. Strain and serve cold. This makes a refreshing drink with a little lemon juice added, and is also a useful diluent for milk, breaking up the curd.

If the patient is troubled with symptoms of acidity, an alkali such as carbonate of magnesia, as much as will lie on a sixpence, or a third part of lime-water may be added. This neutralises the acidity, and also prevents clotting.

Diarrhœa coming on while patient is on a milk diet is usually due to curd, and the above methods will usually stop it. The following can also be tried:—

*Flavoured Milk*

½ oz. ground rice.
½ pint milk.
Sugar.
A few pieces of cinnamon.

Mix the milk with the ground rice, pour into a small lined saucepan, allow it to simmer by the fire for half an hour. Do not let the milk boil over, and stir occasionally with a wooden spoon.

Sweeten to taste. Add some little bits of cinnamon stick, and serve hot or cold.

Rice, tapioca, and sago can be used in the same way. The flavouring can be varied with cloves or nutmeg.

If constipation is present it can generally be overcome by the adding 3j of soda bicarbonate to the diluted milk, or by diluting some of daily allowance of milk with Vals or Vichy water.

Whey.—If milk is badly tolerated even after diluting, whey is often available, and can be made very palatable. It is a pleasant stimulant drink food; the milk sugar in it gives a certain food value, and this can be increased by the addition of one tea-spoonful or more of lactose added to the pint. The absence of curd makes it easily digestible. Whey can be made in the following ways:—

*Whey*

1 pint new milk.
1 tea-spoonful of rennet (Mackay's is tasteless).

*Method.*—Warm the milk in a basin to a lukewarm temperature (blood-heat, the temperature of new milk), and stir in the rennet.

Let it stand in a warm place about fifteen minutes, until the curd forms and the whey is quite clear. When cool, break up curd and strain off the whey through a piece of clean muslin.

*Lemon Whey*

½ pint milk.
1 table-spoonful lemon juice.
Sugar to taste.

Prepare as wine whey, using lemon juice instead of sherry wine. Time, five minutes.

*Wine Whey*

1 gill of milk.
½ gill sherry wine.
1 lump of sugar.

Rinse out saucepan; put in milk and sugar, and bring to the boil. When boiling, add the wine and the milk will curdle. Let it boil again and strain through a piece of muslin.

*Cream Whey*

This can be added to the ordinary whey or to the wine whey.

Add two table-spoonfuls of cream to half pint of whey.

Cream is most useful in cases where feeding up is required, due to its large quantity of fat. It may be added to whey to increase the nutritive value of infants' food when attempting to make an artificial mother's-milk.

It is very largely used in invalid cookery and in the dietary of the aged to supply the place of oily fat food, and is added to soups, fish creams, blanc-manges, etc.

Again, cream is of great use in the dietary of the diabetic, taking the place of milk and whey.

*Skim milk* is an excellent beverage in pyrexial states where fresh milk is too rich.

*Butter Milk.*—This is slightly more digestible than ordinary milk. The lactic acid present shows there must be a diminution of lactose, and this makes it valuable (vol. ii. p. 358) in the thirst of diabetes.

*Koumiss—Fermented Mare's Milk.*—This preparation has been used with a certain amount of success by consumptives in Russia. The secret of the cure is that the milk is slightly fermented, highly digestible, and large quantities can be taken without producing dyspepsia. The Russian mode of cure is to rise early and to take a glass of koumiss every half hour, with the exception of two hours preceding dinner and supper.

*Kephir* is a similar product prepared in the following manner from cow's milk:—

*Kephir*

3 gills new milk.
One small piece sugar.
½ gill boiling water.
German yeast size of a nut.
Quantity for each pint bottle.

Take the milk as new as possible. Fill the

bottle nearly full of milk and water, and put into each a small piece of yeast and a lump of sugar.

Cork them tightly with new corks and fasten them down securely with wire. (The corks should be soaked for some time before being used.) Keep in a warm place, and shake them two or three times a day for from four to six days.

This becomes very effervescent, and unless great care is used in opening a considerable quantity may be lost.

Koumiss and kephir are both more easily digested than ordinary milk, and owing to the destruction of a part of the sugar they are useful in various disorders of the digestive tract.

"Protene," the pure caseinogin from milk prepared as a flour, can now be obtained in the form of bread and biscuits (see vol. ii. p. 358). "Plasmon," a somewhat similar product, obtained from the proteids of milk in the form of a granulated soluble powder, is a recent useful addition to invalid dietetics. It can be added to gruels, meat teas, puddings, etc.

*Milk sugar, or lactose*, has the advantage of not being very sweet. It may be added to milk soups or other liquids to increase the food value without appreciably altering the flavour.

Eggs.—Next to milk an egg is one of the most valuable articles of an invalid's diet. Its composition is as follows:—

| | Water. | Proteid. | Fat. | Ash. |
|---|---|---|---|---|
| White . | 85·7 | 12·6 | 0·25 | 0·59 |
| Yolk . | 50·9 | 16·2 | 31·75 | 1·09 |

The ash is exceedingly rich in an organic iron compound and phosphorus, hence its acknowledged usefulness in the dietary of children in chlorosis.

The excellence of an egg depends upon it being perfectly fresh and properly cooked. If cooked too long it becomes indigestible, but a lightly cooked egg is as digestible as a raw one.

*Raw eggs* can be served in the following ways:—

*Prairie Oyster*

1 fresh egg.
1 table-spoonful of vinegar.

Put the vinegar into a small cup and break the egg into it. Serve at once. This is a very digestible way of serving an egg, and it is very cool and refreshing.

The next three recipes give the way of making up raw eggs into nourishing stimulant drinks. By adding nutmeg, cinnamon, or lemon juice the flavour can be altered.

*Egg Drink*

1 egg.
1 tea-spoonful sugar.
1 table-spoonful sherry wine.
1 teacup of milk.

Beat up the egg, add to it the wine and sugar, and beat together with a fork slightly, and strain through a fine wire strainer.

Heat the milk in a small saucepan, and when almost boiling pour it on to the egg, stirring all the time. Serve hot.

This can be made without wine, flavouring with cinnamon or lemon juice. The yolk of the egg only may be used, and soda water instead of milk.

*Plain Egg Flip*

1 teacup of milk.
1 tea-spoonful of sugar.
1 white of egg.

Boil the milk or make it thoroughly hot; beat up the white of egg to a stiff froth. Pour the boiling milk over the white of egg, stirring all the time. Add sugar to taste, and serve.

*Rich Egg Flip*

1 white of egg.
1 table-spoonful of cream.
1 table-spoonful brandy.
Sugar to taste.

Beat up the white of egg stiffly. Add to it the brandy and cream, with a little sugar if wished. Mix very thoroughly and serve.

*Cooked eggs* may be served by themselves in several ways.

*Boiled Egg*

Put enough water into a small saucepan to entirely cover the egg. Allow the water to boil; lower the egg gently into it. Draw the pan to the side of the fire, and allow the water to simmer slowly. Cook the egg for 3½ to 4 minutes, then lift out and serve at once. If it is allowed to stand it becomes hard.

*Poached Egg*

1 egg.
Boiling water.
Lemon juice.
Pinch of salt.
Round of toast.

Break the egg into a cup, keeping the yolk whole. Into a small saucepan of boiling water, add a pinch of salt and a squeeze of lemon juice. Draw the water to the side, and when just off the boil slip the egg carefully into it. Cook slowly for three minutes, when the white should be quite set. Lift it out with a small fish slice or perforated spoon, and trim off any ragged edges of white. Place on a square of newly-made toast and serve at once.

If required, this egg can be made richer by being served on buttered toast, or a little hot cream poured over it. Also, a poached egg served on two table-spoonfuls of carefully prepared spinach is a very nice dish. For method of preparing spinach, see p. 22.

*Rumbled Egg*

1 egg.
1 table-spoonful milk.
Pepper and salt.
¼ oz. butter.
A piece of toast.

Heat in a small saucepan the milk. Pour into the hot milk the egg beaten up in a cup with pepper and salt.

Stir quickly over the fire until it begins to thicken; remove it then from the fire, and continue stirring until it forms a creamy mixture.

Put on a piece of newly-made toast and serve at once. If cooked too long, or allowed to stand, it becomes leathery and indigestible.

*Baked Egg*

Grease a china or paper ramequin case thoroughly; into this carefully break an egg. Add pepper and salt and a tea-spoonful of cream.

Place in a moderate oven and bake three or four minutes until lightly set, and serve at once.

Eggs also form the basis of omelets, custards, soufflés, and are added to many farinaceous puddings to improve them.

PREPARED AND PREDIGESTED FOODS

These are mainly of two kinds: (*a*) those in which there has been a predigestion of the carbohydrates with malt; (*b*) those in which the albumins have been converted into peptones and the carbohydrates into more soluble sugar, these changes being effected by the action of the active ferments of the pancreatic juice.

The so-called *prepared foods* are those sold by the manufacturers as suitable for babies and invalids. Their right to this description consists in their containing a certain quantity of malt which possesses the power of converting insoluble starch into glucose and grape sugar.

In all these foods the malt is mixed with some variety of baked flour. The proportion of sugar and the presence or absence of milk (desiccated) are the varying factors in their composition. Those made with desiccated milk possess a higher nutritive value, and corresponding care must be taken in their administration when taken dissolved in milk. On the other hand, prepared foods in which milk is not an ingredient must be made up with milk. Those made with milk are decidedly more palatable than those mixed with water.

There are numerous brands of this class of food now in the market, many of which are distinctly valuable in practice. While it is impossible to enumerate all the varieties, reference may be made to Allen & Hanbury's, Benger's, Mellin's, and Savory & Moore's, as these are the brands which the writer can safely recommend from personal experience.

They are all taken mixed with fresh milk.

*Of the Allenbury series* the third food is the best suited for invalids. Nos. 1 and 2 are prepared with desiccated milk, and though very satisfactory for young infants, the taste of them for adults is not quite so palatable. No. 3 has only a certain proportion of its starch converted into a soluble carbohydrate; this leaves some starch (although cooked and rendered easy of digestion) for the digestive powers to act upon. This may with advantage be used as an intermediate food during convalescence when the digestive powers are slowly regaining their function. The great advantage of having a large choice of malted foods lies in the fact that the patient can get a certain change in the flavours and dishes even when on a very restricted diet.

By the use of malt, either in the form of a malt infusion or the extract of malt, many patients who experience much difficulty in digesting the starchy constituents of food, will find that they can do so quite comfortably.

The following valuable recipes may be mentioned:—

*Malt Infusion* (Sir W. Roberts' recipe)

Crushed malt, 3 oz.
Cold water, ½ pint.

Mix the malt thoroughly with the cold water in a jug. Let the mixture stand nine hours. Decant off the liquid carefully, and strain this through muslin until it comes out clear and bright.

This has a colour like dark sherry, and a faint maltish taste.

It should be freshly made as it is liable to ferment.

*Malted Gruel*

The gruel may be made of any of the farinaceous substances — oatmeal, Quaker oats, farina, arrowroot, barley, or lentil flour, preferably those from prepared flours. For method of making gruel, see p. 15.

When cool enough to swallow add the malt infusion or an extract of malt. About one table-spoonful of the infusion or a tea-spoonful of the extract is sufficient to digest a plateful of gruel. The action is very rapid, and in a few moments the gruel becomes thin from the transformation of the starch into maltose.

Several such dishes can be made out of invalid food that form a pleasing variety. Any invalid food can be used as a basis for these recipes; Benger's food is especially well adapted for it.

*Invalid Food*

1 oz. of "invalid food."
4 table-spoonfuls of cold milk.
½ pint of boiling milk.

Mix the food into a paste with four table-spoonfuls of cold milk. Add this gradually to the half pint of milk, which should be almost boiling; keep stirring all the time.

Set aside in a warm place for about twenty minutes. Then boil the whole for a minute, stirring all the time, and strain if necessary.

This can now be taken as an invalid food or used in the making of various dishes, and the recipes are those given by Benger.

*Custard Pudding*

½ pint of the "invalid food" made with milk.
2 eggs.
2 tea-spoonfuls of sugar.

Prepare the food as in the given direction on the tin. After it has been allowed to cool add to it the sugar and the eggs well beaten.

Pour into a small greased pie-dish, and bake in a moderate oven from ten to fifteen minutes. Flavouring, such as nutmeg, cinnamon, or grated rind, may be added. This may be served hot or cold.

*Batter Pudding*

½ pint of the "invalid food" made with milk.
2 eggs.
1 pinch of salt.

Take the prepared food and give it a final boiling. Let it cool slightly, and add the salt and eggs well beaten.

Pour the mixture into a well-greased basin, cover with greased paper, and strain slowly for half an hour. Do not fill the basin too full, as the pudding will rise. When ready, it will be firm to the touch.

It can be served with sugar, preserves, or meat gravy.

*Blanc-mange*

½ pint of "invalid food" made with milk.
3 tea-spoonfuls of corn-flour.
2 tea-spoonfuls sugar.
Flavouring, vanilla or lemon.
2 table-spoonfuls of cream.

Mix the corn-flour and sugar in a basin and gradually add the food, mixing well together. Pour into a saucepan and stir over the fire until boiling. Boil for five minutes. Add the flavouring and cream.

Pour into a wetted mould, and put into a cool place until set. When ready, turn out and serve with stewed fruit.

*Invalid Food Jelly*

½ pint of "invalid food" made with milk.
½ oz. French leaf gelatine.
2 tea-spoonfuls of castor sugar.

Make the invalid food according to directions. Add while hot the gelatine, and stir over the fire until dissolved.

Add the sugar and any suitable flavouring, such as port or sherry wine.

Pour into a wetted mould until set.

Turn out and serve cold.

PEPTONISED FOODS

Peptonised foods should only be employed when it is necessary to assist the digestive organs for a time, by giving the alimentary canal some degree of physiological rest. As soon as the digestive organs have regained their vitality their use should be abandoned. Dr. Fothergill tells the story of the Nottinghamshire farmer whose son had been largely fed on peptonised food, who said, "It is just making a pauper of his stomach, doctor," and as a ratepayer he knew that paupers would not work if they could help it.

Pepsin and dilute HCl may be used to predigest proteid food, but it is more convenient to employ liquor pancreaticus, as it has less effect on the flavour of the food.

In preparing food by means of pancreatic ferments there are many reliable preparations that can be used. Liquor pancreaticus (Benger's) and Fairchild's zymine peptonising tubes may be mentioned. It is well to remember that the ferments must not be added if the temperature of the food is higher than 140° F. Roughly speaking, this is a temperature that will not scald the mouth. If a greater heat is applied the ferment is destroyed.

The extreme solubility of predigestive products, whether starch or albumin, makes them appear thin and watery; but to a delicate invalid without an appetite this is an improvement, as the sensation of thickness is generally much objected to.

If peptonised foods are prepared with care there is no reason why these foods should have an unpleasant taste and smell. The following methods of preparation of different predigested foods may be commended:—

*Peptonised Milk*

Milk, 1 pint.
Water, ¼ pint.
Liquor pancreaticus, 2 tea-spoonfuls.
Bicarbonate of soda, 20 grains (½ small tea-spoonful).

After allowing the milk to stand, skim the milk. Dilute the skimmed milk with ¼ pint of water, heat to a temperature of 140° F.

Mix with the warm milk 2 tea-spoonfuls of liquor pancreaticus and 20 grains of bicarbonate of soda.

The mixture is poured into a covered jar, with cosy over it, and put near the fire. Let this stand for 1½ hours.

Bring the mixture to the boil for two to three minutes to arrest any further fermentative action, which makes the milk unpalatable. Now add the cream that was removed, and it is ready for use.

*Cold Process.*—Mix the peptonising agent in cold water and cold milk as usual, and immediately place the bottle on ice, without subjecting it to any heat.

When needed pour out the required portion, and use in the same manner as ordinary milk.

It is recommended to try the milk prepared by the cold process in those cases in which food is not quickly digested after ingestion, and in which the digestive functions are impaired, or even practically suspended. It has been found in many such cases that the peptonising

principle exerts sufficient action upon the milk in the stomach to ensure its digestion and proper assimilation. If the milk so prepared be not well borne, or any evidence appear of its imperfect digestion, it should be sufficiently predigested—peptonised—by the usual warm process.

Milk by the "cold process" is especially suited for dyspeptics and persons who ordinarily find milk indigestible. This milk has no taste or evidence of the presence of the peptonising agent.

*Effervescent Peptonised Milk.*—Put some finely cracked ice in a glass, and then half fill it with cold Apollinaris, Vichy, or carbonic water as preferred, then quickly pour in the peptonised milk and drink during effervescence.

Peptonised milk may be made agreeable to many patients by serving with a little grated nutmeg, sweetened or flavoured with a little brandy, etc.

*Peptonised Gruel.*—The gruel may be made from any farinaceous article in common use—oatmeal, Quaker oats, farina, arrowroot, barley, pea or lentil flour.

The gruel should be well boiled. It is placed in a covered jar when it is below the temperature of 140° F. Add liquor pancreaticus in proportion of 1 table-spoonful to 1 pint of gruel; and the jug is covered and kept warm as before. After standing for two hours, boil the mixture for five minutes and then carefully strain.

The pancreatic ferments have two actions on the farinaceous matters:—

1. The starch of the meal is transformed into sugar.
2. The albuminoid matters are peptonised.

*Peptonised Milk Gruel.*—This was very highly spoken of by Sir W. Roberts. It is an artificially digested milk, and forms a complete and highly nutritious food for weak digestions.

A thick gruel is made from any farinaceous material (see p. 15).

To the boiling gruel add equal quantity of cold milk. This will then have a temperature of about 140° F.

To each pint of gruel and milk add 3 teaspoonfuls of liquor pancreaticus and 20 grs. of sod. bicarb.

Let it stand covered in a warm place for two hours, boil for five minutes, and then strain.

The bitterness of the predigestive milk is almost completely covered in this peptonised milk gruel, and most invalids will take it without the least objection.

*Peptonised Milk Lemonade*

Juice of 1 lemon.
1 table-spoonful of sugar.
1 gill peptonised milk.
1 gill effervescing water.
Some small pieces of ice.

Fill a small tumbler one-third full of cracked ice. Add the water, lemon juice, and the sugar to the ice, and then fill up the glass with milk.

*Peptonised Milk Punch.*—Take a small tumbler, and fill it about one-third full of finely crushed ice; pour on to this a table-spoonful of St. Croix rum and half a table-spoonful of brandy, then fill the glass with peptonised milk, stir it well, sweeten to taste, and sprinkle over the top of the mixture a little grated nutmeg.

*Peptonised meat broths and teas* can be made in the following manner:—

½ lb. lean beef finely minced.
1 pint water.
20 grains bicarbonate of soda.
1 table-spoonful of liquor pancreaticus.

Mix together and simmer slowly for 1½ hours; when it has cooled down to 140° F. add liquor pancreaticus. Keep it warm under a cosy for two hours; occasionally shake it up. Then decant off the liquid portion and boil for five minutes. This has a relative value in nitrogenous materials almost equivalent to milk. The beef tea can be seasoned with celery seed cooked in the tea, or vegetable flavourings can be added by placing a small muslin bag containing some carrot, turnips, parsley, onion, in the tea while cooking, and afterwards removing it. If desirable, the tea can be thickened by the addition of a roux (paste) made with a little baked flour (p. 13).

In addition to home-made peptonised beef teas and juices there are a large number of reliable proprietary medicines, of which may be mentioned Armour's nutrient wine of beef peptone; Benger's peptonised beef and chicken jelly; Brand's beef, mutton, veal, and chicken peptone; and Fairchild's panopeton. This latter preparation claims to present bread and meat in a perfectly soluble form; it is certainly very palatable.

Messrs. Savory and Moore have manufactured for the use of invalids and convalescents a very palatable form of peptonised cocoa and milk, and also a concentrated peptonised milk and coffee (café zylak). These are delicious preparations, and can be much enjoyed even by people who cannot take cocoa or coffee in any other form.

Recently a new preparation called somatose has been placed on the market. It claims to be a very concentrated and partially digested proteid substance, the albumin being mostly converted into albumose. It is a yellowish powder freely soluble in water, and forms almost a tasteless solution. It is useful in increasing the nutritive value of beef tea, etc. to which it may be added.

*Peptonised Soups, Blanc-manges, and Jellies.*—With a little ingenuity a variety of peptonised dishes can be obtained by preparing soups, blanc-manges, and jellies with peptonised ele-

ments. There is no difficulty in doing this if it is always remembered that if any peptonised fluid is used it must be boiled in order that the action of the ferment may be arrested.

For soups, use peptonised gruel (p. 11) instead of water. For the making of blancmanges (p. 16), peptonised milk is added to the cream. For jellies, mix the liquor pancreaticus with gelatine, the flavouring matter to be employed in the proportion of 2 tea-spoonfuls to the pint; allow it to stand for 1½ hours in a warm place, and then check the further action by boiling. (For jellies, see p. 16.)

*Peptonised Oysters*

Take half a dozen large oysters with their juice and half a pint of water. Heat in a saucepan until they have boiled briskly for a few minutes. Pour off the broth and set aside.

Mince the oysters finely, and reduce them to a paste with a potato masher in a wooden bowl.

Now put the oysters in a glass jar with the broth that has been set aside, and add—

Liq. pancreaticus, 2 tea-spoonfuls.
Soda bicarb., 15 grains.

Let the jar stand in hot water, or a warm place where the temperature is not above 115°, for 1½ hours.

Then pour into a saucepan and add half a pint of milk.

Heat over the fire slowly to boiling-point.

Flavour with salt and pepper or condiments to taste, and serve hot.

There will be found a few very small pieces of the oysters undigested, and these may be strained out or rejected in eating the soup, but will not be unacceptable to the stomach, except in very rare cases.

The *milk* will be sufficiently digested during the few minutes which will elapse before the mixture boils, *if heated gradually.*

Be sure to boil the peptonised oysters to finish the process.

FOOD IN PYREXIAL STATES

When fever is present, two important indications are the supply of *fluid* in the form of pleasant beverages to overcome the dry and parched sensations in throat and fauces brought on by the high temperature; and, secondly, the administration of a liberal supply of carbohydrates, which is necessary to meet the increased oxidation in the tissues resulting from the high temperature.

With suitable feeding the wasting is less, and the patient does not become so reduced, and, consequently, the convalescence is more rapid.

BEVERAGES. — For the thirst the following recipes may be given for refreshing drinks, but they all have a very low nutritive value. If made with barley (p. 6) or rice water, instead of plain water, the value is slightly increased. Again, the addition of one tea-spoonful of milk sugar or lactose to the pint increases the value considerably without affecting the flavour.

*Rice Water.*—Take 2 ounces of rice into an enamelled saucepan with 3 pints of water, and boil for 2½ hours. Stir it frequently and skim carefully.

Strain into a jug through a fine wire sieve, and rub through the glutinous part, but not the hard portion.

Put in no flavouring unless ordered by the practitioner.

*Apple Water*

1 lb. of apples.
½ lb. brown sugar.
1 gallon boiling water.

Cut up the apples into quarters; take them and put into a jug with the brown sugar, and pour the boiling water over it. Let it stand until cold; pulp the apples and the fluid through the colander. Bottle for use; do not cork the bottle. Keep in a cool place.

*Apple Toast and Water.*—Toast a piece of bread slowly until it gets quite black. Placed in a jug of apple water for three-quarters of an hour, then strained, makes a very nice and refreshing drink.

*To make Apple Barley and Apple Rice Water.* —Boil ½ lb. of rice or ¼ lb. of barley, blend with the apple water, and then strain it.

In spring and summer rhubarb, green gooseberries, black and red currants, and raspberries, may all be made into nice cooling drinks if used in the same proportion.

*Imperial Drink*

½ oz. cream of tartar.
Juice of 1 lemon.
2 table-spoonfuls of sifted sugar.

Place the ingredients in a jug, and pour over a quart of boiling water, and cover until cold.

*Lemonade*

(*a*) Juice of 1 lemon.
1 pint of water.
1 oz. of sugar.
1 egg.

Dissolve the sugar in the water; add the juice of the lemon; beat up the white of egg, and add this.

This makes a very palatable drink with slight nutritive value.

(*b*) 4 lemons.
¼ lb. of loaf sugar.
3 pints boiling water.

Rub some sugar on the rinds of two of the lemons until it is yellow.

Strain the juice of the four lemons; put the sugar and juice into a jug, and pour over the water. Cover it until it is cold.

*Orangeade*

2 oranges.
Juice of 1 lemon.
2 or 3 lumps of sugar.
1 pint boiling water.

Wipe the oranges with a damp cloth, and peel the rind off one of them very thinly. Put this into a jug with sugar, and strain in the juice from the oranges and lemons. Pour on the freshly boiled water. Cover closely until cold, and then strain.

*Black Currant Drink*

2 table-spoonfuls black-currant jam.
1 tea-spoonful arrowroot.

Take the black-currant jam and boil it in a quart of water. Cover it and stew gently for half an hour; then strain it, and set the liquor again on the fire. Mix the arrowroot in cold water; pour over it the boiling liquor, and keep stirring. Then let it get quite cold.

(This is a very pleasant drink, and specially nice for a rough throat.)

Malt extracts (see p. 9), with effervescing water, are also excellent as beverages. They quench the thirst, and supply grape sugar (maltose), soluble dextrine, and a small quantity of soluble albuminoids.

Meat Broths and Extracts of Teas.—Meat infusions of beef, mutton, veal, and chicken are now in considerable demand. These are to be regarded more in the nature of pleasant, palatable, and stimulating beverages than as foods. They may be taken hot, cold, or iced. To this class belong the large class of meat extracts now in the market—Bovril, Liebig, Vigoral, Vimbos, and mutton extracts, *e.g.* Hipi.

*Home-made Beef Teas.*[1]—In their preparation it is well to employ beef from the buttock, rump, or thick flank, in preference to shin of beef. In the process of cooking the tea must not be allowed to boil, otherwise the juices are not extracted owing to the insoluble albuminous coating formed on the surface of the meat by the boiling process. In every case care must be taken to ensure that all the fat is removed from the surface before serving.

There are various ways in which the beef tea may be thickened, *e.g.* with egg, tapioca, bread crumbs, or baked flour, thus adding much to the nutritive value.

The baked flour is prepared as follows:—

*Baked Flour.*—Spread some white flour thinly on a dish, bake it in a moderate oven for about an hour until it is a delicate cream colour. Take it out and place on a sheet of kitchen paper; when cool roll it smooth with a rolling pin, pass it through a wire sieve, and then keep in a canister or bottle. This may be used for thickening meat teas and broths.

[1] Should always be made from freshly killed meat.

Puddings made with this form of flour are more digestible.

In serving soup to an invalid do not fill the cup or basin too full. A thin slice of old bread, or a piece of freshly toasted bread, or an unsweetened rusk should be served with the soup.

The following recipes may be commended. A glance at the composition of each will convey some idea of their relative value as nutritive fluids; some of them are mere stimulants.

Very little vegetable flavouring is given in the accompanying recipes; but, if permissible, celery seed, or a muslin bag containing a variety of vegetables, may be cooked with the beef tea and afterwards removed.

*Beef Tea*

1 lb. lean juicy beef.
1 pint cold water.
1 small tea-spoonful of salt.

*Method* 1.—Wipe the meat with a damp cloth and then place on a board, and with a sharp knife shred down as finely as possible. Keep back any pieces of connective tissue and fat.

Place in a basin with salt and water, stir it well up, and cover the basin with a plate, and if time permits let it stand for half an hour. Then pour the contents of the basin into a clean lined saucepan, place the pan over a slow fire, and whisk it well until it almost reaches boiling-point; *do not let it boil.* Then draw the pan to the side of the fire, put on the lid, and allow the beef tea to simmer as slowly as possible for about ten to fifteen minutes. Strain through a coarse strainer, pressing the meat as dry as possible with a wooden spoon. Remove all the fat before serving.

In cases where it is desirable to obtain the maximum of extractives in the tea, *i.e.* to form a very stimulating beverage—

*Method* 2.—Use the same proportions of meat and water, and prepare it in the same way as above, but afterwards place the meat in a beef-tea jar, and cook slowly in the oven for four hours. The jars are strong stone jars with a strong screw lid, and can be obtained from Dall and Company, Edinburgh. They are made in various sizes.

*Caution.*—After making beef tea in this way the jar must not be opened until the contents are cold. If opened before it is cold, the steam rushes up and is very apt to scald the cook's face and arms.

If preferred, chicken, mutton and veal, or veal tea can be made in a similar manner. In making chicken tea, the legs and dark parts of the fowl can be used for soup while the white portions can be served in some other way. The fillet of veal is the best portion to order. In the mutton and veal tea, equal parts of the two meats are used; this gives a very delicate flavour, and is much appreciated by many invalids.

*Thick Beef Tea*

(*a*) ½ pint of made beef tea.
1 yolk of egg.
1 tea-spoonful Grôult's tapioca.

Warm the beef tea and sprinkle in the tapioca, stirring all the time.

Let it simmer slowly by the side of the fire for about ten minutes until the tapioca turns quite clear.

Beat up the yolk of an egg in a basin or cup with a fork; pour the beef tea gradually over it, stirring all the time, and it is ready for serving.

(*b*) ½ pint of made beef tea.
1 tea-spoonful of arrowroot.
1 table-spoonful cold water.

Mix the arrowroot and water in a small basin until quite smooth. Then add it to beef tea that is being warmed in a pan, stir well for a few minutes to prevent it becoming lumpy. Then simmer slowly for ten minutes.

*Beef-Tea Gruel*

1 table-spoonful fine oatmeal.
1 gill cold water.
1 gill beef tea.
Salt.

Make the gruel with oatmeal and water, or milk, as above.

When cooked add to it the beef tea, stir until quite hot, but do not boil.

Season to taste and serve hot.

*Beef Purée*

½ pint of good beef tea.
¼ lb. lean juicy meat.

Warm in a pan the beef tea that is already made.

Wipe and shred very finely ¼ lb. of meat, pound it well, and rub it through a fine wire sieve. Take a cup and make it thoroughly hot; into the cup put the pounded meat, pour the hot beef tea on to it, stir it well up, and serve at once.

*Beef Essence.*—One pound of meat will make one gill of essence.

Prepare meat as in last recipe, shredding it down very finely. Place in a jar with a pinch of salt, cover with a strong piece of greased kitchen paper.

Place the jar in a saucepan containing sufficient cold water to reach fully half-way up the jar, and let it steam slowly for four to five hours.

When ready, strain through a fine strainer and press the meat well with the back of a wooden spoon to extract all the juice. This liquid is thus pure beef juice.

When cold, this is in the form of a jelly.

*Meat Juices.*—This variety of food differs greatly in nutritive value from those extracts previously mentioned. The meat juice is extracted without any heat, and thus a large portion of the albumin is present. Brand's, Valentine's, and Wyeth's meat juices are all excellent preparations, and are to most people very palatable.

Raw meat juice can be prepared in the following manner; its great drawback is its decidedly objectionable appearance, which can be partially overcome by serving in a red glass or a cup.

¼ lb. best rump steak.
½ pint cold water.
Pinch of salt or sugar to taste.

Prepare the meat in the same way as for Beef Purée. Place the shredded meat in a basin with water and the salt. Stir it well with a fork, cover with a plate, and let it stand ¾ hour, stirring occasionally. The liquid will then be a bright red colour. Strain through a fine strainer, pressing the meat with the back of a wooden spoon.

This should only be made in very small quantities, as it very soon becomes rancid.

A recent patent preparation prepared by Brand, and termed "Fever Food," is most nutritive; it consists of essence of beef, eggs, cream. In appearance it resembles custard, and has a very agreeable flavour which is quite distinct from the meaty flavours of the beef juices and extracts.

Milk is the staple food in febrile state. If badly borne it may be taken as whey or diluted with Seltzer, soda, or potash water. The fat present in the milk is most useful, and this method of administration of fat is practically the only one available, as such patients have usually a marked intolerance of other forms of fat. In cases with intestinal weakness, *e.g.* typhoid fever and the like, the milk must be prevented forming too hard and tough a curd, and the measures suggested to remedy this should be carried out (p. 6).

### Diet during Convalescence

As the digestive tract is more or less involved in all cases of acute illness, convalescents may all be regarded as dyspeptics for the time being. Thus, at first, great care must be taken both in quantity and the quality of the food.

However, the digestive capacity of convalescents varies. There is, for instance, the healthy young adult recovering from some sharp attack of an infectious fever, in whom the digestive organs are only weakened, not organically affected. His convalescence is rapid; he is soon capable of eating and digesting everything, and in this class of case there is no necessity to try and tempt the appetite. The food can be simply prepared, the danger is that the patient will overeat. On the other hand, we may have a delicate child recovering from scarlatina complicated by nephritis. Here it would be

absolutely wrong if we did not limit the quantity, and give very strict injunctions as to the quality of the food.

After typhoid fever, when convalescence is thoroughly established, any mass of undigested food, *e.g.* potatoes, or a piece of unmasticated meat, may be the cause of a relapse by setting up irritation in some recently ulcerated patch.

At first, in all cases, a continuance of the fever diet, of milk and potash, beef tea or meat infusions, and acid drinks, is indicated. This diet can be gradually extended to include easily digestible starches, either in the form of some prepared invalid food (p. 9), or as a home-made *gruel*, to which has been added malt infusion.

The two following recipes are useful for gruels:—

*Oatmeal Gruel*

1 table-spoonful fine oatmeal.
½ pint cold water.
Salt or sugar.

Put the oatmeal into a clean basin, and pour the water over it. Cover the basin and let it stand for at least half an hour, stirring occasionally. Then strain the liquid off into a small lined saucepan, and press the oatmeal as dry as possible. Stir over the fire until boiling, and let it boil from five to seven minutes. The thickness of the gruel is very much a matter of taste; if too thick add more water, or if too thin use more oatmeal.

Season with salt or sugar; wine or brandy may be added if required.

*Milk Gruel*

1 table-spoonful fine oatmeal.
½ pint milk.
Salt or sugar.

Made in the same way as last recipe, only milk is used instead of water.

*Barley Meal Gruel*

1 dessert-spoonful barley meal.
½ pint milk.
A small piece of butter.
Sugar or salt.

Mix the milk very gradually with the meal, stirring until quite smooth. Take a small lined saucepan, rinse out with cold water, and pour the barley and milk into it.

Stir constantly over the fire until boiling, and let it boil about six minutes; season to taste, and serve very hot. This is better eaten with a little cream. The thickness can be regulated as in oatmeal gruel. This can be made into a very good drink by making a thick gruel with water, then thinning it down with port wine; heat thoroughly, but do not boil again.

As the condition of the digestive tract improves, the desire for food increases, and this must, to begin with, be met by giving *milk puddings*, and *eggs* in the form of drinks, *e.g.* egg flip, etc., or as baked *custard*.

In the following recipes for baked milk puddings, the eggs can be omitted if desired. The presence of eggs in a pudding greatly improves its flavour and appearance, but they make it richer and more difficult to digest. If the pudding is to be made with eggs it is always lighter when the white of the egg is beaten up separately, as this introduces air into the pudding and causes it to rise.

Care must be taken to thoroughly cook all the farinaceous and starchy elements of the food. For flavouring, the most suitable are fresh lemon rind or juice, grated nutmeg, ground cinnamon, and essence of vanilla.

*Water Arrowroot or Corn Flour*

½ oz. arrowroot or corn-flour.
½ pint cold water.
1 tea-spoonful of sugar.

Put the arrowroot into a small basin; add to it a table-spoonful of cold water; break it with a wooden spoon until quite smooth.

Then pour on the rest of the water, mix well, and pour into a small lined saucepan. Stir this over the fire until it boils and thickens, then let it boil for ten minutes to thoroughly cook the arrowroot.

Sweeten to taste, and serve in a cup or small basin.

A little nutmeg may be grated on the top, and wine or cream added if wished.

*Milk Arrowroot or Corn Flour*

½ oz. arrowroot or corn-flour.
½ pint milk.
½ tea-spoonful of sugar.

Make in the same way as above, using milk instead of water.

*Arrowroot or Corn Flour Pudding*

½ oz. arrowroot.
½ pint milk.
1 egg.
1 tea-spoonful of sugar.

Make in the same way as arrowroot and milk, and thoroughly boil the mixture for five minutes. Remove the pan from the fire; add the sugar and any flavouring.

Separate the yolk from the white; add the yolk to the arrowroot whenever it has slightly cooled; add a pinch of salt to the white, and beat it up to a stiff froth. Stir this lightly into the mixture and pour into a greased pie-dish, wiping round the edges of the dish. Bake in a moderate oven until well risen and of a nice light brown colour.

Sprinkle some white sugar over it and serve at once, as it soon falls if allowed to stand.

*Semolina or Ground Rice Pudding*

¾ oz. semolina or ground rice.
¼ pint milk.
1 egg.
1 tea-spoonful of sugar.

Put the grain into a small lined saucepan and the milk with it. Stir these over the fire with a wooden spoon until boiling, and boil for a few minutes until the semolina swells and thickens. Remove the pan from the fire; add the sugar and seasoning, and when it has cooled mix the yolk of the egg well into it.

Beat up the white on a plate with a knife to a stiff froth and mix thoroughly; pour the mixture into a pie-dish and bake for ten minutes; sprinkle with sugar and serve at once.

*Rice Pudding*

1 oz. whole rice.
½ pint milk.
1 tea-spoonful of sugar.
1 egg.
Flavouring, if wished.

Well wash the rice in several waters; put it into a lined saucepan with cold water to cover it; bring it to the boil and pour the water off. This helps to burst the rice more quickly.

Then pour in the milk and let the rice simmer by the fire until thoroughly cooked; then add the yolk of the egg and the white as described in the last recipe, and bake in a well-greased pie-dish.

*Tapioca and Sago Pudding*

¾ oz. tapioca or sago.
½ pint cold milk.
1 egg.
1 tea-spoonful of sugar.
Flavouring.

If small crushed tapioca or small sago, make as in semolina pudding. If not, follow the directions:—

Let the tapioca soak about an hour in a basin covered with milk.

Rinse out a small lined saucepan, turn the tapioca and milk into it, and stir over the fire until it comes to the boil. Then simmer slowly until it turns clear, stirring every now and then for about twenty to thirty minutes.

If it becomes too thick while cooking add a little more milk. Then finish off the pudding with eggs, etc., as in semolina.

A nice light pudding can be made by cooking the sago or tapioca with water instead of milk, and adding stewed fruit or claret or port wine to it.

*Baked Bread-and-Butter Pudding*

1 or 2 slices of thin bread and butter.
1 egg.
½ pint of milk.
1 tea-spoonful of sugar.
A little grated nutmeg.

Cut some bread and butter rather thin, remove the crusts, and cut it into pieces about an inch square. Lay these into a small greased pie-dish, making the dish just about half full; beat up the egg in a small basin, and add to it nutmeg, sugar, and milk. Mix well together and pour over the bread in the pie-dish.

Allow the pudding to stand about ten minutes until the bread gets thoroughly saturated, then bake in a moderate oven from ten to fifteen minutes until nicely browned on the top. Sprinkle with sugar.

*Blanc-mange*

1 gill of milk.
1 gill of cream.
¼ oz. isinglass.
Rind ½ lemon.
1 oz. sugar.

Rinse out a small saucepan and put into it the milk, isinglass, and thinly-peeled rind of half a lemon. Let this stand by the side of the fire until the isinglass is dissolved and the milk well flavoured with the lemon. Stir occasionally, but do not let it boil.

Add the sugar, and strain into a basin to keep back the lemon rind. Add the cream, and stir occasionally until nearly cold. If not stirred the milk and cream will separate.

Pour into a small basin or mould that has been rinsed out with cold water, and put aside in a cool place to set. Turn out, and serve with a little red or black currant jelly.

*Baked Custard*

2 yolks and 1 white of egg.
1½ gill of milk.
1 tea-spoonful of sugar.
Flavouring.

Beat up the eggs in a basin with the sugar and flavouring; pour in the milk and mix again; strain the custard into a greased pie-dish. Stand the pie-dish in a Yorkshire pudding tin with some cold water round it.

Bake in a moderate oven from fifteen to twenty minutes until firm and nicely browned.

A thin slice of sponge cake may be placed on the top of the custard before baking.

JELLIES

Calf's-foot jelly is very troublesome to make and to clear, and is no better than jelly made from gelatine.

*Wine Jelly*

1¼ oz. French sheet gelatine.
3 gills cold water.
½ gill lemon juice.
½ gill sherry wine.
1 table-spoonful brandy.
3 oz. loaf sugar.
Rind of 1 lemon, *cut*.
2 or 3 cloves.
1 inch of cinnamon stick.
White and shell of 1 egg.
(The proportion of gelatine is 2½ oz. to 1 quart.)

Put all the ingredients into a lined saucepan; whisk until they boil; remove to the side of the fire when the scum begins to rise to the top. Cover the top of the pan with a plate, and allow it to stand fifteen minutes. Strain through a hot jelly cloth, run through three or four times till clear, and when cold mould in a scalded wet mould.

*Whipped Jelly.*—Melt the jelly by standing the basin containing it in a saucepan of hot water. When dissolved, put into a large basin and with a whisk whip it until it is quite cold and should become a firm froth and perfectly cold. By varying the ingredients in the wine jelly a good many different forms can be obtained, *e.g.*—

*Port Wine Jelly.*—Take 3 gills port wine, 1 gill water, and 1 table-spoonful of red-currant jelly instead of the sherry wine, lemon juice, and the large amount of water.

*Orange and Lemon Jellies* are made by substituting orange or lemon juice for the wine.

### Suggested Dietary for a Convalescent

As it is important for the practitioner to be ever ready with suggestions as to what individual patients may partake of, the following is a suggested dietary for five days. Doubtless in many cases a more rapid return to ordinary diet could be made, while in others the changes would require to be more gradually effected. The patient is supposed to have reached the stage when he is able to digest milk puddings when taken in moderation.

For bread-stuffs—thin slices of toast, the crusty portion of a Vienna roll, sweetened and unsweetened rusks, toasted sponge finger biscuits, rice biscuits, and plain biscuits may be recommended.

As the patient advances, eggs, fish, sweetbreads, chicken, small birds, rabbit, game, follow one another. Then come mince, beef quenelles, eye of a tender mutton chop, a tender slice from a joint or steak. The patient should always be reminded of the necessity of eating slowly and masticating well. Vegetables must be cautiously allowed at first.

All meals should be very simple.

#### 1*st Day*

7 A.M.—*If awake early.*—A small cup of freshly-made tea with cream, and small half slice toast.

8.30 ,, *Breakfast.*—Saucerful of oatmeal gruel with cream, and a little piece of Vienna roll—crusty part.

11.30 ,, A teacupful of beef tea unthickened, either hot or cold.

1 P.M.—Steamed whiting; half slice of bread.
Semolina or ground rice pudding.

4 P.M.—Potash and milk with rice biscuit or sponge cake.
A little fruit.

6.30 ,, Benger's food in any form (see p. 9).

9 ,, Some variety of meat tea or infusion.

#### 2*nd Day*

8 A.M.—Saucerful of hominy porridge or barley meal gruel, etc.
A small cup of tea, toast, and a very little butter.

11.30 ,, A teacup of chicken tea or a little fruit.

1.30 P.M.—Baked or steamed fish (p. 18).
Spinach.
Blanc-mange.

4 ,, Potash and milk, or a small cup of fresh tea, bread and butter, and sponge cake.

6.30 ,, Invalid food, or peptonised cocoa and milk.

9 ,, Some variety of meat infusion.

#### 3*rd Day*

8 A.M.—A lightly boiled egg.
A small cup of tea.

11.30 ,, A teacup of soup, or a little fruit and a drink of milk and potash.

1.30 P.M.—Rabbit. Chicken, roast—2 slices from breast. Pigeon, served with bread sauce.
A small helping of vegetable (not potato).
(Stewed vegetable marrow.)
Pudding. Apples (cooked) and water sago, eaten with cream.

4 ,, A small cup of tea, bread and butter, and biscuit.

6.30 ,, Invalid food.

9 ,, Some variety of soup.

#### 4*th Day*

8 A.M.—A piece of boiled or steamed haddock, whiting, or sole.
Tea, bread or toast and butter.

11 ,, Soup or egg drink.

1.30 P.M.—Sweetbread or tripe.
Vegetable. Stewed tomato (do not eat skins).
Stewed fruit with custard.

4 ,, Afternoon tea (avoiding rich cakes and pastry).

6 ,, Poached egg on toast or spinach.
Milk and potash.

9 ,, Cup of soup.

#### 5*th Day*

8 A.M.—A few rolls of well-fried streaky bacon.
Toast and tea.

11.30 A.M.—Egg flip, or soup, or milk and potash.
Fruit.
1.30 P.M.—A small slice of tender roast mutton, or the eye of a tender mutton chop.
Vegetable.
Jelly.
4.30 „ Afternoon tea (avoiding rich cakes and pastry).
6.30 „ Fish.
Milk pudding.
9 „ Cup of soup.

*Fish.*—Whiting, haddock, sole, are the most easily digested fish, and as such should come first in the dietary. They can be steamed and served without any sauce, or stewed in a little milk and served with a white sauce. Nothing is nicer for an invalid than a really well-fried fillet of sole or small whiting, with a slice of lemon to squeeze over it.

*Steamed Fish.*—This is the lightest and simplest mode of cooking fish for an invalid.

1 filleted haddock, whiting, or sole.
A small piece of butter.
A pinch of salt and white pepper.
A squeeze of lemon juice.

Cut the fillets of fish into neat-sized pieces; grease a soup plate or muffin dish with a little butter, and place the pieces of fish on this.

Sprinkle with a little salt and white pepper if it is allowed, and squeeze over some lemon juice, which helps to keep the fish firm and white.

Cover the fish with a piece of greased white paper and then with a lid or basin. Place this over a pan half full of boiling water, seeing that the plate fits well on the pan.

Keep the water in the pan boiling so that there may be plenty of steam, and cook from twenty to thirty minutes, until the fish loses its clear, transparent appearance, and looks quite white.

If the pieces are thick, it is better to turn them while cooking. The liquid that is on the plate when the fish is cooked is the juice from the fish, and should be served with it. Serve the fish with a little plain cold butter, and a piece of plain bread or toast.

*Stewed Fish*

1 filleted fish—whiting, haddock, sole, plaice.
1 table-spoonful bread crumbs.
½ gill cold water.
1 gill milk.
½ oz. butter.
1 tea-spoonful chopped parsley.
White pepper.

Wipe the fish with a damp cloth and cut it into small neat pieces. Rinse out a lined saucepan with water, and place the pieces of fish at the foot.

Sprinkle over them a little salt and white pepper, pour in the milk and water; put the lid on the pan, and let the fish cook slowly by the side of the fire until it is ready, which will be in about fifteen minutes. Do not overcook, or the fish will be hard.

Lift out the pieces of fish on to the plate on which they are to be served, and keep them hot.

Add the bread crumbs and the butter to the water and milk in the pan. Stir over the fire for a few minutes until the bread crumbs swell and thicken the sauce.

Sprinkle in the parsley, and then pour this sauce over the fish.

*Baked Fish*

¼ lb. uncooked fish.
2 table-spoonfuls bread crumbs.
½ oz. butter.
1 egg.
½ gill milk.
Pepper, salt, and a little lemon juice.

Grease a small pie-dish with a little of the butter. Have the fish free from skin and bone, and cut it into neat pieces.

Lay half of these pieces at the foot of the pie-dish; sprinkle over them a little white pepper, salt, and a squeeze of lemon juice, and then put on a layer of bread crumbs. Next, put in the rest of the fish, seasoning, and more crumbs.

Beat up the egg in a small basin, add the milk to it, and strain this into the pie-dish. Put the rest of the butter in small pieces on the top, wipe round the edges of the pie-dish, and bake in the oven until nicely browned.

*Soups.*—The following recipes given for soups are those in which the ingredients are not too rich for the digestive capacity of the convalescent. They form a delightful change after the monotony of the beef teas and extracts. These recipes are very useful for the dietary of the aged (see p. 23).

*Broth—Mutton, Chicken, or Veal*

1 lb. neck or knuckle of mutton or veal, or 1 chicken.
2 pints cold water.
1 dessert-spoonful of rice.
1 tea-spoonful chopped parsley.
1 tea-spoonful salt.

Wipe the meat well with a damp cloth. Cut into small pieces and remove it from the bone, and take away as much fat as possible. Put meat, bones, water, and salt into a clean lined pan, put on the lid, and bring it very slowly to the boil.

Remove with an iron spoon all the scum that rises. Simmer slowly for four hours, skimming when necessary.

When cold, remove all the fat. Return it to a saucepan with the rice well washed; allow it to cook again for twenty minutes until the rice

is soft. Add the parsley at the last, and it is ready for serving.

Tapioca (Grôult's) or arrowroot may be used for thickening instead of rice.

*Rice Soup*

1 pint of mutton, veal, or chicken broth.
1 table-spoonful of Patna rice.
Yolk of an egg.
1 table-spoonful of cream.

Strain the broth and remove all fat from it; put it into a clean saucepan with the rice well washed, and boil until the rice is perfectly soft. Then rub all through a fine sieve, rinse out the pan, and return the soup to it.

Beat up the yolk of egg and cream with a fork; strain them into the soup, and stir carefully over the fire until thoroughly hot—but it must not be allowed to boil.

Tapioca or sago may be used instead of rice to thicken with.

Also three tomatoes might be boiled with the rice, and then rubbed through the sieve. This would make tomato soup.

*Rabbit Soup*

1 rabbit.
1½ pints cold water.
½ pint milk.
½ oz. butter.
½ oz. flour.
A few pieces of parsley.
1 bay leaf.
6 peppercorns.
½ tea-spoonful of salt.

Wash and clean the rabbit well. Let it lie in salt and water for half an hour. Lift out, dry it, and cut into joints. Cut the flesh into small pieces and chop the bones.

Put all these into a saucepan with cold water, with bay leaf, parsley, salt, and peppercorns. Bring to boil and skim well.

Simmer slowly from five to six hours; then strain through a fine sieve. Put the meat into a mortar; pound it well with a little liquid, and rub it through a wire sieve.

Rinse out the pan; melt in it the butter; add the flour, and mix till smooth. Then add the sieved meat, soup, and milk, and stir until boiling.

Boil for ten minutes.

*Game Soup*

1 bird (grouse, pigeon, woodcock, partridge), or the remains of game.
¼ lb. lean juicy meat.
1½ pints cold water.
½ tea-spoonful arrowroot.
A pinch of celery seed, pepper, and salt.

Wipe the game and meat with a cloth; shred the meat as for beef tea; cut the game up into neat pieces. Put all these into a lined saucepan, with cold water and seasoning; bring slowly to the boil, and skim thoroughly. Allow the soup to simmer for four hours. Strain and allow to cool, and then remove the fat.

Mix the arrowroot with cold water; add this to the soup. Boil for two or three minutes until the arrowroot turns quite clear.

*Fish Soup*

Small haddock or whiting, or piece of cod.
½ oz. butter.
½ oz. flour.
3 gills or 1 pint cold water.
1 gill of milk.
1 yolk of egg and ½ gill of cream.
1 tea-spoonful finely-chopped parsley.

Wash and scrape the fish very clean; see that there is no black skin lining the inside part. Remove the eyes.

Cut the fish across into several pieces, and put them in a lined saucepan.

Cover the fish with cold water, and add the salt. Bring to the boil and skim.

After the soup has boiled for a few minutes lift out a few nice little pieces of fish free from skin and bone, and reserve them for serving in the soup at the end.

Allow the rest to simmer slowly for ¾ to 1 hour. Then strain through a wire sieve, and rub some of the white pieces through.

Rinse out the pan the soup was cooked in. Melt in it the butter; add the flour, and mix these two smoothly together, being careful they do not brown. Then pour on the soup that has been sieved, and stir until boiling.

Beat the yolk of egg and cream and milk together; and, when the soup is off the boil, beat these ingredients into it; then strain through a fine strainer, stirring all the time. Do not let the soup boil after the yolk is added, or it will curdle. The pieces of fish that were reserved and the chopped parsley are now added.

## Meats

Of this class of food, poultry, game, and rabbits are the most easily digested. They can be served in the usual manner of roasting, boiling, and stewing. A few extra methods of preparation are here given.

Tripe and sweetbreads form most excellent dishes for persons of weak digestion, and only require to be more thoroughly known to be in common use (see pp. 20, 21).

*Broiled Chicken*

1 small chicken.
1 oz. butter.
Pepper and salt.

Prepare a young chicken for roasting; split it down the back, and lay it open, and take only half at a time. Rub the piece of chicken over with a little butter to keep the skin from cracking, and season with pepper and salt.

Grease the gridiron and make it thoroughly

hot. Lay the chicken on it with the cut side down to begin with; broil either on the top or before a clear fire for about half an hour. When cooked, lift on to a very hot plate, and rub the rest of the butter over it. Serve with rolls of bacon round it.

If broiling an older fowl it must be partially cooked first either by boiling or roasting.

*Chicken Soufflé*

Breast of chicken.
Cream, 1 gill.
Egg, 1.
Flour, ½ oz.
Salt, Pepper } to taste.

Skin the breast of the chicken; chop it finely; pound well in mortar; melt the butter; stir into the flour, and a table-spoonful of cream. Let it come to the boil; pour it over the pounded chicken; add seasoning, pound together, and rub through a wire sieve.

Switch the remaining cream until stiff; mix gently with other ingredients. Butter some cups; half fill with mixture; cover the cups with kitchen paper, and set in stewpan half full of boiling water. Steam for fifteen minutes. Serve with white sauce.

The boiling water should only come half-way up the cups while steaming.

*Chicken or Veal Panada*

¼ lb. breast of chicken or ¼ lb. fillet of veal.
1 tea-spoonful cold water.
1 table-spoonful cream.
A pinch of salt.

Wipe the meat and cut it into small pieces, free it from fat and skin. Put into a cup with a pinch of salt or cold water. Tie over it a greased white paper, and steam slowly for 1 to 1½ hours. Then lift it out, place the contents of the cup into a mortar; pound well and rub through the sieve.

Put the sieved mixture into a pan, add the cream, and heat thoroughly. This may be served on a piece of toast or even taken cold.

*Ragout of Fowl or Rabbit*

Take the wings and breast of a fowl or the back and legs of a rabbit; soak in hot water for five minutes, then put into a jar with salt and mace or peppercorns.

Cover with milk; cover the jar closely, set in an oven, and bake for three hours. Take out the meat. Pour the milk into a basin to cool, so that the fat may be easily removed. Warm in a cup set in hot water when required, or warm a little of the meat in the milk, and serve with thin toast or water biscuits.

*Tripe, Methods of Preparing.* — The best varieties of tripe are those known as the "blanket" (because it has a folded appearance), the dark variety known as the "monk's hood" —these are best for invalids on account of being more tender. The other sorts are known as the "book" and the "honeycomb."

If tripe is properly prepared it is a most delicately flavoured and easily digested article of food, and should be in much more constant use than it is at present. It requires, however, very careful cleaning and boiling. In Scotland we are unable to buy prepared tripe at the butcher's, so it is necessary to understand the whole process. Tripe sold in England has generally had a preliminary boiling.

First wash and scrub in several waters and scrape it with a knife, pulling away any pieces of fat from it.

Cut it into pieces and put it into a clean saucepan with cold water to cover it, and bring it to the boil. This is called blanching, and is repeated until the water in which the tripe is boiled loses all its heavy smell. This may require being done four or five times. Then rinse the saucepan well; put in the tripe with cold water to cover it, bring to the boil, and cook *slowly* for ten to twelve hours. If cooked too quickly the tripe will get hard. When sufficiently cooked, it ought to be so tender that it will pull easily to pieces when you try it with the fingers. Pour it out into a basin and cover it with the liquor in which it was cooked. This prevents it becoming hard and dry. It can then be made up in any way you wish.

The water in which tripe is boiled should never be thrown away, as it contains a certain amount of nourishment. It is sometimes served as an invalid jelly.

The tripe can now be stewed in various sauces for fifteen minutes:—

1. Tripe stewed in white sauce and onions, and served with sippets of toast.

2. Tripe stewed in a thick tomato sauce, and served with little rolls of bacon and croquettes of fried bread.

3. Tripe heated in a good curry sauce, and served with a border of rice.

4. *Scalloped Tripe.*—Lay a layer of prepared tripe in a buttered fire-proof china dish, then a layer of chopped mushrooms and seasoning such as pepper and salt. Add a gill of brown stock, and on the top sprinkle bread crumbs with little pieces of butter. Place in oven to get thoroughly hot and brown.

5. *Baked Tripe.*—Grease out a small pie-dish, and put the tripe with thin square pieces of bread and butter into this in alternate layers. The last layer should be bread with the buttered side up.

Beat up an egg in a basin until it is frothy, add it to half a gill of tripe liquor, and season with pepper and salt. Strain this into the pie-dish, and then wipe round the edge of the dish with a cloth.

Let the mixture stand for ten minutes until the bread gets thoroughly soaked.

Bake in a moderate oven for about fifteen minutes until nicely browned. Serve hot.

*Sweetbreads.*—This is another article of food that must be nicely prepared, or it is quite disgusting. The difficulty is to clear off all the connective tissue and fat from between the lobules.

Soak the sweetbread in cold water for one to two hours. Then put it into a saucepan with cold water to cover it; bring to the boil and let it boil for five minutes; then lift it out into a basin of clean cold water to cool it. This preserves the colour of the sweetbread; then with great care remove from it all the fat and skin, pulling them off with the fingers. It is now ready for cooking.

*Braised Sweetbreads.*—Prepare the sweetbreads as above. Braise them for 1½ hours —that is, place the sweetbreads unbroken into a saucepan containing a little onion, carrot, turnip, celery, savoury herbs and seasoning; add a little meat stock (just enough to cover the meat), cover with paper and a well-fitting lid.

At the end of the time take them out, brown in the oven, and serve on a piece of fried toast, or a bed of mashed potatoes. Pour a good sauce round them, such as sauce Italienne.

*Sauce Italienne*

1 oz. flour.
1 oz. butter.
1 bay leaf.
Onion and spring parsley.
1 gill sherry.
½ pint second stock.
Pepper and salt.

Melt the butter; fry the herbs and onions and flour a good brown colour. Add the sherry, and allow it to cook a few minutes; add the stock when boiling and allow to simmer for an hour. Strain the sauce before pouring round the sweetbreads.

*Stewed Sweetbreads*

1 heart sweetbread.
1 gill of white stock.
1 tea-spoonful arrowroot.
1 table-spoonful cream.
White pepper and salt.

Break the prepared sweetbread into small pieces of equal size, put them into a small lined stewpan with the stock, put on the lid, and allow it to simmer very slowly until tender.

When cooked, lift the pieces of sweetbread out, place them on a neat square of toast on a hot dish, and keep them warm.

Break the arrowroot in a small basin with a little cold water, and add it to the stock in the pan. Stir over the fire until boiling, and boil for five minutes so as to cook the arrowroot.

Add cream, and season to taste. Pour the sauce over the sweetbread, and serve very hot.

Lambs' sweetbreads are very delicate, and can be cooked in this way.

See also recipe in diabetic feeding (p. 30) for sweetbreads and tomatoes.

*Minced Beef*

½ lb. best rump steak.
½ pint cold water.
Pepper and salt.
Sippets of toast.

Choose the beef, and select a nice juicy piece. Either get the butcher to mince it, or, after removing all fat and connective tissue, put it through the mincing machine.

Never buy mince from the shop for an invalid, there is always too much fat in it.

Take a small lined stew-pan; put the minced beef into it with half the water and the seasoning.

Put the pan on the stove, and pound the meat well until it loses its raw appearance.

Then add the rest of the water, simmer gently by the fire for twenty minutes. Do not allow the meet to cook too quickly or it becomes hard. Remove any grease that may rise on it, and serve very hot.

Garnish the dish with some neat sippets of toasted bread.

*Meat Juice Mince*

½ lb. best rump steak.
1 small piece of butter.
Pepper and salt.

Take the meat and rub through the hair sieve until all the red juicy part has gone through; scrape the bottom of the sieve.

Melt a very small piece of butter in a small frying-pan, toss the meat juice in it for three or four minutes until it loses its red colour. Flavour and serve with toast. This looks just like mince; but as none of the fibre is present, it is very digestible. This meat juice mince can be made more easily digestible by omitting the butter, and adopting the following method:— Take the scraped juice and add a table-spoonful of beef tea or simple stock, and stir in an iron pan for three or four minutes, when the juice granulates. If an enamel pan is used the meat has a very unappetising appearance.

*Beef Quenelles*

¼ lb. beefsteak.
Bread crumbs.
Salt and pepper to taste.
1 egg.
2 table-spoonfuls stock.

Pound the beefsteak, half a teacupful of bread crumbs, salt and pepper, egg and stock, well in a mortar, rub through sieve, shape with table-spoons, and poach in shallow pan for ten minutes in boiling water.

Serve with sippets of toast, and pour a gravy made of thickened beef tea round the quenelles.

### Vegetables

As the nutritive value and digestibility of vegetables depend largely on their careful preparation, a short account of the best methods of preparing many of them is here given:—

*Spinach.*—One of the most easily digested vegetables, if properly cooked.

The preliminary difficulty in thoroughly cleaning spinach and the removing of the tough stalks are the two important points in the preparation of spinach. It also reduces enormously in cooking, and 1 lb. will make a very small dish.

Double the leaves lengthways and strip off the stalks. Then wash the spinach thoroughly in several waters until all the grit is removed.

Handle it as lightly as possible, as touching it too much causes the leaves to lose their crispness.

Put it into a saucepan without any water except that which adheres to the leaves; sprinkle it with salt, and put the lid on the pan.

Spinach is the only green vegetable which is cooked with the lid on the pan, as no water is used. Were the lid off, the spinach might burn from the evaporation. Cook until it is quite tender for stirring with a spoon, twenty to thirty minutes.

When ready, drain off the water in a fine wire sieve with a basin below. Then remove the basin, and rub the spinach through the sieve on to this; scrape the sieve well beneath. Return the spinach to a saucepan with a small piece of butter, pepper and salt; stir over the fire until thoroughly hot; add a squeeze of lemon juice or cream.

This can be eaten with small pieces of toast or fried bread.

If served with a poached egg on it, it will make a nice dinner for a delicate person.

*Boiled Lettuce.*—Wash a couple of lettuces thoroughly and remove any discoloured leaves, and let them lie in cold water for a short time.

Drain the water off, and put them in a pan of boiling water and a little salt.

Boil quickly for about twenty minutes, when they should be quite tender. Keep the lid of the pan off.

Remove any scum that rises.

When ready, drain and chop up the leaves on a board.

Return to the saucepan with a small piece of butter, pepper, and more salt if required.

*Tomatoes.*—These have most flavour when baked, but can be boiled or stewed.

Wipe them first and remove the stalks.

Put them on a greased baking-tin or fire-proof dish, add a little pepper and salt, and cover with a piece of greased kitchen paper. Bake for ten minutes until they feel soft. Lift on to a clean hot dish, and serve.

*Steamed Asparagus.*—Trim the asparagus, and steam by putting it in a jam-pot nearly filled with boiling water, placed in a large saucepan half full of boiling water and lightly covered.

The asparagus will take nearly an hour to cook in this manner.

Serve with a sauce made of 1 oz. of melted butter, 1 table-spoonful of cream, the yolk of an egg, and 5 drops of lemon juice. Stir in mixture for a few minutes over the fire until thoroughly warmed.

*Celery, Sea-Kale, and young Leeks.*—For celery, choose it when fresh and crisp; remove the coarse outside leaves, as they can be used for flavouring.

Put away the root; separate the stalks, wash and brush them well in cold water, and scrape off any brown or discoloured parts with a knife; cut the stalks equal lengths, tie them together with tape (string would cut through), and throw them into a basin of clean cold water, and allow them to soak in this for a few moments before cooking.

To cook:—Put into a saucepan of a freshly boiling mixture of equal parts of milk and water, and a little salt. Boil with the lid off for about half an hour, when it should be quite tender.

Drain well, remove the tape and serve on a small piece of toast. This is not meant to be eaten, but it is for the purpose of more thoroughly drawing the moisture of the vegetable.

All these vegetables can be served with a little well-made white sauce served round them. This sauce, unless properly made, is much better away, as butter sauce, as commonly served, is a compound of uncooked flour and milk.

*White Sauce*

¼ oz. butter.
¼ oz. flour.
1 gill of milk.
A squeeze of lemon juice.
A pinch of salt.

Take a small lined saucepan: rinse it out first with cold water to prevent the sauce sticking to the foot of it, and melt in it the butter over the fire, being careful it does not brown. Then add the flour, and mix with a wooden spoon until smooth; cook it for a minute or two over the fire to give it a glossy appearance. Draw the pan to the side of the fire, add the milk or fish stock, and then stir constantly over the fire until boiling.

Boil for two or three minutes in order to thoroughly cook the flour, and season to taste.

*Cauliflower.*—A young cauliflower with a firm, close head. Trim off the thick part of the stalk and nearly all the leaves. Split the stalk in opposite directions that the water may get in and cook it well. Wash in cold water, and let it lie in fresh water and 1 tea-spoonful vinegar to draw out any insects.

Have on the fire a deep saucepan three-quarters full of briskly boiling water; add salt to it, and put the cauliflower in head downwards.

Let it boil from twenty minutes to half an hour, until the flower feels tender, but is not broken up. Remove the scum as it rests; this, besides being dirty, would discolour the head. When ready, lift it out and drain for a minute or two on a sieve, and then serve with the white sauce, or white sauce and grated Parmesan cheese browned as on p. 30.

*Onions.*—Spanish onions are by far the best for eating as a dish, the flavour being not so strong.

When onions are plain *boiled* they are best served on dry toast without any sauce. A large Spanish onion takes about three hours' boiling to become tender.

*Baked.*—Place one or two Spanish onions in a baking-tin with a little butter: baste occasionally. When finished, they should be of a nice brown colour. They will also require about three hours' baking.

*Stewed.*—Place a large Spanish onion in a saucer at the bottom of the saucepan, and put sufficient equal parts of milk and water to reach the edge of the saucer. Keep the lid of the saucepan on tight, and let it steam for about 3½ hours until it is quite tender. The water from the onion will prevent the necessity of adding fresh water. Serve it with the milk in which it has been stewed.

*Plain Boiled Rice.*—Well wash some Patna rice in several waters until the last water looks quite clean. If there is a pot for steaming the rice, it is best to use this; but if not, boil the rice in a saucepan of boiling water containing salt, which is in the proportion of one tea-spoonful to the quart.

Boil quickly with the lid off, stirring it frequently with a fork to prevent it sticking to the pan. Cook from ten to fifteen minutes, until the grains will rub down easily when one is tested between the finger and thumb.

Strain through sieve or strainer, and finish the cooking by drying it—either by putting it into the saucepan by the side of the fire, or putting it on to a plate in a moderate oven.

While drying, stir lightly with a fork every now and then to keep the grains separate.

*N.B.*—The water in which the rice has been boiled contains the best part of the rice, so it should not be thrown away, but kept for the stock-pot.

*Boiled Macaroni.*—Break the macaroni into short lengths, and throw it into a saucepan of freshly boiling water with salt in it.

Boil quickly with the lid off the pan until it has thoroughly swelled and is tender. Stir occasionally to prevent it sticking. The time depends on the variety of macaroni—the large pipe will take about half an hour, the small much longer.

Keep it well covered with water.

When ready, drain; and this may either be served plain with meat, or it may be put back into the saucepan with enough stock to cover and allowed to stew for half an hour.

The pulp of a fresh tomato rubbed through a sieve may also be added to this.

### Food for the Aged

Owing to the failing intestinal activity incident to the later years of life, the digestive tract in old people has to be treated with more consideration than obtains in earlier years. This is especially true of those subjects whose "old age" has been induced by a manifest organic weakness in the kidney, stomach, heart, lungs, or other viscera, but it is also applicable to others in whom no such manifest organic disease exists.

The old adage "once a man and twice a child" applies very aptly to the feeding of the aged. It is impossible to lay down specific directions appropriate to the numerous conditions comprised under this heading, and all that will be attempted is to indicate a few guiding rules as to the choice and preparation of the most suitable forms of nourishment.

It may be stated as an invariable rule that the intake of food should be diminished as the general bodily activity decreases, and further, that red meats and rich oily dishes should give place to white meats and simple farinaceous foods. All foods must be in a form admitting of easy mastication.

*Milk and Cream.*—Cream is a capital substitute for other forms of fatty foods to which the patient has been previously accustomed. Milk should always form a large portion of the dietary, if it is digested. The tendency should be to increase the intake of milk as the vegetables are diminished.

If milk taken alone is too constipating, add a little saline water, such as Vichy or Vals. It is best always to take the chill off the milk before giving it to old people. Whey (p. 7) can be used as a substitute for milk, and with the addition of cream it forms both a pleasant as well as a nourishing food.

Milk is also combined with the various forms of gruels and farinaceous puddings.

Cocoa and milk and coffee and milk are also very good; and if these prove indigestible, the peptonised brands before mentioned (p. 10) may be tried instead.

*Soups.*—These, when made simply, are all permissible; but rich consommés, hare soup, turtle soup, etc. throw too much strain on the digestion to be advisable.

Chicken, veal, and beef teas (p. 13) may be used as beverages, and are most useful for giving to the patient last thing before retiring to bed, or a cup of "tea" left beside the bed to take if the patient is wakeful. In addition

to the soups already recommended for convalescence, the following slightly richer recipes are commendable; they are not too rich, and yet contain sufficient nourishment for them to form an important item in the diet:—

*Brunoise Soup*

1 carrot (young).
½ turnip (young).
2 leaves of celery.
Flower of small boiled cauliflower.
1 onion.
1 oz. butter.
1 pint of water.
1 pint milk.
1 tea-spoonful of salt and pepper.
2 oz. of stale bread, toasted.

Stew the ingredients, except the toast, together for one hour, then break the toast in pieces, add it to the rest, and stew all together for another hour. Pass all through the sieve and return to the stewpan to get hot.

*Lentil Soup*

Wash the lentils thoroughly in cold water, and add the lentils to water in the proportion of ¾ lb. lentils to a gallon of water or of second stock. Add pepper, salt, onion, turnips, carrot, and celery. Boil all for three or four hours. Pass through fine wire sieve or colander, put on a few minutes to heat, and send to table with toast cut in dice A little currie powder may be added if desired.

This soup is somewhat flatulent to those of weak digestion, but if made without the additional vegetables it will be found very acceptable to the most delicate stomach.

Pea soup served with dried mint, and haricot bean soup, can be made in the same way.

*Potato Soup*

1 lb. potatoes.
1 leek.
1 onion.
1 oz. of butter.
1 pint milk.
1 pint water.

Stew the potatoes, put them with leek, onion, and butter into a pint of boiling water in a stewpan. Boil until the vegetables are soft, then pass them through a sieve, adding a pint of hot milk to help the union. Put them all into the stewpan until it boils, then serve.

Serve with dice of fried bread.

*Rice and Tomato Soup*

1 pint of mutton, veal, or chicken broth.
1 dessert-spoonful Patna rice.
3 fresh tomatoes.
A little salt.

Put the broth into a clean lined saucepan, with the rice well washed, and the tomatoes wiped and cut in slices.

Boil slowly for half an hour, stirring occasionally; then rub through a hair or fine wire sieve. Heat again, season to taste, and it is ready for serving.

FISH

Fish are all allowable for old people. The oily fishes—such as salmon, herring, mackerel—are the only ones that are apt to disagree with digestion, unless taken in small quantities.

In addition to the usual methods of boiling, frying, and baking fish, already described, a variety of dishes may be made from this class of food that are found to be appetising as well as quite satisfying.

*Boiled Cod or Haddock Roes.*—Wash the roe well; then weigh it and tie it up in a piece of muslin or a pudding cloth. Put it into a saucepan or fish kettle, with enough boiling water to cover it. Add one tea-spoonful of vinegar and ½ tea-spoonful of salt to each quart of water.

Boil slowly, allowing twenty minutes to the pound, or until the roe feels quite firm to the touch. Lift out, drain well, and serve as much as will be required at one time.

It can be served with plain cold butter or white sauce.

*Grilled and Fried Roe.*—The roe must be boiled first. When it is cold, cut into slices half an inch thick with a sharp knife.

To grill, grease the grill or gridiron with a little butter, and make it quite hot. Lay the slices of roe on it and broil in front of a clear fire for five or seven minutes. Serve this very hot.

To fry, melt 1 oz. butter in a frying-pan; when smoking hot put in the pieces of roe. Fry them a nice brown colour on both sides. When ready, lift out and drain on a piece of double paper. Serve very hot.

*Fish Baked in Batter*

¼ lb. uncooked fish, haddock, or sole.
1 oz. flour.
½ gill milk.
1 egg.
½ oz. butter.
Pepper, salt, and lemon juice.

First make the batter; rub the flour through a wire sieve, and make it free from lumps, and put it into a basin. Beat up the egg with a fork and add it to the flour; beat with a wooden spoon until quite smooth and free from lumps. Then add the milk and beat for a few minutes longer: the more the batter is beaten the lighter it will be.

Have the fish free from skin and bone, and cut into small pieces. Lay these at the foot of a small greased pie-dish, and season with pepper, salt, and a squeeze of lemon juice. Pour the batter over, and put the rest of the butter in small pieces on the top.

Allow this to stand for a few minutes before cooking; this gives time for the flour in the batter to swell, and it will be lighter when baked.

Bake in a quick oven from twelve to fifteen minutes until well risen and nicely browned. Serve at once, as the batter quickly falls.

*Fried Haddock and Tomatoes*

1 dried haddock.
2 tomatoes.
1 small onion.
1 oz. butter.
Pepper, salt, and parsley.

Soak the fish for three hours; then skin it. Take out all the bones, and break up the fish into flakes. Slice the onion and tomatoes; chop up the parsley; sprinkle with pepper and salt, and cook all in the butter until quite soft; then add the fish, and cook for ten minutes longer.

Dish up in a border of boiled rice or mashed potatoes.

*Fish Soufflé*

¼ uncooked fish.
½ oz. butter.
½ oz. flour.
½ gill of fish stock or milk.
2 eggs.
Pepper, salt, and lemon juice.

First make a panada with the butter, flour, and fish. This is done by melting the butter in a small saucepan, adding the flour, and mixing until it is smooth with a wooden spoon. Then pour on the milk or fish stock, and stir until the mixture is thick and free from lumps and leaves the sides of the pan quite clean.

Scrape the fish down finely with a knife. Put the panada into a mortar with the fish, seasoning, and yolks of eggs. Pound well together and rub through a sieve.

Beat up the whites of eggs to a stiff froth and stir lightly into the fish mixture with an iron spoon. Pour into a greased basin which should be only half full. Cover with a greased paper. Steam for twenty minutes. When firm, lift it out and turn out on a hot plate.

*Curried Fish.*—A very nice way of having fish is to curry it and serve it with a border of rice. The following ingredients should be put into the curry:—

1 lb. fish.
1 apple or stick of rhubarb.
2 oz. of fat or butter.
1 small onion.
1 table-spoonful of curry powder.
1 table-spoonful of flour.
Salt and pepper.
A tea-spoonful of lemon juice or vinegar.

*Scolloped Fish*

¼ lb. cold cooked fish.
Bread crumbs.
1 oz. butter.
Pepper and salt.
Fish stock or milk.

Butter a scollop shell of fire-proof china; sprinkle on it a layer of bread crumbs; then a layer of fish broken up into pieces; some pepper, salt, and piece of butter. Cover this with more bread crumbs and bits of butter, and pour on a few drops of fish liquor, or milk. Bake ten minutes.

*Kedgeree*

Cold fish, ¼ lb., or dried haddock.
Rice, boiled, ¼ lb.
Butter, 1 oz.
Egg, 1.
Cayenne pepper; salt.

Wash and boil the rice; boil the egg very hard; break the fish into pieces, and carefully remove all the bones; take the egg, break off the shell, and cut the whites into small square pieces.

When the boiled rice is dry, melt the butter in a stewpan, add the rice, then the fish, the chopped white of egg, cayenne pepper, and salt. Mix them well together, and serve on a hot dish, and sprinkle the yolk of egg over it.

*Macaroni and Fish*

¼ lb. macaroni, well boiled.
¼ lb. cold boiled fish.

Mix the macaroni and the cold boiled fish well together with pepper and salt, half a pint of fish stock or chicken broth, and 1 ounce of butter.

Put the mixture in a flat china dish, sprinkle bread crumbs and a little piece of butter on top; when the mixture is quite hot and brown it is ready to serve.

*Vegetables and Fruit.*—Old people are very apt to give up vegetables owing to their supposed flatulent properties. This practice is serious, for if persisted in, some minor symptoms of scurvy not infrequently develop. Everything depends on the form in which the vegetables are administered and on the amount and nature of the ingredients of the meal.

A small quantity of potato should be taken every day, and also a certain amount of well-cooked vegetable. Spinach, stewed lettuce, stewed or baked tomatoes; vegetable marrow and cucumber, boiled, stuffed, or stewed, or served with grated cheese; celery, sea-kale, asparagus, leeks, the flower of cauliflower, large Spanish onions, green peas when young and quite tender, and French beans are all suitable. Uncooked vegetables, as partaken of in salads, are not very satisfactory, and are very apt to cause flatulence; so also are cabbage, greens, Brussels sprouts, turnips, parsnips, and old carrots. For methods of preparation, see pp. 22, 23.

Fruit taken in small quantity is also advisable. It is best to take it cooked, either stewed or baked, and eaten with cream.[1] This gets over the difficulty of eating a crisp apple when the teeth are imperfect.

In stewing fruit, if cane sugar is added by the cook the resultant product is very prone to turn acid. It is therefore better to neutralise the acidity with an alkali rather than to attempt to mask the flavour with sugar. Thus to each pound of fruit add as much bicarbonate of soda as will lie on a shilling. The bitterness of the fruit will be gone and the natural flavours of the coulere will become apparent, which is usually quite sweet enough. If this simple plan is carried out, many old people can eat and enjoy stewed fruit without feeling that they will have to suffer from acidity afterwards.

If there is a desire for sweetness, saccharine can take the place of sugar. The usual rule, however, will be found to be that the desire for sweetness disappears, and that food with a relish has to be provided instead

*Alcohol.* — A small amount of (measured) alcohol with food is often beneficial. A nourishing stimulant to be highly recommended is an ounce of dry cherry brandy mixed with a wineglassful of cream.

### Cookery in Diabetes

As a special knowledge of cookery is required to enable the physician in cases of diabetes to frame a diet suited to the physiological capacities of the individual, and at the same time to make the necessarily restricted diet more readily tolerated, the points of practical importance in the selection and manner of preparation of individual food-stuffs is here appended. For an account of the principles which must guide the physician in forming the diet the reader is referred to the articles "Diabetes" and "Glycosuria." The principles followed in framing these menus have been to make the food as appetising and varied as possible, while the important articles of food, starch and sugar, have been excluded and their place taken by the hydrocarbon fat, so that the body may not lack entirely the "fuel" food.

If the excess of fat in the food should give rise to dyspepsia, a small quantity of brandy and water after the meal aids digestion.

First, it is advisable to give a list of the articles that may or may not be eaten, as on this the menus are framed.

#### Food

| *May eat* | *May not eat* |
|---|---|
| Butcher meat of all kinds except liver. | Sugar in any form. |
| Tongue, ham, bacon, or other smoked, salted, dried, or cured meats. | Wheaten bread and ordinary biscuits of all kinds. |
| Poultry. Game. | Toast. Rice. Arrowroot. Corn-flour. Oatmeal. Sago. Tapioca. Macaroni. Vermicelli. |
| Fish of all kinds, fresh, salted, or cured. Shell fish, except the bodies of lobsters and crabs. Mussels. | Potatoes. Carrots. Parsnips. Beetroot. Peas. Spanish onions. |
| Animal soups, not thickened with any starchy materials. Beef teas and broths. | Pastry and puddings of all kinds, and honey. |
| Eggs dressed in any way. | All fruit, fresh and preserved, except lemons and unripe fruit. |
| Cheese. Cream cheeses. Butter. Cream. | Liver. |
| Greens. Spinach. Turnip tops. French beans.[1] Brussels sprouts.[1] Cauliflower.[1] | Oysters. |
| Broccoli.[1] Cabbage.[1] Asparagus.[1] Seakale.[1] Vegetable marrow. Lettuce. Cucumber. Tomatoes. Mushrooms. Mustard and cress. Watercress. Endive. Spring onions. Leeks. Celery. Rhubarb. Pickles. | Cockles and mussels. |
| Oil. Vinegar. | |
| Savoury jelly. Jellies and custards sweetened with saccharine. Blanc-manges made with isinglass or gelatine. | |
| All nuts except chestnuts. Olives. | |

[1] Preferably not later than the mid-day meal.

Food—*continued*

[1] These should be boiled in a large quantity of water.

#### Beverages

| *May take* | *May not take* |
|---|---|
| Tea. Coffee. Cocoa from nibs. Dry sherry. Claret. Dry Sauterne. Burgundy. Chablis. Hock. Brandy. Whisky and other unsweetened spirits. Unsweetened aerated waters. Milk (limited to 1 or 1½ pints daily). | Milk, except in limited quantities. Sweet ales. Porter. Stout. Cider. All sweet wines. Port. Tokay. Champagne. Liqueurs. Fruit juices and syrups. Cocoa and ordinary chocolate. |

In following out these tables we see that cream has practically to take the place of milk, cream being almost free from lactose. Sac-

charine and specially prepared glycerine are available as ready and safe sweetening agents to take the place of sugar: saccharine, being intensely sweet, is only required to be used in very small quantities. Fish and vegetables are cooked and served with a liberal amount of butter. Salmon, herrings, mackerel, sardines in oil, pâté de fois gras, cream cheeses, and rich sauces made with eggs, oil and cream, are specially useful in providing a fair supply of fatty foods.

The various preparations of diabetic breads and biscuits in the market are made from almonds and other nuts, bran, gluten, casein, etc. These can be obtained direct from the makers (Blatchley, London, and Callard & Co., London), or the ground nuts, etc. in the form of flour can be purchased and made up in the patient's own house. It should be noted that all reliable diabetic food-stuffs are expensive.

Almond flour made into biscuits is very satisfactory; the oil in their composition renders them a very desirable food in diabetes

Almonds in a solid form, unripe fruits and rhubarb, sweetened with saccharine or neutralised with an alkali, have to take the place of ripe fruit.

The deprivation of starch is the greatest trial to all patients, and to many this restriction becomes so irksome that the strictness of the dietary has to be relaxed. However, there are at the present time a great number of substitutes which can be tried to make bread and biscuits, some totally free from starch; others, again, containing small quantities are apt to cause indigestion, but when broken into a powder the process of digestion is not interfered with. Cocoa-nut, hazel-nut, and pine seeds are also available in powder forms, and these are made into biscuits, which are flavoured in various ways.

Bran flour is another product which, if carefully prepared, is entirely free of starch; this can be bought by the pound and made into bran cakes or bread. Biscuits can be procured, either plain or mixed with almonds, cocoa-nut, etc. These latter are certainly very palatable.

Gluten bread in slices, and dinner rolls, are other almost starch-free articles; they are fairly satisfactory, and are quite edible when eaten with butter.

Gluten flour or farina contains, in good specimens only, a small quantity of starch, about 10 per cent. French gluten farina is one of the purest varieties; the ordinary makes contain 25 to 42 per cent of starch; this can be made into loaves, girdle and oven scones.

A special form of brown bread, which is very like ordinary brown bread, but contains no starch, is now prepared by Messrs. Callard.

Casein biscuits are very palatable.

Soy flour and biscuits are not at all palatable, and in addition contain generally 24 per cent of starch (Kinch). Most aleuronat biscuits are almost inedible.

Every medical man should test all these articles to see if they are adulterated with starch. The presence of starch can easily be detected in food by applying a few drops of a solution of iodine in water, when the starch will immediately turn a violet-black.

Torrified or much-toasted bread is recommended by some writers, who seem to think that by evaporating the water from a piece of bread, some of the starch is also got rid of; the fact is, that well-toasted bread contains nearly twice as much starch and dextrine as equal weight of ordinary bread.

*Almond Cake*

1 oz. almond flour.
2 eggs.
Salt to taste.

Beat the whites of the eggs to a stiff froth, add the almond flour, and beat well together. Spread out into a buttered baking-tin about half an inch thick, and bake in a moderately quick oven from fifteen to twenty minutes.

The mixture should be done quickly and baked as soon as the ingredients are mixed.

These cakes are much improved by adding a small quantity of gluten flour.

*Cocoa-nut or Almond Cakes*

4 oz. desiccated cocoa-nut or almond flour.
1 pinch of saccharine.
German yeast.
1 egg.
2 tea-spoonfuls cream.

The almond flour is mixed into a paste with a little water and the German yeast. Allow the mixture to stand in a warm place for twenty minutes; then add the egg, two tea-spoonfuls of cream, and water sufficient to work the mixture. Divide into cakes and bake in a *quick oven.*

*Gluten bread in slices*, cut into small pieces, soaked in butter and toasted or fried, is very palatable, and will be found a useful article in the preparation of many dishes.

*Bran Cakes*

4 oz. prepared bran flour.
1 tea-spoonful of bicarbonate of soda.
5 eggs.
¼ pint of warm milk.
2 oz. butter.

First mix the bicarbonate of soda with the bran flour; then beat up 2 ounces of butter in a warm basin, shake into it the dry ingredients, beating with a spoon all the time. Next beat up the five eggs in a separate basin before the fire until quite warm, and stir them gradually into the mixture of bran flour, soda, and butter. Beat up all well for ten minutes, and add gradually the warmed milk. Place in buttered

tins, and bake in a brisk oven for about ten minutes. The cakes are done when they will turn out of the tins quite easily. This will make about five cakes the size of buns. The cakes can be cut into slices, buttered, and toasted.

Even more palatable cakes can be made with an equal mixture of bran and almond flour. The bran reduces the excessive richness of the almond flour and gives a pleasant flavour.

*Bran Bread*

½ lb. bran (prepared).
2 oz. almond flour.
3 oz. butter.
6 eggs.
½ pint milk.
2 tea-spoonfuls of bicarbonate of soda.
1 tea-spoonful of tartaric acid.

Place the butter in a basin and beat it to a cream; then add the almonds and well beat, add the eggs one at a time. Partly mix in the bran before adding the milk. Well mix the whole together, and place it in a well-buttered tin and bake for an hour in moderate oven.

*Gluten Bread*

1 lb. gluten flour.
¼ lb. of prepared bran.
1 oz. yeast.
1 oz. ground almonds.
2 eggs.
Pinch of salt.

Make a sponge as for ordinary bread and set it to rise. This will take about three-quarters of an hour. Then bake.

The addition of a little ground almonds and two eggs is an improvement.

There are a few forbidden articles of food which, when prepared specially, are quite allowable to this class of patient:—

Sponge cakes, made from almond flour and sweetened with glycerine, are excellent.
Gluten macaroni.
Gluten vermicelli.
Cocoa, when specially prepared.
Marmalade, made with gelatine, glycerine, and orange rind; it is an attractive-looking compound, and a great boon to the diabetic's breakfast table. The flavour is somewhat peculiar, due to sweetness from glycerine.
Chocolate, specially prepared, makes an excellent sweetmeat, and also a very good cup of chocolate. It can be bought unsweetened, and flavoured with vanilla or sweetened with saccharine.
Glycerine jujubes can also be procured.

*Menus—First Day*

*Breakfast*

Tea and cream.
Whiting fried in butter. Squeeze lemon juice when served. Savoury omelet.
Gluten toast. Brown diabetic bread. Butter.

*Lunch*

Home-made lemonade sweetened with saccharine.
Vegetable marrow, stuffed. Cold ham.
Cream cheese. Almond and bran biscuits. Gluten rolls.

*Dinner*

Clear soup with mixed vegetables.
Steamed halibut. Slices of lemon.
Fillet of beef and cauliflower.
Green apples stewed, sweetened with saccharine. Cream.
Whipped jelly.
Cheese. Biscuits (made with gluten flour).
Pancakes (made with gluten flour, milk, eggs, sweetened with glycerine).

*Stuffed Vegetable Marrow.*—Small marrows make an excellent dish, boiled and stuffed with the stuffing of mushroom forcemeat. The marrow should be first peeled very slightly; then cut long-way into three slices, the pips removed, and the interior filled with the forcemeat. The forcemeat should be made hot before it is placed in the marrow; if not, the marrow will be cooked before the stuffing is heated through. The marrow should be placed in boiling water and boiled until tender; this takes about twenty minutes to half an hour.

*Mushroom Forcemeat*

1 lb. mushrooms.
½ tea-spoonful lemon juice.
Two hard-boiled eggs.
1 oz. butter.

The mushrooms after being cleaned should be chopped and fried in the butter; lemon juice should be added before they are chopped in order to preserve the colour. Add two hard-boiled eggs to the mixture, and rub the whole through a wire sieve while hot.

When hot this mixture is moist, but on standing gets hard.

*Savoury Omelet*

Parsley, 1 tea-spoonful chopped.
Eggs, 3.
Butter, 2 oz.
Herbs, savoury, Pepper, Salt } for flavouring.

Melt the butter in a frying-pan; beat up the eggs thoroughly; add a little pepper and salt, parsley and herb; pour the beaten-up eggs into the frying-pan as soon as the butter begins to frizzle, and with a table-spoon keep scraping the bottom of the frying-pan in every part. Go on scraping until two-thirds of the mixture have become lumpy.

Now lift the pan a little off the fire, push the omelet into half the frying-pan. When nearly set, hold the pan in a slanting direction in front of the fire; and, as soon as set, slide the omelet

from the pan on to a hot dish and serve at once.

This may be varied by addition of a little cooked fish, grated cheese, tomato, ham, crab.

*Second Day*

*Breakfast*

Tea or coffee with cream.

Toasted sardines on fried gluten bread. Boiled egg.

Bran bread. Butter. Marmalade.

*Lunch*

Fried bacon served au purée of spinach.

Cauliflower au gratin with black butter sauce.

Gorgonzola cheese. Gluten dinner rolls.

Slice of almond, pound cake (Messrs. Callard's).

*Dinner*

Tomato purée.

Steamed sole. Sauce Hollandaise.

Boiled fowl.

Carrageen jelly.

Stuffed tomatoes.

*Cauliflower au gratin.*—This makes a nice course by itself. The cauliflower should be boiled until quite tender. While boiling, remove any scum that rises to the top.

When ready lift it out, drain for a minute or two on a colander, and serve with black butter sauce poured over it. Sprinkle the sauce with Parmesan cheese.

*Sauce Hollandaise*

Yolks of 4 eggs.
2 oz. fresh butter.
½ gill of cream.
Pinch of pepper, salt, nutmeg.
1 tea-spoonful tarragon vinegar.

Place ingredients into small bowl; stand it into large pan on the fire containing boiling water. Briskly whisk the sauce over the fire until it begins to thicken and presents a rich, smooth, creamy appearance. Great care is required to prevent it from curdling; if, however, this should happen, add yolks of two more eggs.

*Stuffed Tomatoes*

3 ripe tomatoes.
½ oz. of butter.
1 spring onion.
½ tea-spoonful chopped parsley.
1 pinch mixed herbs.
2 mushrooms, or a tea-spoonful Parmesan cheese.
Crumbs of 1 almond biscuit.
Pepper and salt.

Cut off the stalks, leaving a hole the size of a threepenny bit. Squeeze out the juice and pips. To the juice and pips add the onion chopped, the chopped parsley, mixed herbs, and the mushrooms or cheese. Melt the butter and fry the mixture. Then add sufficient almond biscuit crumbs to make the whole into a moist paste.

Fill the tomatoes with the mixture until they resemble their original shape. Put a few crumbs and a little piece of butter on the top of each.

Place the filled tomatoes in a baking dish and bake until tender, and moisten with a little oil or butter. Serve in a dish with fried parsley round.

*Carrageen Jelly or Irish Moss.*—Irish moss is a seaweed, and is collected on the northern shores of Ireland. Its nutritive value is slight; it contains mainly mucilage, and also some iodine and sulphur. It should be well washed and soaked for some hours previous to cooking. The flavour is somewhat peculiar, but delicate, and to some very palatable.

1 oz. Irish moss.
1 quart cold water.
Lemon juice.
Saccharine to taste.

Wash the Irish moss well, and soak it for several hours in cold water. Then put into a saucepan with one quart of cold water and simmer slowly for from four to five hours. Strain, add the lemon juice, and sweeten to taste. Pour into a wetted mould and set aside until cold.

*Third Day*

*Breakfast*

Tea or coffee with cream.

Scrambled egg on toasted brown bread. Broiled kippered salmon.

Gluten flour bread. Butter.

*Lunch*

Cocoa nibs and cream.

Chicken panada and green peas (see p. 20). Potted head.

Imperial cheese. Gluten cracknels. Gluten flour scones.

*Dinner*

Chicken broth (as p. 18). Thicken with almond flour.

Fish soufflé (p. 25).

Curry and French beans. (The basis of this is cocoa-nut, green apples, or green rhubarb.)

Gluten and almond baked pudding. (Make as semolina pudding, using gluten and almond flour—p. 16.)

Savoury eggs.

*Sardines.*—There are various forms in the market. The smallest fish are the best. They can be procured smoked, or preserved with tomatoes. If rolled in slices of streaky bacon and fried, they form a very appetising dish.

*Girdle Scones*

½ lb. of gluten flour.
½ oz. of butter.
½ tea-spoonful of soda.
½ tea-spoonful of cream of tartar.
Salt or saccharine to taste.
½ tea-cupful of butter milk.

Rub butter into the flour and other dry ingredients; make into a soft dough with milk. Form into a scone, or little breakfast rolls or twists, and bake in oven for fifteen minutes.

*Savoury Eggs*

3 hard-boiled eggs.
¼ gill of cream.
½ oz. of butter.
1 table-spoonful of cheese (Parmesan).
1 small piece of onion, chopped.
Pepper.
Salt to taste.
Mustard.
1 tomato.

Divide the eggs in half; take out the yolks.

Mix together the yolks, cream, cheese, butter, onion finely chopped; pepper, salt, and mustard to taste. Fill the white parts of eggs with the mixture.

Cut six rounds of brown bread; butter them; on each piece lay a slice of tomato, and on this half of a filled egg. Garnish the dish with cress.

*Almond Pudding*

2 eggs.
¼ lb. of almond flour.
¼ lb. of butter.
2 tabloids of saccharine dissolved in a table-spoonful of brandy.

Warm the butter; beat in the almond flour and the yolks of the eggs, adding the dissolved saccharine. Whisk the whites to a stiff froth, beat altogether. Put into small cake moulds, bake in a quick oven, and serve with a little hot sauce made with dry sherry and saccharine.

Cocoa-nut pudding can be made in the same way.

*Fourth Day*

*Breakfast*

Tea, coffee, or cocoa from nibs.
Grilled kidney and bacon.
Crab omelet.
Bran bread and cocoa-nut biscuits.

*Lunch*

Lime juice and aerated water. (Unsweetened Montserrat lime juice.)

Anchovies on fried gluten bread slices; if cold, a spoonful of cream on the top.

Cold meat and tomato salad.

Pâté de foie gras, with bran biscuits.

*Dinner*

Soup Julienne.
Salmon.
Mayonnaise sauce and cucumber.
Sweetbreads and tomato.
Junket.
Mushrooms au gratin.

*Tomato Salad.*—Slice up two perfectly firm and ripe tomatoes; sometimes it is necessary to remove the core and pips. Add a tea-spoonful of oil and two tea-spoonfuls of vinegar, and sprinkle on a little pepper and salt.

*Mayonnaise Sauce*

Yolks of 3 eggs.
½ pint best salad oil.
½ gill tarragon vinegar.
Pepper and salt.

In a round basin begin stirring, with a wooden spoon, the yolks of the eggs, pepper and salt, until they are quite mixed. Then add, drop by drop, the salad oil and the vinegar, stirring all the time in the same direction; if added quickly the egg curdles. When finished, the sauce must have a creamy appearance.

*Sweetbreads and Vegetables.*—(For the preparation of sweetbreads, see p. 21.) The prepared sweetbreads are first stewed in milk, then rolled in slices of fat bacon, and placed in the oven for a quarter of an hour.

The bacon is then removed; the sweetbreads are cut in slices, and grated Parmesan cheese is shaken over them. They are again placed in the oven and braised in a rich brown gravy.

Served on croûtons of gluten bread, in the centre of which can be placed stewed tomatoes, green peas, fresh French beans, stewed mushrooms.

*Mushrooms au gratin*

10 mushrooms.
Piece of onion.
1 tea-spoonful chopped parsley.
1 salt-spoonful of thyme, fresh.
„ „ dried.
Pepper, Salt, Lemon juice } to taste.
Almond biscuit crumbs.
1 oz. butter.

Select ten cup mushrooms about the same size. Peel the mushrooms very carefully without breaking them; cut out the stalks close down with a spoon, and scoop out the inside. Peel the stalks, and chop them up with the scooped-out portion; add the onion, parsley, and thyme. Fry all this in the pan with a little butter. Add sufficient biscuit crumbs to make the whole into a moist paste, and season to taste. Bake in the oven until the mushrooms are quite tender. Serve with some nicely-fried parsley round it.

*Fifth Day*

*Breakfast*

Tea, chocolate, coffee, and cream.
Findon haddocks, stewed in milk or broiled.
Poached eggs on spinach.
Almond loaves. Gluten cracknels. Butter.

*Lunch*

Cocoa nibs.

Potted herrings. Stewed cucumber and cheese sauce.

Stilton cheese. Pine biscuits. Toasted gluten flour bread.

*Dinner*

Purée of lettuce or spinach.
Turbot and tartar sauce. (See *infra.*)
Roast chicken and water-cress.
Blanc-mange. (See p. 16, using saccharine instead of sugar.)
Tarragon creams.

*Stewed Cucumber and Cheese Sauce.*—Cut a fresh cucumber in pieces about 4 inches long. Boil until tender.

Pour over the cucumber, when cooked, the following cheese sauce.

Make the sauce in the same way as sauce Hollandaise (p. 29), omitting the tarragon vinegar, and add three table-spoonfuls of grated Parmesan cheese before serving.

*Tartar Sauce*

2 table-spoonfuls of Mayonnaise sauce.
1 tea-spoonful chopped parsley.
A small piece of very finely chopped shallot.
1 tea-spoonful of mustard.

Mix these ingredients well together and gently heat.

*Tarragon Creams*

2 eggs.
¼ pint cream.
Pepper and salt.
2 tea-spoonfuls chopped tarragon.

Put into a basin one white and yolks of two eggs, the cream, and a little white pepper and salt. Beat up with a fork until smooth, add a little chopped tarragon.

Butter some little moulds, sprinkle the top with chopped tarragon and truffles.

Pour in the mixture, and stand the moulds in a stewpan of boiling water reaching to three-quarters height of the moulds.

When the water boils, draw the pan to the side of the stove and poach for about twenty minutes till the creams are set.

Turn out on a warm dish, and serve with sauce Hollandaise round them.

*Sixth Day*

*Breakfast*

Tea, coffee, with cream.
Cutlets of egg, ham, or crab. Camp pie.
Gluten and bran bread. Butter. Marmalade.

*Lunch*

Fish roe soufflés.
Smoked tongue and salad.
Cheese, with almond biscuits.

*Dinner*

Cock-a-leekie.
Whitebait. Lemon sliced. Brown bread and butter.
Roast lamb. Mint sauce. Asparagus.
Gooseberry fool.
Hot caviare on toast. Gluten bread.

*Fish Roe Soufflés.*—Take six roes of fresh herrings; blanch and pound them, then flavour with salt, pepper, pounded mace, and nutmeg. Add half ounce butter and yolks of two eggs beaten well together. Whisk the whites of six eggs into a stiff froth; mix the same with the roes, and bake in ramequin cups for about five minutes.

Serve immediately the soufflés are removed from the oven.

*Meat and Hot Water, or "Salisbury" Diet*

Great care and consideration are called for, both as to the recommendation of this diet and the best means of giving effect to it. The writer recently saw a patient who came complaining of intense weakness and loss of flesh consequent on a course of treatment, lasting nine weeks, carried on "by correspondence." The patient had been instructed to take a moderate amount of exercise, including golf, and was greatly surprised to find that not only was exercise impossible, but at times she had hardly strength to stand. Very special precautions are always necessary in stout subjects.

The value of the treatment is undoubted in some cases of indigestion and chronic auto-intoxication associated with it. Further reference is made to it in other articles (*vide* "Gout," "Indigestion," "Obesity," "Sprue").

*Meats and Beef Juices.*—At first the patient begins with *lean meat or beef* in mince or cakes. However, if the patient is unable to assimilate solid food, Carnick's liquid peptonoids, peptonised home-made beef teas and essences, Valentine's, Wyeth's beef juice, Bovril, Vimbos, Brand's beef juice or meat jelly, may be used instead. Gradually the patient begins to take lean mutton cakes and the white meat of chicken. The white of an egg raw, lightly boiled, or poached, is also allowed.

The following directions will be useful in the preparation of the muscle cakes:—

*Lean Meat Cakes.*—The beef should be used from well-grown animals, and steaks cut from the centre of the round are the best for this purpose. The beef pulps can be prepared in the following manner:—All the fat, fascia, and connective tissue and bone are removed, and the meat is placed on a board or in a chopping tray, and is shredded down with a blunt knife; the pulp is then scraped together with a spoon, the result is that all the tough fibrinous parts remain behind. The scraped pulp is lightly moulded by the hand into cakes from half to one inch thick, and slowly broiled over a clear fire free from smoke. When cooked, serve on a hot plate with a little butter, and season to taste with pepper and salt. The flavouring may be varied by using Worcestershire or Halford sauce, mustard and horse radish, or lemon juice. A small quantity of celery is also permissible.

*Drinks.*—The patient is usually advised to

drink from one to two pints of hot water (110° to 120° F.) at one and a half hours before each meal, and half an hour before retiring. If the water is drunk slowly the stomach will not be distended, and the patient will not experience any sense of discomfort. If plain hot water is nauseating, it can be made more palatable by the addition of sal volatile, ginger, or lemon juice. When there is intense thirst, a little chloride of calcium or nitrate of potash will make the water a much better thirst-quencher. If constipation is present, a tea-spoonful of magnesium sulphate can be added to the water.

When the strictness of the diet is first relaxed, the following rice scones, preferably taken hot, are very palatable and easy of digestion.

*Rice Scones.*—Boil a teacupful of whole rice for 2½ hours. To the rice add quarter of a teacupful of sifted flour, a tea-spoonful of baking powder, and sufficient water to make a thick batter; then stir in a pinch of salt and the white of one egg well beaten up. Pour the mixture into well-greased cake-tins and bake in an oven for thirty minutes. To be eaten with butter and more salt if desired.

Oysters are a welcome addition to the monotony of the diet, and may be prepared as follows:—

*Oysters.*—Panned.—Take six deep-sea oysters, place them in a colander and pour cold water over them. Drain for ten minutes. Place the oysters in a very hot iron pan; add salt, pepper, a small piece of butter, and a tea-spoonful of meat stock. Cook for a few minutes, and serve the oysters garnished with a thin slice of lemon.

Broiled.—Take six large oysters. Lay them on a board and dry them, season with salt and cayenne pepper. Have a gridiron thoroughly heated; place the oysters on the gridiron and brown them on both sides. Place the oysters on a very warm plate, and pour round them a little heated beef juice and a little melted butter.

**Invasion.**—The onset of a disease (*e.g.* smallpox) or of a physiological process (*e.g.* menstruation), also the extension of a morbid process to an unaffected part of the body.

**Inversion.**—The turning of a hollow viscus inside out (*e.g.* inversion of the uterus); also the incomplete development of an organ, giving it the appearance as if it were turned inside out (*e.g.* inversion of the bladder); also the transposition of parts (*e.g.* inversion of the viscera, or heterotaxy). *See* LABOUR, INJURIES TO THE GENERATIVE ORGANS (*Acute Inversion of the Uterus*); BLADDER, INJURIES AND DISEASES (*Malformations, Ectopion Vesicæ*); TERATOLOGY (*Heterotaxy* or *Transposition of the Viscera*).

**Invertase.**—The soluble ferment of yeast, capable of converting sucrose into glucose.

**Invertins.**—Enzymes capable of splitting up sugars, *e.g. Invertase* (*q.v.*). *See* ENZYMES; PHYSIOLOGY, PROTOPLASM (*Enzymes or Zymins*).

**Involucrum.**—A sheath, capsule, or investment of an organ or part, especially the sheath of new osseous tissue round a sequestrum of necrosed bone. *See* BONE, DISEASES OF (*Suppurative Osteomyelitis, Repair*).

**Involution.**—The folding in of developing parts, especially membranes, in Embryological processes (*see* EMBRYOLOGY); the return of an organ or part to its normal size and characters after a temporary hypertrophy (*see* PUERPERIUM, PHYSIOLOGY, *Involution of the Uterus*); also the retrograde changes in the organs in old age.

**Iodalbin.**—An organic iodine or iodo-proteid compound, consisting of a reddish powder, almost tasteless, insoluble in water and alcohol but soluble in alkalies; said to be readily assimilable and free from the objectionable features of the inorganic salts. *Dose*—5-10 grs.

**Iodic Acid Test.**—A chemical test for morphine. *See* MORPHINOMANIA AND ALLIED DRUG HABITS.

**Iodides.** *See* IODINE.

**Iodine.** *See also* DRUG ERUPTIONS (*Iodine and the Iodides*); KIDNEY, SURGICAL AFFECTIONS OF (*Actinomycosis, Treatment*); LIVER, DISEASES OF (*Cirrhosis, Treatment*); PHARMACOLOGY; PURPURA (*Symptomatic*); SYPHILIS (*Treatment*); TOXICOLOGY (*Iodine*).—IODUM. Symbol, I; atomic weight, 125·9. Obtained from seaweeds and from mineral iodides and iodates. It occurs as small dark-coloured rhombic prisms, which give off a violet vapour when heated. It is soluble 1 in 5000 of water, and readily in alcohol, ether, chloroform, and solutions of potassium iodide. *Preparations*—1. Liquor Iodi Fortis. Strength, 11¾ per cent of iodine. Corresponds to Linimentum Iodi, B.P. 1885. 2. Tinctura Iodi. Strength, 2½ per cent. *Dose*—2-5 m. 3. Unguentum Iodi. Strength, 4 per cent.

Iodine is a very powerful antiseptic and irritant. The latter action is the one chiefly made use of in medicine. It is applied to the skin as a counter-irritant in pleurisy, in rheumatism, in arthritic affections of various kinds, in periostitis, in chilblains, in chronic glandular enlargements, in pleurodynia, and in a host of conditions, too numerous to mention, where an active local application is required. For most patients the liquor will be found to be too strong in effect, and the tincture too weak. A mixture containing equal parts of each is a favourite application; but wide variations in reaction are observed, and whatever preparation is employed, it should be used sparingly at first until the individual susceptibility has been ascertained.

For use over exposed parts of the body a colourless alcoholic solution of iodine, strength 1 in 40, prepared by decolorisation with strong solution of ammonia, has been recommended, but it is very weak in action and can have little real effect as a counter-irritant. Iodine should not be applied to the skin during the progress of any acute inflammatory condition, nor when there is fever. The official tincture may be injected into a hydrocele sac after tapping so as to set up the formation of obliterative adhesions. This proceeding causes the most exquisite agony, and should either be performed under a general anæsthetic or should be preceded by the injection of a solution of cocaine. Morton's fluid, which consists of iodine 10 grains, potassium iodide 30 grains, and glycerin 1 fl. oz., was formerly employed as an injection for spina bifida, but it is quite unreliable and has been almost entirely abandoned. The injection of iodine into the pleura for hydrothorax and empyema, and into joints and abscess cavities, has also been given up on account of the risks of poisoning from absorption. As an antiseptic, iodine has been used in ulcerative stomatitis. In unhealthy conditions of the teeth with a purulent discharge from between the teeth and gums, the direct application of the tincture is particularly beneficial; it has also been employed in ozæna and in nasal catarrh. In chronic pharyngitis Mandl's paint, which contains 6 grains of iodine to the ounce of glycerine with 20 grains of potassium iodide to effect solution, is of very great value. The tincture, and sometimes even the liquor if it can be borne, may effect a cure in ringworm when other means have failed. 20-30 m. of the tincture to 2 pints of boiling water, as an inhalation, has been recommended for chronic fibroid phthisis and other chronic chest conditions. Recently a solution of iodine with potassium iodide has been advocated as a simple and efficient means of sterilising catgut for surgical purposes. From time to time iodine has been administered internally for the treatment of diseases of obscure pathology, but with no very marked success. In conditions associated with enlargement of lymphatic glands it may be of service, but it should not be given when suppuration is present. In countries where goitre is common it is said to act as an efficient remedy, given either by the mouth or by direct injection into the gland. It is useless in exophthalmic goitre and in cystic enlargement of the gland, but may prove of some value when simple overgrowth of the connective tissue of the gland is present. Small doses of the tincture, well diluted with water, frequently repeated, may check obstinate vomiting when other remedies fail.

### Iodides

1. *Potassii Iodidum.*—Prepared by dissolving iodine in liquor potassæ, evaporating, and heating the residue with charcoal, when by solution in boiling water, filtering, washing, and evaporation, large, slightly opaque, cubical crystals are obtained. It is soluble in less than its own weight of water, 1 in 12 of alcohol, and 1 in 3 of glycerin. *Dose*—5-20 grs. *Preparations*—(1) Unguentum Potassii Iodidi. (2) Linimentum Potassii Iodidi cum Sapone.

2. *Sodii Iodidum.*—Made in the same way as the potassium salt, a solution of soda being used in place of potash. It is a white, crystalline, deliquescent powder, freely soluble in water, glycerin, and alcohol. *Dose*—5-20 grs.

3. *Ammonii Iodidum*, a deliquescent powder consisting of minute crystalline cubes, is sometimes employed. It is non-official.

Administration of iodides is the recognised treatment in all cases of syphilis. In the primary stage mercury is preferred, but in the secondary and especially in the tertiary stages iodide is invaluable. Syphilitics usually, though not always, tolerate the drug well, and large doses must be given if the full benefit is to be obtained. The initial dose of 10 grains three times daily must be rapidly increased until symptoms of iodism occur, and then the amount should be diminished somewhat. Some patients will tolerate as much as three or four drachms daily. Nodules, gummata, etc. disappear rapidly under this treatment, but the parasyphilitic sclerotic conditions, particularly those of the nervous system, are little, if at all, influenced by it except in the very early stages. In tumours of doubtful nature the effect of large doses of iodide is a great aid in diagnosis. In chronic lead, zinc, arsenic, and mercurial poisoning, iodides form soluble salts with the metals and aid in their elimination from the tissues. In chronic articular rheumatism, in rheumatoid arthritis, in sciatica, in lumbago, and in some cases of neuralgia, iodides may prove of great service. In conditions of increased arterial tension associated with degenerative changes in the vessel walls, iodides, by lowering the blood pressure and increasing elimination of waste products, relieve symptoms and also retard the progress of the degenerative changes. It is to be noted in this connection that fairly large doses must be given, as small ones increase the blood pressure and thus aggravate the condition. In aortic aneurysm good results have been obtained from long-continued administration of large doses, but what the mode of action is in these cases has not yet been decided. In asthma, iodide may accomplish a complete cure when all other drugs have proved useless; and in bronchitis of the larger tubes and emphysema it is of great value. It has been recommended for the treatment of chronic Bright's disease; but it must be used cautiously, for if not properly eliminated by

the kidneys, it may produce untoward results. In the late stages of pneumonia where resolution is slow, iodide may promote absorption of exudates; and it has also been employed in chronic pleurisy and pericarditis. It should be given in the early stages of hepatic cirrhosis as it may possibly retard the overgrowth of connective tissue. Full doses are indicated in the treatment of actinomycosis. It is occasionally employed to arrest the secretion of milk. The depressant effect of potassium iodide may be counteracted by the addition of a little ammonium carbonate, or the sodium or ammonium salts may be used instead. Iodides are very disagreeable to taste and should be prescribed with sarsaparilla or liquorice, or should be taken in milk.

**Iodipin.**—A compound of iodine with oil of sesame, given, usually by subcutaneous injection, in cases of syphilis and in other morbid states in which the iodides are commonly administered (*e.g.* asthma, lead-colic, etc.); a 10 per cent. to a 25 per cent. solution is employed.

**Iodism.** *See* Drug Eruptions (*Iodine*); Nose, Acute Inflammation (*Acute Rhinitis, Etiology*); Pharmacology; Syphilis (*Treatment of Tertiary Syphilis*); Toxicology (*Non-metallic Elements, Iodine and Iodides*). — The prolonged use of iodine and the iodides is apt to be followed, especially in susceptible individuals, by coryza, watering of the eyes, headache, sore throat, and acne; these symptoms constitute iodism.

**Iodo-.**—In compound words *iodo-* usually signifies relating to iodine or an iodide, or combined with them, as *iodocaffein*, *iodocamphor*, *iodocodein*, etc.

**Iododerma.**—A skin eruption due to the use of iodine, especially *iododerma pustulotuberosum*, in which there is hyperkeratosis and parakeratosis. *See* Drug Eruptions (*Iodine*).

**Iodoform.** *See also* Ankle-Joint, Region of, Diseases (*Tuberculous Arthritis, Treatment*); Colour Vision (*Acquired Colour-Blindness, Causes*), Dressings (*Iodoform*); Drug Eruptions (*Erythematous, Papular*); Hip-Joint, Diseases (*Tuberculosis, Treatment, Iodoform Injections into Joint*); Joints, Diseases of (*Tuberculous, Treatment, Conservative*); Knee-Joint, Diseases of (*Tuberculous, Injection of Iodoform Glycerine*); Toxicology (*Non-metallic Elements*). —Iodoformum. Tri-iodomethane, $CHI_3$. Small yellow hexagonal crystals, with a characteristic disagreeable odour, are obtained by acting upon alcohol with iodine in the presence of caustic potash at a temperature of about 70° C. It is almost insoluble in water, soluble 1 in 95 of alcohol, and freely in fixed and volatile oils, ether, and chloroform. *Dose*—½-3 grs. *Preparations*—1. Suppositoria Iodoformi, 3 grs. in each. 2. Unguentum Iodoformi.

Iodoform is widely used in surgery for application to wounds, ulcers, sores, sinuses, etc. It is supposed to act as a mild disinfectant and antiseptic, and although the theory of its action by the liberation of iodine in the tissues has been greatly called in question, there is no doubt that clinically it is of the very greatest value. It is especially efficacious in the treatment of syphilitic and tuberculous ulcers, and it lessens pain and itching. It promotes the formation of plastic adhesions, and is therefore employed for this purpose in operative surgery. An emulsion of iodoform in glycerin (1 in 10) as an injection into tuberculous joints and abscess cavities has given very good results. For tuberculosis of the bladder, an emulsion of the same strength in mucilage of tragacanth and water is less irritating. An insufflation of iodoform with equal parts of starch or boric acid is of great value in tuberculous ulceration of the larynx; ⅙ gr. acetate of morphia may be added if there is great pain. The same preparation is useful in ulcerative conditions of the mouth and throat and in ozæna. Painful defæcation from fissure of the anus or irritated hæmorrhoids may be prevented by the introduction of a suppository shortly before going to stool.

Bougies of iodoform for the treatment of gonorrhœa and pencils for introduction into the uterus have been prepared. Recently the intravenous injection of a solution of iodoform in ether has been introduced by Dewar for the treatment of phthisis. When the solution reaches the lungs the ether evaporates and the iodoform is deposited. Iodoform is of little value in internal medication although it has been tried in a large number of diseases, including tertiary syphilis, phthisis, catarrhal jaundice, and hepatic cirrhosis. Iodoform in capsules made of glutoid, which is dissolved by the pancreatic secretion but not by the gastric and intestinal, has been made use of to estimate the time of the passage of food from the stomach to the intestine, and to demonstrate the activity or otherwise of the pancreatic secretion. When the capsule has been dissolved and the iodoform absorbed, iodates and iodides can be detected in the saliva by testing with chloroform and dilute nitric acid (a rose-red or violet coloration of the chloroform is obtained). If no iodoform is absorbed the pancreatic secretion is absent or inactive — as is found in some cases of infantilism.

A great many substitutes for iodoform have been placed on the market within recent years. *Aristol*, di-thymol iodide, is a useful dusting powder, and has also been recommended for psoriasis, lupus, and eczema. *Iodol*, tetra-iodopyrrol, an almost odourless, crystalline, brownish powder, has been used for much the same purposes as iodoform. *Iodoformal*, *Europhen*,

*Nosophen*, *Isoform*, and *Traumatol* also come under this group, but it cannot be said that any particular advantage attaches to any of them.

**Iodoformin.**—A substitute for iodoform, having the formula $C_2H_6N_2I_2$; when acted upon by ethyl iodide *iodoformal* is produced.

**Iodoformism.** — Poisoning with iodoform. *See* IODOFORM; TOXICOLOGY (*Non-metallic Elements*).

**Iodol.**—Iodol or iodopyrrol ($C_4I_4HN$) is a non-official iodoform-like substance (containing 90 per cent. of iodine), often used in place of iodoform; *pyroll* ($C_4H_5N$) is got from the distillation of bones, and iodol is obtained from it.

**Iodophilia.**—The iodine or iodophilous reaction is a peculiar susceptibility of the protoplasm of leucocytes (especially the polymorphonuclear) to the action of iodine applied to the dried film, which stains it a variable shade of brown instead of a pale lemon-yellow tint; it is met with in toxæmias, both bacterial (*e.g.* sepsis) and non-bacterial (*e.g.* diabetes mellitus) in character. *See* GLYCOGEN REACTION.

**Iodothyrin.**—A substance with a marked power of combining with iodine, found in the thyroid gland and probably forming the active constituent of its internal secretion. *See* PHYSIOLOGY, INTERNAL SECRETIONS (*Thyroid Gland*).

**Ionic Action.**—When electrolytes (salts, bases, and acids) are brought into solution it is believed that their molecules are broken up into two parts (*ions*); these ions are regarded as intermediate bodies between atoms and molecules, and are charged with negative electricity (*kations*) or with positive (*anions*); they are supposed to have a powerful influence on certain physiological and pathological phenomena (*e.g.* osmosis, coagulation of the blood, agglutination of bacteria, etc.), and on the action of various drugs.

**Iothion (or Jothion).**—Iothion or di-iodo-hydroxypropane ($C_3H_5I_2OH_2$) is a preparation which can be used externally in place of the tincture and the ointment of iodine.

**Ipecacuanha.** — Ipecacuanhæ Radix, the dried root of *Psychotria ipecacuanha*, imported from Brazil. It contains an uncrystallisable alkaloid *Emetine*, and *Ipecacuanhic acid*. *Dose*—¼-2 grs. (expectorant); 15-30 grs. (emetic). *Preparations*—1. Acetum Ipecacuanhæ. Strength, ·1 per cent of alkaloids. *Dose*—10-30 m. 2. Extractum Ipecacuanhæ Liquidum. Strength, 2-2·25 per cent of alkaloids. *Dose*—½-2 m. (expectorant); 15-20 m. (emetic). 3. Pilula Ipecacuanhæ cum Scilla. *Dose*—4-8 grs. 4. Pulvis Ipecacuanhæ Compositus. Dover's powder. *Dose*—5-15 grs. 5. Trochiscus Ipecacuanhæ. ¼ gr. in each. 6. Trochiscus Morphinæ et Ipecacuanhæ. $\frac{1}{12}$ gr. ipecacuanha and $\frac{1}{36}$ gr. morphine hydrochloride in each. 7. Vinum Ipecacuanhæ. Strength, ·1 per cent of alkaloids. *Dose*—10-30 m. (expectorant); 4-6 ʒ (emetic).

Externally, ipecacuanha has been recommended as a dressing for anthrax, the drug being taken by the mouth at the same time. A paste made from the powdered root is said to relieve the pain caused by bee-stings. Internally, it is a valuable emetic, especially in children when the stomach is overloaded with food, or where the expectoration from bronchitis has been swallowed in large quantities. In poisoning it is hardly rapid enough in action; it acts on the medulla after absorption, and sulphate of zinc or mustard, which stimulate the stomach directly, are to be preferred. In small doses it is employed as a stomachic to check obstinate vomiting, especially the vomiting of pregnancy. It is widely used as a depressing expectorant, particularly in the early stages of an attack of bronchitis. It is a frequent ingredient of cough mixtures and is indicated where the secretion is dry and scanty. In robust children suffering from laryngitis or bronchitis full emetic doses will often produce marvellous results, the act of vomiting clearing out the obstructed air-passages. Dover's powder is a very efficient remedy in the early stages of a feverish cold. Ipecacuanha is a useful addition to cholagogue pills for the treatment of bilious dyspepsia: it stimulates the secretion of bile. It is regarded as a specific for dysentery of the true acute type. A large dose (30-60 grs.) of the powdered root is to be given on an empty stomach, and should be preceded by a dose of laudanum so as to prevent vomiting. Deemetised ipecacuanha has also been employed for this purpose, but its value is questionable. The inhalation of a spray of ipecacuanha wine, diluted with equal parts of water and alcohol, has been recommended for asthma and chronic bronchitis.

**Ipomœa.** *See* JALAP.

**Iridalgia.**—Pain in the iris (due to synechiæ).

**Iridectomy.**—Excision of a portion of the iris in order to form an artificial pupil. *See* CATARACT (*Treatment, Iridectomy*); GLAUCOMA (*Operative Treatment, Iridectomy*); IRIS AND CILIARY BODY (*Operations on Iris, Iridectomy*); OCULAR MUSCLES, AFFECTIONS OF (*Nystagmus*); SCLEROTIC, DISEASES OF (*Sclerotitis, Treatment*).

**Irideremia.** — Absence of the iris, antenatal, acquired, or the result of operation; aniridia. *See* IRIS AND CILIARY BODY (*Congenital Abnormalities*).

**Iridin.**—A dark brown powder obtained from the root of the blue flag, *Iris versicolor*.

It has a bitter acrid taste, and possesses cathartic and diuretic properties. *Dose*—1-5 grs. in pill. It is a cholagogue, and has the advantage that it rarely gripes. It may be given in combination with other purgatives.

**Irido-Choroiditis.** — Inflammation of the iris and choroid; uveitis. *See* CHOROID, DISEASES OF (*Irido-choroiditis or Uveitis*).

**Iridocinesis.** — The movement of contraction or expansion of the iris. *See* PHYSIOLOGY, SENSES (*Vision, Mechanism, Iris*).

**Irido-Cyclitis.** — Inflammation of the iris and ciliary body. *See* CATARACT (*Operation, After-Results*); GOUT (*Irregular, Eye Symptoms*).

**Iridodesis.**—An operation for lengthening the pupil or for making an artificial pupil (for optical purposes).

**Iridodialysis.** — Separation of the iris from adhesions or from its attachments. *See* EYEBALL, INJURIES OF (*Detachment*); IRIS AND CILIARY BODY (*Injuries*).

**Iridodonesis.**—Tremulousness or quivering of the iris, due to partial dislocation of the crystalline lens. *See* EYEBALL, INJURIES OF (*Dislocation of the Lens*); IRIS AND CILIARY BODY (*Anatomy, Iridodonesis*); LENS, CRYSTALLINE (*Displacements*).

**Iridoparesis.**—Partial paralysis of the iris.

**Iridoplegia.** — Paralysis of the iris; failure of the iris to respond to the action of light or to the effort of accommodation. *See* IRIS AND CILIARY BODY (*Injuries*).

**Iridoptosis.** — Prolapse of the iris (*e.g.* through a wound of the cornea).

**Iridotomy.**—Incision into the iris (*e.g.* for the making of an artificial pupil). *See* IRIS AND CILIARY BODY (*Operations*).

## Iris and Ciliary Body.


ANATOMY . . . . . 36
PHYSIOLOGY . . . . . 38
CONGENITAL ABNORMALITIES . . . 39
INFLAMMATORY AFFECTIONS . . . 40
*Iritis* . . . . . 40
*Cyclitis* . . . . . 40
*Treatment* . . . . . 42
OPERATIONS ON THE IRIS . . . . 44
*Iridectomy* . . . . . 44
*Iridotomy* . . . . . 46
TUMOURS OF IRIS AND CILIARY BODY . 46
INJURIES TO THE IRIS AND CILIARY BODY . 46


*See also* ACCOMMODATION; CHEEK, FISSURE OF; EYEBALL, INJURIES OF (*Anteversion and Retroflexion of the Iris*); PHYSIOLOGY, SENSES (*Vision*); PUPIL.

ANATOMY.—The iris is the anterior part of the tunica vasculosa or uveal tract. It is the beautifully coloured and contractile membrane seen through the transparent cornea. By its circumference it is attached to the ligamentum pectinatum and ciliary body. Its anterior surface is free, whilst the posterior surface rests by its pupillary margin against the capsule of the lens; an absence of this support causes the iris to be tremulous—*iridodonesis* It separates the anterior chamber, between it and the cornea, from the posterior chamber, between it and the lens. Slightly to the nasal side of its centre is the aperture of the pupil, whose diameter, when the iris is resting, varies from three to six millimetres. In thickness the iris is about 0·4 millimetre.

Developmentally, the iris consists of two parts, the anterior portion being mesodermic, the posterior portion ectodermic, in origin. The mesodermic iris is composed of the following layers:—

1. *The anterior endothelium*, continuous with, and similar to, the lining cells of Descemet's membrane and the ligamentum pectinatum. It is broken here and there, where crypts in the succeeding layer exist.

2. *The substantia propria*, consisting of a stroma of connective tissue, containing branching connective-tissue cells, many of which in dark eyes are pigmented. The anterior part of this layer is somewhat condensed and free from blood-vessels; this has been termed the *anterior boundary layer*. Within the stroma are found muscular fibres, blood-vessels, lymphatic tissue, and nerves. The muscular fibres are unstriped, and are arranged to form two definite tracts. One of these, the *sphincter* or *constrictor pupillæ*, consists of a flattened ring around the edge of the pupil, nearer to the posterior than to the anterior surface of the iris. The second muscular tract, the *dilator pupillæ*, is radially placed in the posterior part of the stroma, immediately anterior to the membrane of Bruch. It is composed of delicate spindle cells, which cannot be seen unless the iris is first bleached.

3. *The posterior limiting membrane*, or *membrane of Bruch*, a thin hyaline membrane, continuous with the lamina vitrea of the choroid and ciliary body.

The ectodermic iris is composed of two layers of pigmented cells: (*a*) an anterior layer of spindle cells, the continuation of the pigmented layer of the retina; and (*b*) a posterior layer of polygonal cells, the representative of the remainder of the retina. These layers can only be seen in albino irides, or where the iris has been bleached. Together they form *the uvea*.

The colour of the iris in dark eyes is due to pigment granules in the connective-tissue corpuscles of the substantia propria, especially those in the anterior portion. If pigment is absent from the stroma, and only present in the

uvea, the iris appears blue, if comparatively thin, as is the case in children and young adults; where the iris becomes thicker the colour changes to gray. The eyes of newly-born children, even among the dark races, are always blue, since at this age the pigmentation is confined to the uvea. If pigment is absent from both stroma and uvea, the eye appears pink, owing to the choroidal reflex shining through the iris; this is seen in albinos.

The ciliary body is that part of the tunica vasculosa which extends backwards from the base of the iris to the ora serrata. Anatomically, it consists of three parts: (1) an anterior thick portion, which supports the ciliary processes, the *pars plicata* or *corona ciliaris*; (2) a posterior portion, devoid of processes, which gradually thins off into the choroid, the *pars non-plicata* or *orbicularis ciliaris*; and (3) the *ciliary muscle*.

The *ciliary processes* are very vascular folds of a connective-tissue stroma similar to and continuous with that of the iris, and limited externally by the ciliary muscle. They are about seventy in number, and between two and three millimetres long. Lining them, as well as the pars non-plicata, are two layers of cells separated from the underlying connective tissue by the *lamina vitrea*. The layer next this lamina is pigmented, and is continuous with the pigmentary epithelium of the retina behind and the corresponding layer of the iris in front. The inner layer of cells, devoid of pigment, is the sole representative of the remaining layers of the retina, and is called the *pars ciliaris retinæ*; in the iris it is continued as a pigmented layer. It consists of two kinds of cells, one set being cuboidal, granular, and with prominent nuclei; the other set being extremely fine and elongated, so as to form slender fibrils, which unite together and aid in the formation of the suspensory ligament. Secretory tubular glands have been described by Treacher Collins. They exist, for the most part, at the junction of the plicated and non-plicated portions of the ciliary body.

The *ciliary muscle* arises from the fibres of the ligamentum pectinatum opposite the sclero-corneal junction; the greater part of its fibres (meridional) pass directly backwards to be inserted into the external layers of the choroid. The different muscular bundles are separated by small tracts of connective tissue. Other fasciculi (oblique), more loosely arranged, pass inwards to the ciliary processes; these run divergingly, and frequently anastomose with each other. Others, the most internal, on reaching the base of the iris, pass into a direction almost circular, forming the *circular muscle of Müller*. This annular muscle is better developed in hypermetropes than in emmetropes. In myopes, on the contrary, the circular fasciculi are small, the meridional fibres constituting nearly all the muscle.

The fibres are of the unstriped variety; the muscle possesses a network of capillaries and a plexus of non-medullated nerve-fibres, with numerous ganglion cells.

The *blood-supply* of the iris and ciliary body comes from the long posterior and the anterior ciliary arteries.

The two *long posterior ciliary arteries*, arising from the ophthalmic artery, pierce the sclerotic close to the optic nerve, one on either side, and pass forwards between the choroid and sclerotic, supplying neither, till they reach the ciliary region. Close to the posterior border of the ciliary muscle each divides into two branches, which take a circular direction parallel to the equator of the eyeball, and unite with branches from the anterior ciliaries to form the circulus arteriosus iridis major, which lies between the meridional and circular portions of the ciliary muscle.

The *anterior ciliary arteries*, about five in number, come from the muscular and lachrymal branches of the ophthalmic artery; they pierce the sclerotic near the margin of the cornea, and then divide into branches to the ciliary muscle and to the circulus arteriosus iridis major.

The *circulus arteriosus iridis major* gives off branches to the ciliary processes. It also sends branches to the iris, which pass radially towards the pupillary margin. Close to the latter they form an anastomotic ring, the *circulus arteriosus iridis minor*, from which, in the fœtal eye, fine branches pass to the pupillary membrane.

The iridic arteries have thick middle and outer coats, and but little muscular tissue.

The veins of the iris and ciliary body do not exactly correspond to the arteries. Thus the anterior ciliary veins are quite small, and only receive blood from part of the ciliary muscle. They anastomose with the conjunctival veins and with Schlemm's canal. The blood from the veins of the iris and the rest of the ciliary body is returned to the choroidal veins, and so to the venæ vorticosæ.

The *nerves* of the iris and ciliary body are derived from two sources. The *long ciliary nerves* are branches of the nasal branch of the ophthalmic division of the fifth nerve, and are therefore sensory. They supply the ciliary muscle. The *short ciliary nerves*, about fifteen in number, are branches of ciliary ganglion, being derived from the oculo-motor nerve, and pierce the sclerotic near the optic nerve entrance, passing forwards between this tunic and the choroid. On reaching the ciliary body they form the ciliary plexus, from which fibres pass to the ciliary muscle. Passing on, a second plexus is formed in the iris, from which vaso-motorial branches supply the vessels; muscular branches from the third nerve supply the constrictor pupillæ; sympathetic fibres pass to the dilator pupillæ; while sensory fibres supply the anterior surface of the iris. Ganglion cells are found in

the ciliary body, but in the iris their presence is doubtful.

The *lymphatics* of the Iris and Ciliary Body.—There are no distinct lymphatic vessels in the iris, but the sheaths of the blood-vessels contain lymphatic sinuses, and the whole stroma is riddled with lymph spaces. Whether these spaces are in direct communication with the anterior chamber through the crypts described above is not definitely known; they, however, open into the spaces between the fibres of the ligamentum pectinatum, and so reach the canal of Schlemm.

Between the ciliary muscle and the sclerotic coat is a lymph space continuous with that between the lamina suprachoroidea and the lamina fusca, and limited anteriorly by the attachment of the ciliary muscle. It is a common seat of inflammatory exudation or hæmorrhage in cyclitic trouble.

Physiology of the Iris and Ciliary Body.—Under this heading the following important subjects are included—the action of light and certain poisons on the pupil; associated movements; the mechanism of accommodation; and the function of the ciliary processes.

Movements of the Pupil.—The iris, with its central aperture, the pupil, serves as a diaphragm to shut off marginal rays; spherical aberration is thus prevented. It also regulates the amount of light entering the eye.

*Contraction of the pupil* to light is brought about by the action of the sphincter pupillæ, which is governed by a reflex mechanism, of which the optic nerve is the afferent path, and the oculo-motor nerve the efferent path, the centre being situated in the floor of the aqueduct of Sylvius. The method of examining the pupillary light reflexes is as follows:—Let the patient be placed in a good bright light, daylight by preference, then cover both eyes with the hand; upon exposing one eye—for example, the right—to the light, its pupil contracts (*direct light reflex*), and the left pupil also contracts (*indirect* or *consensual light reflex*), though to a less extent, being indirectly stimulated through the light entering the right eye; upon exposing the left eye, a further slight contraction occurs in the right eye (consensual reflex) in conjunction with the direct reflex in the left. Consensual light reflex cannot be obtained in those animals whose optic nerves decussate completely. If, after shading the eyes for a few minutes, they are suddenly exposed to a bright light, a slight and temporary rhythmical contraction and dilatation of the pupils ensue —*hippus*; this is very evident in neurotic persons.

Other causes of contraction of the pupil are: (1) stimulation of the optic nerve by agents other than light, as electricity; (2) accommodation of the eye for near objects; (3) the early stages of poisoning by chloroform, alcohol, etc., and nearly all stages of poisoning by morphia, eserine, and some other drugs; (4) deep sleep; and (5) the local application of eserine and other myotics.

*Dilatation of the pupil* is brought about by the dilator pupillæ, which is governed by a reflex mechanism, just as is the constrictor pupillæ. The dilator pupillæ, as to the existence of which there has been much controversy, has been demonstrated both physiologically and anatomically. It is supplied by sympathetic fibres arising from the cilio-spinal centre in the upper dorsal region of the cord. This centre is subordinate to a higher reflex nerve-centre in the medulla. The fibres leave the cord by the lower cervical and upper dorsal anterior nerve-roots, and, passing into the last cervical and first thoracic ganglia, enter the sympathetic cord, pass upwards to the Gasserian ganglion, and through the nasal nerve enter by the long root the lenticular ganglion, from which they proceed by the ciliary nerves to the iris.

Dilatation of the pupil occurs: (1) when the stimulus of light is withdrawn from the retina; (2) when the eye is adjusted for distant vision; (3) during dyspnœa, powerful irritation of the sensory nerves, violent muscular efforts, in the later stages of chloroform poisoning, and in all stages of poisoning by atropine and certain other drugs; and (4) after the local action of atropine and other mydriatics.

Besides the reflex mechanisms regulating the size of the pupil, a local mechanism is supposed to exist either in the iris itself or in the choroid, where ganglion cells are present. When eserine is applied to the eye, contraction of the pupil is caused whether the third nerve has been divided or not, and with a strong dose it cannot be overcome by stimulation of the sympathetic.

Associated Movements.—Two movements are said to be associated when the special central nervous mechanism used in carrying out the one act is so connected with that employed in carrying out the other that when we set the one mechanism in action we unintentionally set the other in action also. Thus the movements of convergence, accommodation, and contraction of the pupil are produced together during near vision by simultaneous stimulation of all the fibres of the third nerve, and if one of these is in excess, as in convergent concomitant strabismus with hypermetropia, it may be merely necessary to correct the excessive accommodation in order to cure the squint. The nucleus of the third nerve consists of definite component parts; the most anterior portion presides over accommodation; below this is the centre for the action of the pupil; farther down come the cells governing the levator palpebræ and the internal rectus; while most posteriorly the nucleus supplies fibres to the rectus superior, obliquus inferior, and rectus inferior.

The Mechanism for Accommodation.—All are agreed that during accommodation the anterior surface of the lens becomes more convex, but how exactly this is brought about has not been definitely proved. The capsule of the lens is attached by the suspensory ligament to the ciliary processes, and any alteration in its tautness will produce an alteration in the form of the elastic lens. Two chief theories have been brought forward. (1) According to the *theory of Helmholtz*, during the act of accommodation for near objects the suspensory ligament is relaxed. This is chiefly brought about by the annular part of the ciliary muscle, which on contracting causes the ciliary processes to approximate to the axis of the eyeball. Help is also given by the meridional fibres, which, acting on the choroid, draw it and the ciliary body forwards. As a result, the ligament is relaxed, and the elasticity of the lens causes the whole of its anterior surface to become more spherical.

(2) According to the more recent *theory of Tscherning*, the suspensory ligament is tightened during accommodation for near objects. The annular part of the ciliary muscle uses the choroid, to which it is attached, as its fixed point, and draws the ciliary processes towards this, thus tending to cause a posterior displacement of the lens. This is prevented by the rest of the ciliary muscle, which, with the sclero-corneal junction as its origin, pulls on the choroid, causing a forward movement of the vitreous. As a result of these actions, the anterior surface of the lens becomes hyperboloid in form; in the neighbourhood of the anterior pole it becomes more convex, with flattening of the more peripheral zones.

The evidence in support of these two views is considered in the article on "Accommodation" in vol. i.

The Function of the Ciliary Processes.—The ciliary processes have a double function; they support the lens by means of the suspensory ligament, which is partly attached to them, and they take part in the production of the aqueous humour. Exactly what portion of their structure is engaged in this secretion is still a matter of some doubt. The ciliary glands discovered by Collins are considered by him to be the source of the fluid of the eye, but it is doubtful whether they are sufficiently numerous, and it seems better to consider that the processes as a whole are the structures concerned. It has been suggested that the ciliary glands, of whose existence there seems to be no doubt, secrete pigment. They are found in greater number in dark eyes than in blue eyes, and appear to be absent in albinos.

Congenital Abnormalities of the Iris.—Abnormalities in Pigmentation.—Pigment may be completely or almost completely absent from the iris in the condition known as *albinism*. The eyes look pink, on account of the choroidal reflex shining through the non-pigmented iris. For further description of albinism see "Skin, Pigmentary Affections of."

Pigment may be arranged in the iris not only diffusely, but in small aggregations, giving rise to a mottled appearance.

The iris of one eye may remain blue while that of the other eye is pigmented, becoming brown; a sector of one iris may, again, be of a different colour from that of the rest. These conditions are included under the term *heterochromia*. When the heterochromia is complete, the colour of one may correspond to the colour of the father's irides, and that of the other to the colour of the mother's irides.

Occasionally small masses of pigment are seen at the pupillary margin; these are extensions of the uveal pigment of the iris, and are found congenitally as well as in chronic glaucoma. It is normally found in the horse, and is termed *ectropion of the uveal pigment*.

Abnormalities in Form. — *Irideremia* or *Aniridia* is a more or less complete absence of the iris, a narrow rim alone remaining. It is frequently found in members of the same family and is often accompanied by choroidal coloboma and lenticular opacities.

*Coloboma iridis* consists in a congenital cleft in the iris. It is usually directed downwards, or downwards and slightly inwards, and resembles the gap made by iridectomy, except that the constrictor muscle can be seen to line its walls. It varies in extent and frequently occurs in both eyes. It is often accompanied by coloboma of the choroid. It is frequently hereditary, and has been traced through four generations. It has been produced artificially in the offspring of animals, a small sector of whose irides has been caused to become atrophic. Most cases of downward coloboma are probably due to a late closure of the choroidal fissure. On account of this, there is a cleft in the ciliary body, and consequently here either no iris is developed or it is rudimentary. This, however, does not explain the condition of upward coloboma, nor of a double coloboma in the same eye, an abnormality which has been described. These cases are probably due to some irregularity in the production of the secondary optic vesicle, hindering the development of a sector of the iris.

*Polycoria* is an exceedingly rare condition, in which the natural pupil is divided into two or more apertures by bands of iridic tissue. As many as eight extra pupils have been described, each surrounded by muscular tissue, as shown by the behaviour to atropine.

*Corectopia* is a congenital misplacement of the pupil. It may be situated quite at the periphery, close to the sclero-corneal junction. It is a rare deformity, and is frequently accompanied by dislocation of the lens.

*Persistent remains of the pupillary membrane* are sometimes found. The membrane is usually represented by one or more fine filaments which arise from the circulus arteriosus iridis minor (thus being distinguished from posterior synechiæ), and are attached either to the iris opposite or to the anterior capsule of the lens. Occasionally a definite membrane is seen in front of the lens.

*Iridodonesis*, or trembling iris, is found in cases of congenitally dislocated lens.

Inflammatory Conditions of the Iris and Ciliary Body.—Hyperæmia of the iris attends many local inflammatory changes such as acute conjunctivitis; keratitis, especially if due to a foreign body deeply imbedded in the cornea; other forms of injury; and after intraocular operations, as iridectomy, cataract extraction, etc. Being the first stage of inflammation, its early recognition, combined with prompt treatment, will often cut short an impending attack of iritis. It is characterised by contraction and sluggish action of the pupil; slight discoloration of the iris with indistinctness of its reticulum; engorgement of its vessels, which can often be seen with a strong magnifying lens; media which are not hazy; and a pupil whose outline is clear and which dilates fully and regularly under a mydriatic. The circumcorneal zone is injected, and lachrymation with photophobia is usually present. The treatment consists mainly in local depletion by leeches and the use of atropine.

Inflammatory conditions of the iris and ciliary body are frequently found together; it will be well to consider the symptoms of each separately before seeing what their etiological factors are.

Symptoms of Iritis.—The earliest symptoms are those of hyperæmia of the iris:—1. *The mobility of the iris is diminished*; its movements are either sluggish or lost, on account of spasmodic action of the sphincter pupillæ.

2. *The colour of the iris is altered.* This change is sometimes very slight, but by careful examination with oblique focal illumination there will nearly always be found an alteration in the colour of the tissues surrounding the edge of the pupil. The two irides should be very carefully compared. In many cases the change in colour is very marked, the blue or gray iris becoming yellowish green, whilst the dark brown iris assumes a brownish red or rust colour.

3. *The blood-vessels immediately surrounding the cornea are injected*, forming a pink or deep red ring; there is conjunctival injection, with photophobia and lachrymation.

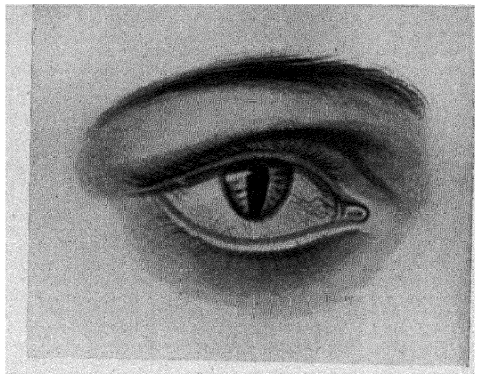
In addition to these, we have symptoms resulting from inflammatory exudation. *Turbidity* of the iris, due to exudation into its substance, and of the aqueous fluid, ensues. This produces a muddy appearance of the pupil. Pus may collect in the anterior chamber (*hypopyon*), or an iridic vessel may rupture, causing hæmorrhage (*hyphæma*). If the lymph which occupies the pupil becomes organised, we get an opaque persistent membrane, which may be perforated by one or more small apertures. This condition is termed *occlusion of the pupil.* Again, lymph in the posterior chamber may become organised, forming adhesions between the iris and the capsule of the lens (*posterior synechiæ*). If the whole of the pupillary margin is adherent to the lens capsule, the condition is termed *annular posterior synechia*; the anterior chamber is cut off from the posterior chamber (*seclusion*, or *exclusion*, of the pupil). Lastly, the whole of the posterior surface of the iris may be adherent to the capsule of the lens, a condition known as *total posterior synechia.* This, however, is rarely found in absence of cyclitis.

Other symptoms of iritis are *impaired vision* and *pain.* The amount of diminution of vision varies very considerably. With occlusion of the pupil there is very much less sight than with exclusion. Pain may be entirely absent, or may exist in various degrees of severity within the eye and in the surrounding parts. The amount depends largely on the etiological factor.

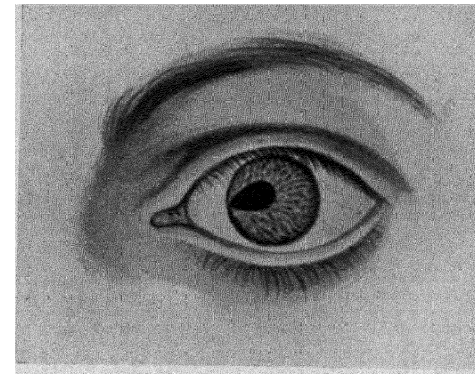
Symptoms of Cyclitis.—This affection, which, when it occurs without iritis, is usually chronic, with few inflammatory symptoms, is recognised by a somewhat dilated sluggish pupil, a normal-looking iris, slightly raised tension, and by the presence of so-called *keratitis punctata* (or *precipitates*), being a dotted appearance on the lower part of the posterior surface of the cornea. Its pathology will be considered later. Cyclitis, when it accompanies iritis, is much more acute, and is recognised by the following additional symptoms:—(1) tenderness over the ciliary region; (2) increased intraocular tension; (3) vitreous opacities; (4) total posterior synechia; and (5) cellulitis of the eyelids and cheek. It is rare for cyclitis to occur without iritis, the irido-cyclitis usually found being that disease formerly named serous iritis, in contradistinction to plastic iritis with its marked exudation, as described above.

In considering the etiology, pathology, diagnosis, prognosis, and treatment, iritis and cyclitis will be taken together.

Etiology of Iritis and Cyclitis.—In the large majority of cases of iritis and cyclitis the cause is some constitutional taint. It is probably only in the case of a perforating injury that iritis is a purely local affection. Cases of iritis occur, the cause of which cannot be discovered; these, till they are better understood, are termed idiopathic. Hence we have the following etiological classification of iritis and cyclitis:—(1) Symptomatic; (2) traumatic; (3) idiopathic. In addition to these we have (4) secondary iritis, *i.e.* iritis following inflammation of neighbouring structures.



Coloboma.



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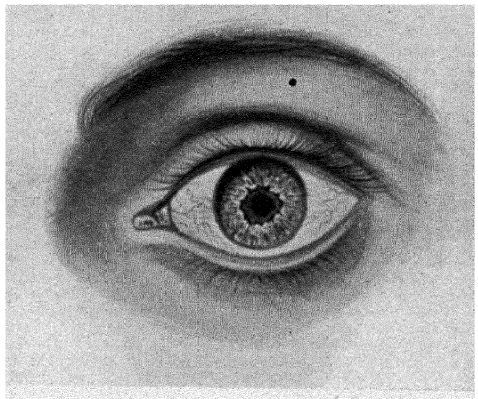
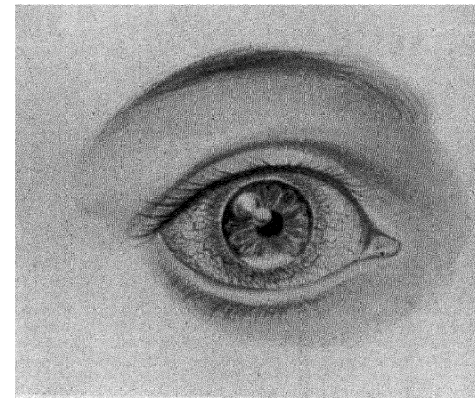
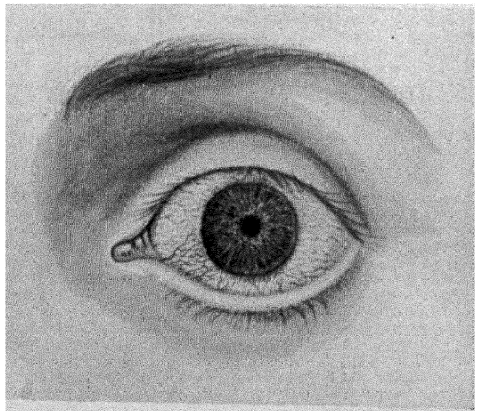


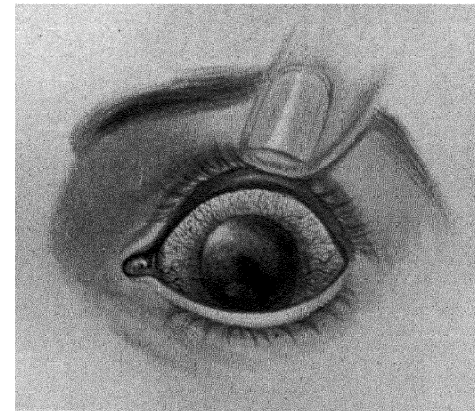
Iritis (with adhesions).



Syphilitic Iritis.



Iritis.



Iritis.

(1) *Symptomatic iritis* occurs as a result of syphilis, rheumatism, gonorrhœa, tuberculosis, gout, and diabetes; it may follow one of the exanthemata.

(*a*) *Syphilitic iritis* is much the most common form of iritis. It appears as a rule within the first nine months of contagion, usually in the fourth or fifth month. It occurs in about 4 per cent. of all cases of syphilis. There are four chief forms—plastic iritis, iritis gummosa or papulosa, gumma of the iris and ciliary body, and irido-cyclitis.

The *plastic* form differs from the other forms in the large amount of exudation and in the rapidity with which this becomes organised, in showing no tendency to recur when once cured, and in the absence of very severe pain.

*Iritis gummosa* or *papulosa* appears as a secondary manifestation, and must not be confounded with the next form. It occurs in 18 per cent. of all cases of plastic iritis. Its characteristic feature is the formation of multiple orange or rust-coloured nodules of lymph, either at the ciliary or the pupillary margin of the iris. The ordinary signs of iritis are present. As a result of this form, a broad posterior synechia is very common.

True *gummata* of the iris and ciliary body may occur as a tertiary symptom of syphilis, but are very rare. They usually start at the iridic angle, and cause marked staphyloma Vision is usually greatly affected, though a case has been recorded (*Ophth. Review*, vol. xvii. p. 238) in which, after correction of the resulting astigmatism, useful vision was restored.

*Irido-cyclitis*, the fourth variety of syphilitic iritis, is a late secondary or tertiary manifestation, since it may occur several years after the primary infection. It is frequently accompanied by choroiditis.

Hereditary syphilis is occasionally characterised by *congenital syphilitic iritis*, occurring at, or a few hours after, birth. A little later, usually between the ages of six weeks and sixteen months, iritis may develop—*infantile iritis*. Still later, interstitial keratitis may be complicated by iritis, extensive adhesions sometimes resulting. Occasionally cyclitis, with no keratitis, results from hereditary syphilis.

(*b*) *Rheumatic iritis* may follow acute rheumatism, but is more common in the chronic form. It shows a great tendency to recur, the relapses often alternating with the attacks of rheumatism, though in some cases the two are synchronous. It is attended with greater pain and conjunctival injection than the syphilitic form. The plastic exudation, however, is less; there is consequently less change in the colour of the iris, and the adhesions are less extensive and form less rapidly. Sight may be little affected, even after repeated attacks. Both the iris and ciliary body usually are implicated.

(*c*) *Gonorrhœal iritis* resembles rheumatic iritis very closely. It is found especially with gonorrhœal rheumatism, although it may occur in cases where the joints are not affected. Like rheumatic iritis, this form shows a special tendency to relapses, especially after a renewal of the urethral discharge or a return of articular swelling.

(*d*) *Tuberculous irido-cyclitis* is a form of iritis occasionally seen, though, from the rarity of its occurrence, its earlier stages are very liable to be overlooked. It is essentially chronic, and differs chiefly in this respect from all other varieties. This disease is probably always secondary to a primary tuberculous focus elsewhere in the body. In the disseminated variety the pupil is usually contracted and immobile. At first nodulated on its surface, the iris eventually fills up the anterior chamber; its colour is a dull brick-red mottled with gray, and it appears to be very vascular. The pain and circumcorneal injection are not marked at first, though in the later stages, owing to the supervention of secondary glaucoma, the pain becomes intense and persistent in spite of all palliative measures; chemosis and intense circumcorneal injection accompany this increase of tension. It usually occurs in young subjects. The diagnosis is determined by the chronicity of the affection, by the characteristic appearance of the iris, and by the resistance to all ordinary treatment for iritis. Upon making an antero-posterior section of the excised eye, the tubercular appearance of the iris is most striking. It fills the whole anterior chamber and moulds itself to the shape of the cavity, becoming adherent to the cornea and lens. It presents to the naked eye the appearance of small white nodules massed together and interrupted here and there by the disturbed uveal pigment, giving the growth the resemblance of marble. Microscopically, these nodules present the characteristic features of miliary tubercle, central giant-cells surrounded by epithelioid cells and small round cells. They are non-vascular, though the vessels between them are enlarged and numerous. The ciliary body is usually involved, and the uveal pigment is so interrupted that it would be difficult to believe from the appearance that it was ever a continuous tract. The tubercle bacillus has in many instances been found. Recovery has occasionally taken place with gradual absorption of the growth, but as a rule the prognosis is bad, the eye being lost, and the patient usually dying from general tuberculosis or meningitis. The solitary variety is very similar in its symptoms, pathology, and prognosis, but starts singly.

(2) *Traumatic iritis* results from a perforating injury, mechanical violence, or chemical irritation. Of these the first is much the most common. The iritis may go on to a suppurative

form, with the formation of hypopyon. Suppurative iritis is seldom confined to the tissues of the iris, but is usually either derived from, or extends to, the surrounding tissues.

Under this heading may be included sympathetic iritis; this will be described under sympathetic ophthalmitis.

The treatment of suppurative traumatic iritis is similar to that of suppurative choroiditis.

(3) *Idiopathic iritis.*—Many cases of iritis seem to have no definite cause, either from injury or from constitutional taint. They apparently come on spontaneously, and resemble in every respect an acute plastic iritis or a more chronic irido-cyclitis.

(4) *Secondary iritis* is uncommon. The primary cause is usually a suppurative keratitis; more rarely it follows choroiditis.

Pathology of Iritis and Cyclitis.—In plastic iritis there is hyperæmia of the vessels with small-celled infiltration of the iridic stroma. Exudation follows, consisting of fibrin, with a varying number of leucocytes. This exudation may become completely absorbed, or it may be organised into connective tissue, synechiæ being the result. A similar set of changes takes place in cyclitis, but added to them we have *keratitis punctata*. This condition is found microscopically to be due to small aggregations of round cells cemented together with fibrin, containing a small amount of pigment. They are not formed by any inflammatory condition of the cornea, but result from an inflamed ciliary body, being carried by the lymph stream through the pupil to the back of the lower part of the cornea, where they are usually arranged in the form of a triangle, with the apex at the centre of the pupil.

The complications and sequelæ of iritis are few, though important. *Synechiæ*, anterior and posterior, may occur. The former are almost invariably the result of a perforating wound or of an ulcer of the cornea; rarely they are due to tuberculous or gummatous inflammation of the anterior surface of the iris. The latter may arise from not dilating the pupil early in the attack; exclusion and occlusion are the more serious forms. As a result of annular posterior synechia, atrophy of the iris occurs, with a bulging forwards of the ciliary portion, a condition known as *iris bombé*. This closes the iridic angle, and secondary glaucoma may be set up. Another sequela is opacity of the lens, resulting from deficient nutrition; this may occur with or without *phthisis bulbi*, which is found as a sequela of suppurative iritis following perforation, and in some cases of irido-cyclitis, where there has been much exudation into the posterior chamber and upon the posterior surface of the lens. Lastly, especially in rheumatic iritis, the inflammation may recur, each attack leading to fresh adhesions.

Diagnosis of Iritis.—The more acute form of conjunctivitis may be mistaken for an early attack of iritis. Special attention should be paid to the colour of the iris, its activity, and the condition of the pupil. Glaucoma and iritis have certain signs in common, namely, conjunctival and circumcorneal injection, a muddy appearance of the iris, and a fixed pupil. The history of onset must be carefully investigated; in glaucoma it is much more sudden, and is preceded by characteristic premonitory signs, such as coloured halos seen round artificial lights. The cornea is steamy and anæsthetic in glaucoma; the pupil is dilated rather than contracted; the anterior chamber is shallow, and there is increased intraocular tension. The diagnosis between iritis alone and iritis accompanied by cyclitis has already been discussed.

The *prognosis* of iritis, if the disease is seen early and judiciously treated, is, in the majority of cases, good, especially if it is syphilitic in origin. Rheumatic iritis is less amenable to treatment, and more liable to recur. If the pupil responds to atropine completely, a good prognosis may be made. On the other hand, the more extensive the synechiæ, especially if there is exclusion or occlusion of the pupil, the graver the condition, secondary glaucoma being very liable to be set up. Occlusion of the pupil results in loss of all useful vision save perception of light. Operative measures in such cases are alone attended with any success; the result may be very decided, but if the condition has existed for years, much improvement cannot be expected. Should the tension be subnormal, operative treatment is contraindicated. The more chronic the iritis, the more likely are atrophic changes in the eyeball to occur, and so the prognosis is bad. Sympathetic irido-cyclitis and tuberculous iritis are most serious; in the former the restoration of much sight cannot be hoped for, and in the latter enucleation is imperative. Suppurative iritis rarely exists as a local affection, except in septic infection following injury or operation, and consequently the prognosis is unfavourable.

The *treatment* of iritis and cyclitis may be considered under four headings—(1) local; (2) symptomatic; (3) constitutional; and (4) the treatment of complications and sequelæ.

(1) *Local treatment* is of paramount importance, and consists in the dilatation of the pupil, which should be effected immediately the condition is recognised. The best mydriatic is undoubtedly atropine sulphate; it is a vaso-constrictor, thus diminishing the hyperæmia and the exudation; it paralyses the ciliary muscle and the sphincter pupillæ, thus keeping the iris and ciliary body at rest; it prevents the formation of synechiæ and breaks down recent adhesions; and it is an anodyne. At the outset atropine should be used constantly, especially if any synechiæ already exist, when it should be pushed, 0·5, 0·75, or even 1 per cent solutions

of the sulphate being successively instilled into the palpebral sac every hour or oftener. This treatment is likely, if anything will, to break down the adhesions, leaving perhaps some pigment upon the lens capsule. Dots of pigment seen in front of the lens are always indicative of previous iritis. If the adhesions have become organised, the atropine will not break them down, but it will prevent the formation of others. When once the pupil has been well dilated, the amount and frequency of atropine administered should be diminished; a solution of 0·25 per cent should now be used three times a day.

The use of atropine occasionally gives rise to atropine irritation, or even atropine poisoning. The former consists of irritable conjunctivitis, and of swelling and erythema of the eyelids and cheek. When this arises the atropine must be stopped at once, and some other mydriatic carefully substituted. For this purpose a $\frac{1}{2}$ per cent solution of sulphate of duboisine, or a 5 per cent solution of hyoscyamine, or a 0·2 per cent solution of scopolamine (formerly known as hyoscine) may be used. Care should also be taken to compress the lachrymal sac with the fingers for a minute or two after the instillation, so as to prevent the fluid from passing down to the nose and pharynx.

*Atropine poisoning* is recognised by heat and dryness of the mouth and throat, difficulty in swallowing, thirst, and loss of appetite. The face becomes hot and flushed, the pulse is quickened, and the patient may have convulsions, or hallucinations, or be delirious. The pupil of the other eye will be dilated. The atropine must be stopped at once, strong coffee administered, together with a hypodermic injection of a quarter of a grain of morphia or half a grain of pilocarpin.

Besides atropinism there are other contra-indications for the use of atropine and other mydriatics. In cases of irido-cyclitis not only is the tension somewhat raised, but the instillation of atropine is sometimes followed by increased pain, photophobia, and lachrymation. In these cases the atropine solution must be either very weak or replaced by a weak solution of physostigmin.

(2) Of *the symptoms* that must be relieved the chief is pain. Heat, in the form of boracic or belladonna fomentations, is often a great comfort to the patient; dry heat is sometimes more effective than moist. Some patients prefer cold to heat; this may be applied in the form of iced compresses, or by means of a modification of Leiter's tubes Cocaine may be combined with the atropine; it acts as a sedative, a mydriatic, and a vaso-constrictor. Several leeches applied to the temple close to the outer margin of the orbit often relieve pain and congestion. Each leech will remove rather more than a drachm of blood. Bleeding may be encouraged for an hour after the leeches have dropped off by means of fomentations. Should there be any difficulty in stopping the hæmorrhage, the leech-bite must be touched with lunar caustic and a dossil of lint tightly bandaged over the bites. It is often seen that after the application of leeches the pupil responds better to atropine.

An important aid in the relief of pain, when other methods have failed, is the operation of *paracentesis* of the anterior chamber. It is never attended by bad results, and its value should not be overlooked, especially when the aqueous humour is turbid or contains purolymph.

Several measures are important in order that the patient may be placed under the most favourable conditions for a speedy recovery. At the outset a sharp mercurial purge should be given, and throughout the disease a daily action of the bowels must be obtained. If the inflammation is severe, he should be kept in bed in a darkened room, a large double shade or dark goggles being worn. In debilitated subjects tonics will be required, and a light but nutritious diet should be ordered, with alcohol only in strict moderation. In obstinate cases the use of hot air baths causing copious sweating is often very helpful.

(3) *The constitutional treatment* will vary with the etiology.

(*a*) *Syphilitic Iritis.*—It is extremely important that the patient be rapidly got under the influence of mercury, but before this is done certain precautions must be taken. It is important that the mouth should be in a healthy condition. Stumps should be removed, carious teeth filled or extracted, and tartar scraped away. While the drug is being taken the mouth must be kept scrupulously clean. The condition of the kidneys must be ascertained by a careful examination of the urine. During the treatment the patient must be urged to take plenty of good, nourishing food, but the amount of alcohol must be small. Chills must be guarded against, and flannel should be worn next the skin.

Mercury may be administered by inunction, inhalation, injection, or by the mouth.

Method of Inunction.—This, perhaps the best method, is performed by rubbing into the skin on the inner side of the arm or thigh, thirty to sixty grains of the blue ointment, the skin being first carefully cleansed. The ointment must be rubbed in for about ten minutes every night just before going to bed, when the patient should be well covered with blankets to produce sweating. Each morning a warm bath may be taken to remove the drug for the day, or a woollen vest may be worn over the same. This treatment should be continued for about six weeks, care being taken to watch against salivation, and after an interval of about three

weeks repeated for the same period. During the interval and subsequently mercury should be taken by the mouth.

Method of Inhalation.—Every other night the patient is subjected to the vapour of calomel mixed with steam. Seated on a cane-seated chair, and covered with blankets, he is surrounded by the vaporised calomel, and made to sweat profusely by the accompanying steam. Twenty to sixty grains of calomel should be used each time, and the baths continued till just short of salivation. They should then be stopped, and mercury continued to be given by the mouth.

Method of Injection.—The bichloride or biniodide is used for this purpose, not more than a quarter of a grain being injected into the skin or the muscles at each time. Some surgeons largely use and speak highly of intravenous injections followed up by inunctions. Lately Darier has used subconjunctival injections of bichloride of mercury, 1 in 1000, for all deep-seated inflammatory conditions of the eyes, not necessarily syphilitic in origin. Much pain, however, follows in many cases, an adhesive inflammation being set up. It has been found by Mellinger and others that subconjunctival injections of a 2 per cent solution of chloride of sodium have the same beneficial result without any ill effects. Their action depends probably on the acceleration to the lymph current which they produce, and not on any antiseptic or antisyphilitic properties the injection may have. *Vide* article "Gout," vol. iii. p. 512. I have seen several cases of syphilis treated by this recent method, apparently with very satisfactory results. If equally useful, the advantages, from convenience, etc., are very considerable.

Method by the Mouth.—In the treatment of syphilitic iritis this method is usually postponed till the patient has been well brought under the influence of mercury. It may then be continued either in the form of the pill, three grains of blue pill being prescribed, or as a mixture containing the liquor hydrargyri perchloridi with or without iodide of potassium. Treatment should be continued at intervals for eighteen months.

(*b*) *Rheumatic iritis* should be treated with salicin or salicylates combined with alkalies. Iodide of potassium is often useful. Turkish baths may be of great service.

(*c*) The pain in *gouty iritis* is sometimes completely relieved by colchicum; at other times this drug appears to be quite inert. Alkalies should be freely used and the diet carefully attended to.

(*d*) *Tuberculous irido-cyclitis* is rarely amenable to treatment, either local or constitutional; it should be treated by excision of the globe, especially where no other symptoms of tubercle can be discovered in the body.

(*e*) *Gonorrhœal iritis* should be treated with potassium iodide by the mouth, together with local applications to the urethra if any gleet is present.

(4) *Treatment of the Complications and Sequelæ.*—For annular posterior synechia, which leads to secondary glaucoma, an iridectomy must be performed to put the anterior and posterior chambers again into communication. For total posterior synechia a similar operation is usually indicated, but the prognosis is much less favourable; extraction of the lens is often necessary. Chronic recurrent iritis is often cured by an iridectomy. Isolated posterior synechiæ should be left alone, though formerly they were operated upon by either Passavant's or Streatfield's method (corelysis).

Operations on the Iris.—*Iridectomy* consists in the excision of a portion of the iris. This operation is performed for various affections of the eye; as a preliminary stage to cataract extraction, for glaucoma, occasionally for purulent infiltration, and in certain forms of ulcer of the cornea, in many cases of chronic and recurrent iritis and irido-cyclitis, and for optical purposes, as in central corneal opacities and stationary lamellar and anterior polar cataracts.

The instruments required are—(1) speculum; (2) fixation forceps; (3) a bent triangular keratome or a Graefe's cataract knife, or a bent broad needle; (4) iris forceps, or a Tyrrel's hook; (5) iris scissors; and (6) a curette.

The operation varies in detail according to the object for which it is performed. The first stage consists in opening the anterior chamber by an incision through the sclerotic close to or at the sclero-corneal junction; the second, in seizing, drawing out, and excising a portion of the iris; the third, in the "toilet" of the wound. When the operation is intended for the relief of glaucoma, or for the purpose of subduing or preventing inflammatory affections, the iridectomy should be made upwards, the coloboma being situated beneath the upper lid. In many cases, such as cataract, chronic glaucoma, and opacities of the cornea, the anæsthetic action of cocaine is sufficient; but where inflammation is actually present, as in iritis, subacute and primary glaucoma, a general anæsthetic is necessary. The patient must lie on a firm table or couch of such a height that his head reaches the level of the umbilical region of the surgeon, who stands behind the patient. The eyelids are kept open by a spring-stop speculum, and the globe is held steady by seizing the conjunctiva and subconjunctival tissue with fixation forceps at the part immediately opposite to that at which the incision is about to be made. A bent triangular keratome is then inserted into the sclerotic at from 1 to 2 mm. from the edge of the cornea, and pushed downwards across the anterior chamber, until the wound thus made is from 6 to 8 mm. wide

(Fig. 1). In doing this the instrument is first passed in obliquely, and in such a direction that if continued it would cause a wound of the iris and lens. As soon as its point is seen through the cornea the handle is slightly depressed to bring the blade into a plane anterior and parallel to that of the iris. The keratome is now steadily withdrawn, its apex being kept well away from the plane of the iris and lens.

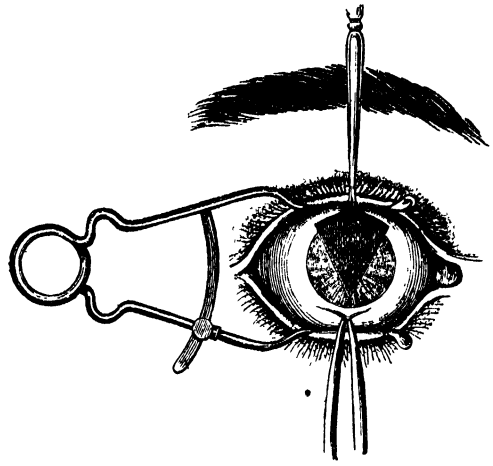


FIG. 1.—The incision with keratome.

The closed iris forceps are now passed into the anterior chamber (Fig. 2), the iris seized near its pupillary edge, dragged just outside the wound, and the projecting portion cut with one snip of the scissors; if necessary, an assistant with fixation forceps slightly rotates the globe downwards without undue pressure or traction. Should, however, a large iridectomy be indicated, the iris is dragged just outside one angle of the

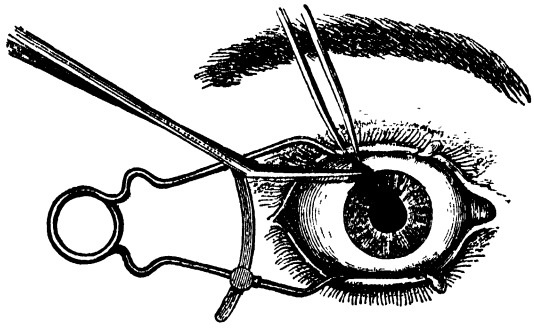


FIG. 2.—The iridectomy.

wound, and a snip made through its outer part; the portion held in the forceps is then drawn gently across to the other angle, and the excision completed as near to its periphery as possible. Finally, the curette should be passed into the edges of the wound, to liberate any portion of the iris that is entangled there, and the edges of the wound are brought into exact apposition. The speculum is removed, the eyelids gently closed, and a small pad of aseptic gamgee tissue applied by adhesive plaster.

When the anterior chamber is very shallow the danger of wounding the lens is lessened by the use of a Graefe's knife. Some surgeons always make use of this knife in performing iridectomy.

When the operation is required for optical purposes only, the position of the new pupil necessarily depends upon that of the lesion of the lens or cornea. The best position when possible is downwards and slightly inwards, or straight downwards. Only so much of the iris as is necessary for distinct vision should be removed (Fig. 3). Instead of the large bent keratome a broad bent needle is employed. The position of the incision must depend upon the situation in which the new pupil is required.

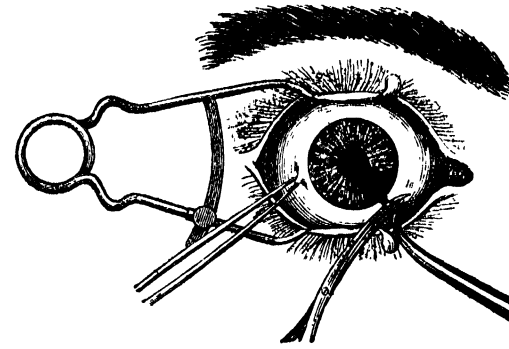


FIG. 3.—Optical iridectomy.

When this is only slightly excentric it can be made just within the margin of the cornea. When the pupil is required to be opposite the corneal margin the incision must be made in the sclerotic, about 1 mm. beyond the sclero-corneal junction. The width of the incision in either case should be at least 3 mm. Instead of iris forceps a Tyrrel's hook may be used. This is introduced on the flat as far as the centre of the pupil, half rotated downwards, so as to catch the pupillary edge of the iris, then once more turned on the flat, and withdrawn, by which means the iris is extracted.

*Accidents and Complications of Iridectomy.*—(1) The lens may be wounded by the keratome.

(2) The blade of the keratome may get between the corneal layers instead of passing directly into the anterior chamber. This accident arises from it being held too obliquely at the commencement of the incision. As soon as this is discovered the instrument should be immediately withdrawn, and another position selected for a fresh incision. If, however, the blade has finally entered the anterior chamber, so as to cause escape of the aqueous, the eye had better be closed at once, and the operation postponed for twenty-four hours, that a re-secretion of the aqueous may take place. Without this precaution the iris and lens are

so pushed forward after the escape of the aqueous that they are sure to be wounded at the time of the fresh incision.

(3) When the incision is made in the sclerotic there may be considerable hæmorrhage into the anterior chamber either before or after the excision of the iris. The blood can usually be made to flow out by depressing the upper lip of the wound with the curette. When the excision of the iris is completed no anxiety need be felt on account of the presence of a moderate amount of blood in the anterior chamber, as it usually becomes absorbed in a few days.

*Iridotomy* consists in the formation of an artificial pupil by simple incision of the iris. It can only be safely adopted when the lens is absent, and is mostly applied to those cases in which the iris has become tightly drawn upwards towards the cicatrix, as the result of inflammation after cataract extraction.

The eyelids being separated and the globe steadied, as in the previous operations, a small keratome is passed through the upper part of the cornea about 2 mm. from the sclero-corneal junction; it is then pushed onwards through the membranous exudation to the back of the iris, and finally withdrawn. The iridotomy scissors with closed blades are now passed through the corneal wound. When they reach the iris one blade is passed behind and the other in front of that structure, which is now divided by a single snip from above downwards. In some cases it is necessary to make a second incision at an acute angle with the first, so as to include a V-shaped piece of iris, which can either be left to atrophy or removed.

Tumours of the Iris and Ciliary Body.—Primary sarcoma of the iris is very rare. About half of the recorded cases are melanotic. The diagnosis of the leucotic variety from tubercle is impossible; the melanotic form may be mistaken for melanoma, which, however, is stationary and non-vascular. Metastasis is common. The eye should be enucleated as early as possible.

Primary sarcoma of the ciliary body is even more rare.

Primary Carcinoma of the Ciliary Body.—A few cases of this disease have been recorded. The cells are pigmented, and are probably derived from the ciliary glands.

Tubercle and gumma of the iris and ciliary body are described under inflammatory conditions.

Cases of melanoma and nævus of the iris have been recorded.

Cysts of the iris are of three varieties, all of which are rare.

(1) *Epithelial Cysts.* — These are probably always implantation cysts, in some case a hair, in others corneal epithelium, being carried by a perforating injury into the iridic stroma. They are lined by squamous epithelium and contain epithelial débris. They appear some months or even years after the injury, grow slowly, press on the cornea, producing opacity, and encroach on the pupil, causing loss of sight. Ultimately they may set up glaucoma. When small they should be removed by an iridectomy. If not seen till late, enucleation or evisceration may be necessary.

(2) *Serous cysts* differ pathologically from epithelial cysts, though their mode of origin is not definitely known. Some suppose that they are formed from the crypts on the anterior surface of the iris, the outlets being occluded by chronic inflammatory exudation; others consider their formation to be secondary to the formation of synechiæ. Microscopically they are lined by a single layer of epithelium. Their course, symptoms, and treatment are similar to those of epithelial cysts.

(3) Cysts have been described as occurring between the two layers of the uvea. Iritis usually supervenes.

Injuries to the Iris and Ciliary Body.[1]—Severe contusion of the eyeball without perforation may injure the iris in several ways. *Rupture of a blood-vessel* may be the result of the blow. This is characterised by the presence of blood in the anterior chamber (hyphæma). The eye must be carefully examined for other lesions of the iris and of other structures, but it may be necessary to wait till the blood has become absorbed. Should other lesions be absent the prognosis is good, and the blood will be absorbed in a few days. The eye should be kept at rest by atropine and a shade, and no reading should be attempted. Very rarely iritis supervenes.

*Traumatic mydriasis* is probably always due to some laceration of the iris, and is consequently usually accompanied by hyphæma. The laceration is radial, and may only implicate the pupillary border, being often so minute as to require high magnification before it can be seen. Paralysis of the sphincter pupillæ, *iridoplegia*, results. Combined with it may be paralysis of accommodation, *cycloplegia*. Iridoplegia may occur without cycloplegia, but traumatic ciliary paralysis alone is rare. The treatment consists in rest in bed and the application of cold. Atropine should not be used unless iritis, which is very uncommon, is threatened. Later, pilocarpine is indicated. The prognosis must be guarded, as the condition is often permanent.

*Irido-dialysis*, or detachment of the iris from the ciliary body, is usually only partial. The ciliary portion of the iris is its weakest part, and consequently is subjected to the greatest strain when the cornea is flattened by a blow. Hyphæma is always present, and, if severe, may hide the detachment. The circulus arteriosus iridis major is probably ruptured, together with

[1] See also "Eyeball, Injuries of," vol. iii.

Schlemm's canal. The appearance is that of two irregular pupils. Ophthalmoscopically the ciliary processes and the suspensory ligament of the lens, unless this has been ruptured, may be seen. The prognosis is not good, as the condition is usually permanent, though few cases of spontaneous cure have been reported. The treatment should be iced compresses and atropine, which will help to approximate the edges of the gap.

*Traumatic aniridia or irideremia* is the name given to total iridodialysis. The totally detached iris floats forwards and sinks to the lower part of the anterior chamber.

*Retroflexion and anteflexion* are very rare dislocations of the iris, and are usually only partial. In retroflexion a part of the iris is doubled back so as to cover the ciliary processes, the suspensory ligament being ruptured with dislocation of the lens. The appearance differs from that of a coloboma, since the ciliary processes cannot be seen. In anteflexion the ciliary border of the iris is not only detached from the ciliary body, as in irido-dialysis, but is doubled forwards over the rest of the iris, the posterior pigmented layer coming into view.

Penetrating wounds of the anterior part of the eyeball, unless quite small, are usually followed by prolapse of the iris, or of the ciliary body, or of both.

*Traumatic Prolapse of the Iris.*—This is especially found in wounds of the sclero-corneal junction. Besides the prolapse of the iris the lens may be injured. Strict antiseptic precautions should be taken in the treatment. The patient should be put to bed, and the wound carefully cleansed with perchloride of mercury lotion, 1 in 5000. The protruding portion of the iris must be excised, and the edges of the wound freed as much as possible from the iris to prevent the formation of anterior synechia. Atropine must be instilled into the eye three times a day. These cases often do well, very useful vision being regained. Should pyogenic organisms enter the eye, however, at the time of the injury or subsequently, suppurative iritis is very liable to be set up, followed by panophthalmitis.

*Traumatic prolapse of the ciliary body* is of much more serious import. If only slight it may be possible to suture the sclerotic or conjunctiva over it, but on no account should it be removed, as vitreous will then escape. If at all extensive, with collapse of the eyeball, there is very great danger of sympathetic ophthalmitis being set up. Consequently the eye should be removed without delay.

Perforation of the eyeball with a *foreign body* in the iris or ciliary body is very serious, since it is imperative that the foreign body should be removed as soon as possible. If this is impossible the eye must be enucleated or eviscerated without delay. Of the two a foreign body in the iris is much the more favourable. The diagnosis is seldom difficult. It is best removed through an incision at or near the periphery of the cornea. If magnetic, the nozzle of an electro-magnet should be introduced through the incision.[1] If not magnetic, or if the electro-magnet fails to remove it, it is well to proceed very cautiously in attempting its removal by means of small forceps. In attempting to remove a small particle of non-magnetic substance from the iris it is safer to remove a portion of the iris as well as the foreign body. The small iris forceps should be introduced through an incision of the cornea made in the vicinity of the particle as far away from the centre of the cornea as possible. Each blade of the forceps should then be made to grip the iris on either side of the particle, so as to include the latter in iris tissue; the forceps are then withdrawn through the incision, and the iris cut off with iris scissors as in ordinary iridectomy, when with good luck the particle will be found inside the excised portion of iris. The temptation is to try and pick the foreign body off the iris; but this usually fails, and causes immediate hæmorrhage into the anterior chambers, so that the particle is obscured, and is probably detached from its first position.

A foreign body in the ciliary body is often difficult to diagnose, and always extremely dangerous. Occasionally the history is sufficiently definite to make the diagnosis certain, but where this is not the case recourse must be had to other methods of investigation. During the last few years the discovery of the Röntgen rays has caused great strides to be made not only in the diagnosis of, but also in the exact localisation of foreign bodies in the eye and orbit. When thus localised the body must be cut down upon through the sclerotic with strict antiseptic measures, extracted, and the incision sutured. Another method, applicable, however, only to magnetic substances, is by the use of the large electro-magnet of Haab, which will often bring foreign bodies from the ciliary body into the anterior chamber, or even completely out of the eyeball. But in spite of these recent additions to the diagnosis and treatment, this condition remains very serious, and often necessitates evisceration or enucleation.

Other results of a penetrating injury are *implantation* and *serous cysts*, which are referred to previously (p. 46).

**Irish Moss.**—A seaweed having a slight nutritive value; it contains *pectin* ($C_{32}H_{40}O_{28}\cdot_4H_2O$). *See* CARRAGEEN; INVALID FEEDING (*Diabetes*).

**Irisin.**—A carbohydrate, somewhat similar to inulin, obtained from the rhizome of *Iris*

[1] See also "Eyeball, Injuries of," vol. iii.

*pseudacorus* or *Gladiolus luteus*; *iridin* is also called *irisin*.

**Iritis.**—Inflammation of the iris. *See* IRIS AND CILIARY BODY; *see also* ADRENALIN (*Uses*); CATARACT (*After Treatment of Extraction, Infection*); CORNEA (*Interstitial Keratitis, Complications*); GOUT (*Eye Symptoms*); HERPES (*Complications*); MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*Symptoms, Eyes*); RHEUMATISM, ACUTE (*Symptoms*); RHEUMATISM, CHRONIC (*Clinical Features*); SYPHILIS (*Acquired, Secondary, Tertiary, in Children*); TYPHOID FEVER (*Ocular Complications*).

**Iron.** *See* FERRUM; *see also* ANÆMIA; CHILDREN, CLINICAL EXAMINATION (*Examination of Fæces*); CHLOROSIS; DIET (*Mineral Constituents of Food*); DRUG ERUPTIONS (*Papular*); PHARMACOLOGY; PHYSIOLOGY, PROTOPLASM (*Ferro-Proteids*); PHYSIOLOGY, BLOOD (*Constituents*); PIGMENTS OF THE BODY AND EXCRETA (*Melanins*); TOXICOLOGY (*Perchloride of Iron*); URINE, PATHOLOGICAL CHANGES IN (*Inorganic Constituents, Metals*).

**Irreducible Hernia.** *See* HERNIA (*Conditions of, Irreducibility*).

**Irrespirable Gases.**—Gases which, when breathed, cannot support life, or which cannot be breathed on account of their causing spasm of the glottis.

**Irrigation.**—The cleansing of a part, external or internal, by a continuous slender stream of water; also, a method of the disposal of sewage. *See* ASEPTIC TREATMENT OF WOUNDS (*Irrigation*); BLADDER, INJURIES AND DISEASES (*Chronic Cystitis, Treatment*); ENEMATA (*Irrigation of the Bowel*); INTESTINES, SURGICAL AFFECTIONS OF (*Intestinal Obstruction, Treatment*); SEWAGE; SEWAGE AND DRAINAGE (*Sewage Farms*).

**Irritability.** *See* PARALYSIS (*Spastic, Muscle Tonus, Myotatic Irritability*); PHYSIOLOGY, TISSUES (*Muscle, Physical Characters*).

**Irritable.**—Capable of being irritated and thrown into spasm, generally painful, *e.g.* irritable bladder (*see* URINATION, DISORDERS OF, *Frequency of Micturition*); irritable ulcer (*see* ULCERS AND ULCERATION, *Irritable Condition*); and irritable spine (*see* NEURASTHENIA, *Spinal Irritation*). Theoretically, inflammation is regarded as absent.

**Irritants.**—The term irritants is usually applied to drugs which, by their local action, cause redness of the skin (dilatation of the vessels), and it includes rubefacients, vesicants, pustulants, and caustics (*e.g.* ammonia, ether, iodine, croton oil, etc.). Irritant poisoning is that due to the ingestion of such irritants as Arsenic, Antimony, Mercury, Lead, Copper, Silver, Zinc, and Chromium (*see* TOXICOLOGY, *Irritants*); and, strictly speaking, it does not include the action of the corrosives, such as the mineral acids, carbolic acid, and the alkalies.

**Isaconitina.**—Isaconitine or napelline is an amorphous alkaloid, obtained from the *Aconitum Napellus*. *See* ALKALOIDS.

**Isadelphia.**—That type of double monster in which there are two symmetrically developed and united fœtuses attached only by soft tissues.

**Isapiol.**—An isomer of apiol ($C_{12}H_{14}O_4$), which it closely resembles. *See* APIOL.

**Isatropyl-Cocaine.**—A poisonous base ($C_{19}H_{23}NO_4$) found in coca, and differing chemically from cocaine in having an isatropyl group instead of a benzoyl one; cocamine.

**Ischæmia.**—A condition of local and partial anæmia (Gr. ἴσχω, I check, and αἷμα, blood).

**Ischæmic Paralysis.** *See* MUSCLES, DISEASES OF (*Changes in Muscles due to Vascular Paralysis*).

**Ischia.** *See* MINERAL WATERS (*Muriated Saline*).

**Ischiadelphia.**—The type of double monster in which the fœtuses are united by the pelves (Gr. ἰσχίον, the hip-joint).

**Ischiagra.** Gout in the hip-joint. *See* GOUT.

**Ischialgia.**—Pain in the hip-joint. *See* HIP-JOINT, INJURIES AND DISEASES OF.

**Ischidrosis.**—Suppression of perspiration (Gr. ἴσχω, I check, and ἱδρώς, perspiration).

**Ischio-.**—In compound words *ischio-* means relating to the ischium or to the hip, *e.g.* the ischiococcygeus muscle.

**Ischiocele.**—A hernia protruding through the great sciatic notch.

**Ischiomelus.**—A type of double monster in which the parasitic fœtus consists of an accessory limb attached to the pelvis of the autosite (*e.g.* Dos Santos, the "human Tripod.")

**Ischiopagus.**—A type of symmetrical double monster in which the twins are united by the innominate bones, and in which there is a single pelvic cavity; lecanopagus.

**Ischium.**—The posterior and inferior part of the innominate bone, originally constituting a separate portion in early antenatal life and entering into the composition of the acetabulum.

**Ischl.** *See* BALNEOLOGY (*Austria, Muriated Waters*); MINERAL WATERS (*Muriated Saline*).

**Ischochymia.** — Suppression of the gastric secretion.

**Ischolochia.**—Suppression of the lochial discharge. *See* PUERPERIUM, PATHOLOGY (*Clinical Features, Septicæmia*).

**Ischuria.** — Retention of urine. *See* HYSTERIA (*Disorders of the Urinary System*); NEURASTHENIA (*Visceral Neurasthenia*).

**Isinglass.** — Fish glue or ichthyocolla, prepared from the swimming bladder of the sturgeon (*Acipenser huso*); it consists of gelatine; it may be added to tea, soup, etc. in cases of diarrhœa.

**Isle of Wight.** *See* THERAPEUTICS, HEALTH RESORTS (*English*).

**Iso-.**—In compound words *iso-* (Gr. ἴσος) has the meaning of equal, *e.g.* isobutane, an isomeric form of butane, etc.

**Isobars.** — Lines drawn on maps "connecting places on the earth's surface at which the barometric pressure is the same in a given time, or on the average for a given period" (*New English Dictionary*).

**Isobe.** *See* BALNEOLOGY (*Japan*).

**Isochronous.**—Taking place at equal periods of time or at equal intervals.

**Isoform.** — Para-iodo-anisol, $C_6H_4(OCH_3)IO_2$, a white powder, used internally as a gastric and intestinal antiseptic (dose, 8 grains), and externally in ulcers, syphilitic skin diseases, etc.; it is not employed in the pure state but mixed with calcium phosphate, for alone it is explosive.

**Isogens.**—The lines in diagrams showing the various combinations of the ages of the parents which are associated with the same average birth-rate (*New English Dictionary*); "in natality tables, the ages of the father and the mother take the place of the latitudes and longitudes in weather charts, and lines of similar birth-rates, or, as I would call them, 'isogens,' take the place of isobars" (*F. Galton*, 1894).

**Isolation.** *See* DIPHTHERIA (*Isolation and Disinfection*; SMALLPOX (*Isolation Houses*).

**Isomer and Isomeric.**—In chemistry this term is applied to two substances which contain the same elements in the same proportions but with the atoms differently arranged in the molecules, and which have different properties; chemical *isomerism* is the condition of being thus isomeric, and the substances are called *isomers*. An *Isomere*, however, is a segment of a limb in one species of animal homologous to a segment in another species.

**Isometric Method.** — A method of recording the force of contraction during different parts of the contraction period by making the muscle pull upon a strong spring, the bending of which is recorded by a long lever; by the *isotonic* method the muscle acts on a light lever. *See* PHYSIOLOGY, TISSUES (*Muscle*).

**Isometropia.**—The condition in which the refraction of both eyes is the same.

**Isopral.** — Trichlor-isopropyl-alcohol, a useful hypnotic, given in doses of 4 to 30 grs.; it is a white crystalline substance, soluble to some extent in water but more easily in alcohol and ether, and it has a somewhat unpleasant bitter taste; it is said to be contraindicated in cases of gastric or cardiac disease.

**Isotachiol.**—A silver salt, with marked antiseptic powers, said to be more stable than tachiol.

**Isotherms.**— Lines drawn on maps or charts through points on the earth's surface having the same mean temperature; isothermal lines.

**Isotonic.**—In Physiology a distinction is drawn between isometric and isotonic conditions. "If, before and during excitation, its opposite attachments are so fixed that they cannot be brought nearer together by the effort of the muscle to contract, the excitation of the muscle is said to occur under isometric conditions. If, on the other hand, one end of the muscle is left free, so that it can shorten on excitation, and in so doing lift a weight which is attached to it, the excitation is said to take place under isotonic conditions." (Sir J. Burdon-Sanderson in Schafer's *Physiology*.)

**Isotropy.** — The condition of being equally endowed with any particular quality in all parts; thus isotropy of the ovum means that the substance of it contains no parts specially set aside to form definite structures in the embryo. The opposite condition is *anisotropy*, in which, in the case of the ovum, each organ, tissue, and cell of the embryo is regarded as represented by a distinct rudiment.

**Ispaghula.**—Ispaghula or SPOGEL SEEDS (*Plantago ovata*) are official in the Indian and Colonial Addendum to the British Pharmacopœia of 1898; the dose is from 50 to 150 grs., and the official preparation is the *Decoctum Ispaghulæ* (dose, ½ to 2 fl. oz.); the decoctum is a good demulcent, and the seeds can be used like linseed to form poultices, for they contain much mucilage.

**Issue.**—An ulcer made artificially for the purpose of discharging and continuing to discharge, and so to act as a counter-irritant.

**Isthmus.**—A narrow portion or passage connecting together two parts of an organ of the body, *e.g.* the isthmus of the thyroid gland, or of the uterus, or of the Eustachian tube.

**Italy.** *See* BALNEOLOGY; THERAPEUTICS, HEALTH RESORTS.

**Itch.** *See* SCABIES.

**Itch, Cuban.**—A mild form of smallpox, regarded at first as a new disease.

**Itch, Dhobie.** *See* SKIN DISEASES OF THE TROPICS (*From Vegetable Parasites, Burmese Ringworm*).

**Itching.** *See* PRURIGO AND PRURITUS; *see also* DIABETES MELLITUS (*Symptoms, Skin*); GOUT (*Cutaneous System*); NEPHRITIS (*Chronic, Symptoms, Nervous*).

**Iter.**—A narrow passage leading from one cavity of an organ to another, *e.g.* the aqueduct of Sylvius in the brain leading from the third to the fourth ventricle.

**Itrol.**—The citrate of silver, an antiseptic salt used in the treatment of gonorrhœa and of some skin diseases.

**Ixodes.**—A variety of acarus; wood-ticks. *See* SCABIES (*Other Acari*).

**Ixodiasis.** — Tick-fever, due to a tick (probably *Argas Acari*). *See* SCABIES (*Other Acari*).

**Izal.**—A hydrocarbon (dark brown liquid) obtained from coal, acting as an antiseptic; it has been used in dysentery as an intestinal injection, and as a disinfectant for typhoid stools; it has a characteristic "sheep-dip" smell.

**Jaborandi.** *See also* DIAPHORETICS; ECLAMPSIA; PHARMACOLOGY; etc. — Jaborandi Folia, the dried leaflets of *Pilocarpus jaborandi*, from South America. The most important constituents are—1. *Pilocarpine*, a colourless syrupy alkaloid, insoluble in water. The leaves should contain from ½ to 1 per cent. of pilocarpine. 2. *Jaborine*, a liquid alkaloid, with an action resembling that of atropine and, therefore, antagonistic to pilocarpine. 3. A *volatile oil*. *Preparations*—1. Tinctura Jaborandi. *Dose*—½-1 ʒ. 2. Extractum Jaborandi Liquidum. *Dose*—5-15 m. 3. Pilocarpinæ Nitras, a white crystalline powder, soluble 1 in 9 of water, and 1 in 50 of alcohol. *Dose*—$\frac{1}{20}$-½ gr. by the mouth; $\frac{1}{10}$-⅓ gr. hypodermically.

Pilocarpine has been employed externally as an ointment or lotion to promote the growth of the hair. On account of its myotic influence on the pupil it is of great value in the treatment of some eye conditions, especially those associated with increased intraocular tension. It is dropped into the eye in solutions of from ¼-4 grs. of pilocarpine nitrate to the ounce of water. It is less powerful in its action than eserine, and is especially indicated where the treatment has to be kept up for a long time. The daily administration by hypodermic injection of $\frac{1}{10}$ gr. of pilocarpine is recommended in the treatment of opacities of the vitreous humour. It is also said to be a good remedy in tobacco and alcoholic amblyopia. Internally, pilocarpine has been and still is widely used as a diaphoretic in Bright's disease and in uræmia. The profuse diaphoresis aids in the elimination of the uræmic poison; and the extraction of fluid from the body and the lowering of the general blood pressure remove the strain from the kidneys. For this purpose it is usually given hypodermically in a dose of ⅛ gr., the patient being wrapped up in blankets, surrounded by hot bottles, and given hot drinks. On account of the depressant effect of the drug, strong coffee or other stimulant should be given at the same time. In children and in the aged, and in patients of any age who exhibit signs of cardiac weakness, pilocarpine is absolutely contraindicated. Where there is present any œdema of the lungs it must not be given, as the great increase of this œdema caused by pilocarpine may rapidly prove fatal, especially if there is cardiac weakness as well. Within recent years the tendency has been to employ other methods of inducing diaphoresis in kidney disease. In doses of $\frac{1}{20}$ gr. pilocarpine may, by stimulating the atonic sweat glands, check profuse night sweats where atropine has failed. In cases of aural vertigo the hypodermic injection of pilocarpine every few days, in sufficient dose to produce slight salivation, is stated to give good results. It has been strongly recommended for the relief of itching in jaundice, administered hypodermically in doses of from $\frac{1}{12}$ to $\frac{1}{6}$ gr. Its use in the treatment of pleurisy with effusion has been entirely abandoned. Nitrate of pilocarpine is greatly preferable for general puposes to the tincture and fluid extract of jaborandi; the latter are more uncertain in composition, they cause more nausea and depression, and they contain jaborine, an antagonistic alkaloid.

**Jacket, Plaster.**—A jacket made of plaster-of-Paris, and used in cases of spinal disease to form a rigid support for the head and shoulders, and so take the direct pressure off the spine. *See* SPINE, SURGICAL AFFECTIONS (*Spinal Caries, Treatment*).

**Jacket, Straight.** — A short coat reaching to the waist, made of strong material, used for the purpose of confining the arms of the insane; it may or may not have sleeves; the straight-waistcoat or camisole.

**Jacksonian Epilepsy.**—An epileptic condition in which the convulsions are due to a lesion of the cerebral cortex, are localised at first, becoming general later, and are often unaccom-

panied by unconsciousness; it was first described by J. Hughlings Jackson. *See* BRAIN, TUMOURS OF (*Symptoms, Convulsions*); BRAIN, SURGERY OF (*Intracranial Inflammation, Leptomeningitis*); HYSTERIA (*Hysterical Convulsions, Diagnosis*); PHYSIOLOGY, NERVOUS SYSTEM (*Cerebrum, Discharging Mechanism*).

**Jacobson's Nerve.** *See* FIFTH NERVE; GLOSSO-PHARYNGEAL NERVE; PHYSIOLOGY, FOOD AND DIGESTION (*Digestion in the Mouth, Parotid Gland*).

**Jacquemier's Test.**—The presence of a purple discoloration of the vulvar and vaginal mucous membrane, used as an indication of the existence of pregnancy. *See* PREGNANCY, PHYSIOLOGY (*Local Changes, Vagina and External Genitals*).

**Jactation or Jactitation.**—A restless tossing about, seen in fevers and after excessive hæmorrhage (*e.g.* post-partum bleeding).

**Jaeger's Test-Types.** *See* EYE, CLINICAL EXAMINATION OF (*Visual Acuity*).

**Jaffe's Test.** *See* URINE, PATHOLOGICAL CHANGES IN (*Indicanuria, Detection*).

**Jail Fever.** *See* TYPHUS FEVER (*History*).

**Jalap.** *See also* PHARMACOLOGY; PURGATIVES; etc.—Jalapa, the dried tuberous root of *Ipomœa purga*, a native of Mexico. It contains two resins, *jalapin* and *convolvulin*, neither of which is used by itself therapeutically. The root should contain 9 to 11 per cent of the resins. *Dose* — 5-20 grs. *Preparations* — 1. Extractum Jalapæ. *Dose*—2-8 grs. 2. Pulvis Jalapæ Compositus. *Dose* — 20-60 grs. 3. Tinctura Jalapæ. *Dose* — ½-1 ʒ. 4. Jalapæ Resina. *Dose*—2-5 grs. Jalap is contained in Pulvis Scammonii Compositus, and jalap resin in Pilula Scammonii Compositus. Jalap is a powerful hydragogue purgative, and causes a large outpouring of fluid into the intestinal canal, and a copious watery evacuation about four hours after administration. It does not gripe so much as scammony. It is given whenever it is desired to withdraw large quantities of fluid from the body, notably for the relief of dropsy of any origin. It is an excellent cathartic to employ in many cases of Bright's disease, in uræmia, in eclampsia, and in general plethora with cerebral congestion. It must be used with great caution in old or feeble people, and is contraindicated when gastric or intestinal irritation is present.

**James's Powder.**—Pulvis antimonialis is the official substitute for this powder; it consists of antimonious oxide, 1 part, and calcium phosphate, 2 parts; the dose is from 3 to 6 grains. *See* ANTIMONY.

**Janet's Method.**—The treatment of gonorrhœa by irrigation.

**Janiceps or Janus.**—The teratological type in which the double monster has a single head with two faces, placed opposite to each other; both faces may be perfect (*J. teleus*), one may be imperfect (*J. ateleus*), *e.g.* one may have a single median eye (*J. cyclopus*). *See* TERATOLOGY.

**Japan.** *See* BALNEOLOGY (*Vapour Bath; Thermal Waters*); SKIN DISEASES OF THE TROPICS (*Shima Mushi, River Fever*).

**Jarisch's Ointment.**—An ointment containing 60 grains of pyrogallic acid to the ounce of lard; it is used in chronic psoriasis. *See* PYROGALLIC ACID.

**Jasminum.** *See* GELSEMII RADIX; TOXICOLOGY (*Alkaloids and Vegetable Poisons, Gelsemium*).

**Jaundice.**


DEFINITION . . . . . 52
CAUSATION . . . . . 52
PATHOLOGY . . . . . 52
TOXÆMIC OR HÆMOHEPATOGENOUS JAUNDICE—
*Clinical Characters and Diagnosis* . 53
OBSTRUCTIVE JAUNDICE—
*Signs* . . . . . 54
*Symptoms* . . . . . 55
DIAGNOSIS OF JAUNDICE FROM OTHER CONDITIONS . . . . . 55
CAUSES AND DIFFERENTIAL DIAGNOSIS OF OBSTRUCTIVE JAUNDICE . . . . 56
PROGNOSIS OF JAUNDICE . . . . . 58
TREATMENT . . . . . 59
JAUNDICE OF PHOSPHORUS POISONING . 60


*See also* ADRENAL GLANDS, ADDISON'S DISEASE (*Diagnosis, Urobilin Jaundice*); ANÆSTHESIA, CHLOROFORM (*After-Management, Jaundice*); ASCITES (*Recognition of Cause*); BLACKWATER FEVER (*Symptoms*); GALL-BLADDER AND BILE DUCT, DISEASES OF (*Cholelithiasis, Cholangitis, Tumours of Bile Ducts*); HÆMOGLOBINURIA; HEART, AFFECTIONS OF MYOCARDIUM AND ENDOCARDIUM (*General Pathology, Secondary Conditions, Jaundice*); ICTERUS; KIDNEY, SURGICAL AFFECTIONS OF (*Movable and Floating Kidney, Intermittent Jaundice*); LABOUR, OPERATIONS (*Induction of Premature Labour, Indications*); LIVER, DISEASES OF (*Hepatoptosis; Cirrhosis, Signs of; Hepatic Tuberculosis; Weil's Disease; Perihepatitis; Hyperæmia and Menstrual Jaundice*); MUMPS (*Complications*); NEPHRITIS (*Renal Cirrhosis*); NEW-BORN INFANT (*Diseases, Icterus Neonatorum*); OVARIES, DISEASES OF (*Ovariotomy, Complications following*); PANCREAS, DISEASES OF (*Pancreatitis, Chronic, Malignant Disease of Pancreas*); PHOSPHORUS; PNEUMONIA, CLINICAL (*Complications, Jaundice*); PREGNANCY, AFFECTIONS AND COMPLICATIONS

(*Jaundice*); PRURITUS (*Causation*); PURPURA (*Symptomatic*); TOXICOLOGY (*Non-metallic Elements*); TYPHOID FEVER (*Complications and Sequelæ*).

DEFINITION.—Jaundice is the condition due to the presence of bile pigment in the blood, and is recognised clinically by staining of the skin, conjunctivæ, mucous membranes, and urine by bile pigment.

CAUSATION.—The formation of bile pigment from hæmoglobin is carried on by the cells of the liver, for, as shown by experiments on animals, this transformation does not take place when the liver is removed or, what comes to the same thing, when all the vessels going to it are ligatured. Jaundice, or the presence of bile pigment in the blood, is due to the bile manufactured by the liver passing into the circulation instead of into the intestines. This miscarriage of the bile may occur either directly the bile pigment is formed by the liver cells, *i.e.* before the bile enters the bile ducts, or later after it has entered the ducts.

In cases of long-standing biliary obstruction, where the dilated intrahepatic ducts only contain mucus, the bile formed by the liver cells passes almost directly into the adjacent lymphatics, thence into the thoracic duct, and so reaches the general circulation. Cases of this kind, in which no bile is visible in the ducts, were formerly thought to prove that jaundice might result from suppression of the bile-excreting function of the liver, and be due to the resulting accumulation in the blood of bile pigment formed in the circulation from hæmoglobin. The presence of bile pigment in the liver cells in such cases shows that the formation of bile still continues.

When there is obstruction in the ducts the bile passes from the bile capillaries or ducts into the lymphatic vessels of the liver, and not into the blood-vessels. This has been shown by the experiments of Saunders early in this century (1809), and later by Fleischl (1874), V. Harley (1892), and Szubinski (1899). It has also been shown that jaundice due to ligature of the bile duct may be removed or prevented by ligature of the thoracic duct. Obstruction to the flow of bile through the ducts leads to a rise of the normally low pressure of bile, and as a result the bile passes into the lymphatics, and so into the general circulation.

This clearly explains the production of jaundice in cases where a gross mechanical obstruction in the ducts exists, but it is necessary to consider the method of causation of jaundice in those cases where there is no manifest obstruction in the larger bile ducts.

### PATHOLOGY OF JAUNDICE

Jaundice was formerly divided into (1) hepatogenous or obstructive, where there was an unmistakable obstruction in the larger ducts, and

(2) Where there was no gross obstruction visible to the naked eye. In contrast to the first category, this form was spoken of as non-obstructive. It included cases—(*a*) Where the jaundice was thought to be due to changes occurring in the blood, leading to the liberation of hæmoglobin and its transformation in the circulation into bile pigment. This constituted "hæmatogenous" jaundice. (*b*) Where an excessive secretion of bile into the intestine was followed by excessive absorption from the mucous membrane of the bowel and subsequent overflow into the general circulation. This was called jaundice from polycholia. (*c*) Where owing to suppression of the bile-excreting function of the liver cells an accumulation of bile pigments, manufactured from hæmoglobin in the blood, occurred in the circulation. This was known as jaundice by suppression.

*Consideration of "Hæmatogenous" Jaundice.*—In various toxic and septic conditions of the blood, where the hæmoglobin is liberated from the red blood corpuscles, jaundice of a slight degree of intensity is often seen. A good example of this "toxæmic" jaundice is provided in the experiments of Hunter with toluylenediamine. This poison, when introduced into the circulation, gives rise to destruction of the red blood corpuscles with liberation of hæmoglobin (hæmolysis), and to jaundice. After its administration the flow of bile is at first increased, and there is an increase in the bile pigments poured out (polychromia), owing to an augmented amount of free hæmoglobin, the antecedent of bile pigments, reaching the liver. After a time the amount secreted diminishes, and the bile becomes more viscid, until finally the flow of bile becomes almost arrested. This slowing and diminution in the flow of bile is found to depend on inflammation of the smaller intrahepatic bile ducts, which become swollen, secrete thick mucus, and are in a condition of catarrhal cholangitis due to the toxic effects of the toluylenediamine. The jaundice, therefore, is really obstructive, but being due to changes in the small intrahepatic ducts is readily overlooked. This explanation accounts for the slight jaundice accompanying morbid conditions such as septicæmia, pyæmia, hæmoglobinuric fever, pernicious anæmia, and poisoning by phosphorus, arseniuretted hydrogen, and snake-bite. This jaundice, therefore, is toxæmic, and depends on the presence of a poison in the blood which sets up inflammatory changes, and consequently obstruction in the small intrahepatic ducts. It may therefore be conveniently described as hæmohepatogenous, but not as hæmatogenous jaundice. Experiment shows that the presence of free hæmoglobin in the blood, though it leads to an increased secretion of bile pigment in the bile (poly-

chromia), does not of itself give rise to jaundice. The frequent clinical association of jaundice with conditions giving rise to destruction of red blood corpuscles is due to the fact that the hæmolysis is of toxic origin; inflammation of the small intrahepatic bile ducts is another manifestation of the toxæmia.

*The Question of so-called Jaundice from Polycholia.* — When jaundice was found to be associated with bile in the fæces it was supposed that there was such an excessive secretion of bile (polycholia), that an excessive amount of bile was absorbed from the intestines and passed through the liver into the general circulation.

Bile may be present in the fæces of jaundiced patients under various conditions; thus, in obstruction of one hepatic duct icterus results, but the other hepatic duct pours bile into the duodenum. When a calculus lies in a common duct it may act like a ball valve, and allow bile to escape into the bowel at intervals; in biliary cirrhosis bile is usually present in the fæces. In none of these examples is there any suggestion to the effect that an excessive secretion of bile exists.

In hæmohepatogenous or toxæmic jaundice there is in the early stage a secretion of bile rich in bile pigment (polychromia) and poor in bile acids, the pigment appearing in the excreta; later, obstruction in some of the small intrahepatic ducts occurs and produces jaundice. This is the explanation of so-called jaundice from polycholia. But it should be pointed out that, strictly speaking, there is not an excessive secretion of normal bile (polycholia), but only of bile pigment (polychromia), and that the bile salts, far from being increased, are diminished.

*The Question of the so-called Jaundice from Suppression of the Bile-secreting Function of the Liver.*—It was formerly supposed that the liver might, from nervous or other influences, cease to secrete bile, and that an accumulation of refuse blood-pigment in the circulation resulted, and became changed, without the intervention of the liver, into bile pigment. Emotional jaundice was instanced; but there is no proof of the liver thus striking work, and moreover, inasmuch as bile is only formed by the liver, if its bile-forming functions were suppressed jaundice could not result. This last argument, indeed, is fatal to the idea of jaundice from suppression of the bile-forming function of the liver. In acute yellow atrophy and phosphorus poisoning the jaundice is almost certainly due to obstruction in the small intrahepatic ducts from inflammation, and to the passage of bile into the lymphatics, which results as a natural consequence. In long-continued biliary obstruction the ducts contain clear mucous fluid devoid of bile, and it might be, and indeed has been, thought that the liver ceased to secrete bile under these conditions; but here the liver cells, as shown by the presence of bile pigment inside them, still manufacture bile, which passes almost directly into the lymphatic vessels and so into the circulation.

"*Urobilin Jaundice.*"—A few words may be added on this subject. Darkening of the skin may occur in the melanæmia of malaria, in hæmochromatosis, and in other conditions, but does not really resemble jaundice. It has, however, been thought that a staining of the skin indistinguishable from jaundice may be due to other pigments than those of bile. Thus in some cases where there is an excessive amount of urobilin in the urine a yellow tinging of the skin has been referred to the effects of urobilin, and not of bile, in the circulation. Excessive urobilinuria may occur without any jaundice; when they are associated together the jaundice is due to bile pigment and not to staining of the skin with urobilin. As Hunter points out, the term urobilin *jaundice* is a misnomer, and moreover is confusing and misleading.

*Classification.*—Jaundice may be divided, therefore, into—

(1) Obstructive—due to some gross obstruction to the flow of bile along the larger bile ducts.

(2) Toxæmic or hæmohepatogenous jaundice, where there is obstruction in the small intrahepatic ducts, due to poisons excreted from the blood into the bile ducts setting up cholangitis.

Jaundice is therefore always due to obstruction, and is a symptom, not a disease in itself. It may be a symptom of a purely local condition, viz. pressure on the large ducts, or, on the other hand, be the result of a general infection or intoxication setting up changes in the small intrahepatic bile ducts resulting in obstruction.

### Toxæmic or Hæmohepatogenous Jaundice

*General Characters and Distinctions from Obstructive Jaundice.*—Toxæmic or hæmohepatogenous jaundice is essentially a symptom of some underlying infection, or possibly intoxication, and is usually subordinate to its other constitutional symptoms. The patient suffers comparatively little from the direct results of bile pigment circulating in the blood, but is definitely ill from the general toxæmic state.

The jaundice is slight as a rule; in acute yellow atrophy and icterus gravis the yellow colour may be bright and marked, but the dark green or black tint of chronic obstructive jaundice does not occur.

Bile pigment is found in the urine. It has usually been stated that bile acids are either absent, or at any rate not present to the same extent as in jaundice due to mechanical obstruction of the larger ducts; but little importance can be attached to results obtained by Pettenkoffer's well-known test, since, as shown by Hunter, it is entirely unreliable as employed in ordinary clinical practice.

The motions nearly always contain bile, and thus differ from the clay-coloured stools of icterus due to obstruction.

The course of the disease is more acute as a rule than in obstructive icterus, and is not accompanied by the itching of the skin, xanthopsia or yellow vision, and slow pulse that may occur in obstructive jaundice.

Signs of constitutional disturbance, such as enlarged spleen, fever, and albuminuria, are common, while grave symptoms develop sooner and more frequently than in obstructive jaundice, where, when they do occur, it is at the termination of long-continued jaundice.

In severe cases of hæmohepatogenous jaundice the "typhoid state," with dry tongue, delirium, coma, and multiple hæmorrhages, may rapidly develop.

The important features, therefore, are the slight degree of jaundice and the presence of marked constitutional symptoms, the want of proportion between the icterus and the symptoms, and the evidence of hæmic infection or intoxication.

After Hunter, hæmohepatogenous or toxæmic jaundice may be divided into—

(i.) Toxic jaundice, due to poisons, such as toluylenediamine, arseniuretted hydrogen, phosphorus, snake poison.

(ii.) That occurring in connection with the specific fevers, such as yellow fever, pyæmia, septicæmia, relapsing fever, and malaria.

(iii.) A group containing acute yellow atrophy, icterus gravis, infectious jaundice (Weil's disease).

### Obstructive Jaundice

*Signs.*—Jaundice appears first in the conjunctiva, then successively on the face, body, and extremities. The "whites" of the eyes are the first part to show the bile pigment, but before this bile pigment is present in the urine. The masses of fat (pinguecula) often seen under the conjunctivæ frequently have a slightly yellow colour, and may mislead a careless observer into the belief that jaundice is present. A slight degree of icterus is more readily detected in a fair-skinned patient than in one of a dark complexion, in whom the skin is often somewhat sallow. It should also be remembered that jaundice is easily overlooked in artificial light. The mucous membrane of the lips and palate shows an icteric tint. When jaundice has existed for a long time the bile pigment in the skin turns of a dark green colour (biliverdin), and the skin becomes of an olive-green colour, spoken of as "black jaundice." After recovery from severe jaundice the skin is often left discoloured for a very considerable period.

In jaundice continued for more than a year the skin may show the peculiar change known as xanthoma or xanthelasma.

When as the result of complete biliary obstruction, or from other causes, the protective function of the liver fails, and toxins pass into the circulation and give rise to cholæmia or biliary toxæmia, hæmorrhages may take place into the skin and mucous membranes, giving rise to petechiæ, epistaxis, melæna, etc.

The urine is acid in reaction, somewhat diminished in amount, and becomes bile-stained before the conjunctiva or skin. There may be also a considerable excess of urobilin. Stress was formerly laid on the presence of bile acids in the urine as a point of importance in the diagnosis from jaundice occurring without manifest obstruction to the ducts. The test relied on for the detection of bile acids in ordinary clinical work, Pettenkoffer's, is so fallacious that no value can be attached to its result, and hence the question of bile acids in the urine is no longer of any practical importance. The colour of the urine varies in different cases and at different times, from an intensified yellow to brown, olive, or a very dark brown. When shaken up the froth becomes yellow. The colour must be distinguished from that in urobilinuria, hæmaturia, melanuria, and the alteration effected by rhubarb and chrysophanic acid, by senna and santonin, by employing Gmelin's test for bile pigment. When cholæmia or biliary toxæmia has supervened, the renal epithelium may be so damaged by toxins that albuminuria results.

The other secretions as a rule do not contain bile; sometimes the sweat, especially in the axilla, is undoubtedly bile-stained, but generally the perspiration is colourless. The secretion of the intestines and nose, the saliva, tears, and, in women, milk, are, in spite of statements to the contrary, free from bile. In inflammatory conditions the pathological secretions and exudations become icteric, as shown by pneumonic sputum, pleural and peritoneal effusions, and saliva in mercurial salivation.

In obstructive jaundice there is constipation, and the motions are extremely offensive. They are clay-coloured from absence of bile and the presence of an excess of fat, the digestion of which is interfered with.

Exceptionally bile may be present in the fæces, for example, when jaundice is due to obstruction to one of the two hepatic ducts, where a calculus in the common duct allows some of the bile to escape into the duodenum.

*The Circulatory System.*—In the absence of fever or pain the pulse tends to be slow; this has been ascribed to the action of the bile salts on the cardiac ganglia. It is, however, especially in catarrhal jaundice that the slowing is chiefly marked.

The blood-pressure is low. Though from the destructive (hæmolytic) action of bile acids on red blood corpuscles anæmia might be expected, it is not a marked result of jaundice itself. From muscular incompetence a mitral systolic

murmur may become audible, and from increased pressure in the pulmonary circulation due to this cause, or possibly to reflex constriction of the pulmonary vessels referred from the bile ducts, the second sound over the pulmonary artery becomes accentuated.

*The liver* is often enlarged from damming up of the bile, and may be tender. In malignant disease and cirrhosis its surface may be knobby or irregular.

Enlargement of the gall-bladder is a valuable sign of obstruction of the common duct. When chronic jaundice is due to gall-stones, the gall-bladder is collapsed from previous cholecystitis, while in cases of malignant disease pressing on the common bile duct the gall-bladder may form a tense, pear-shaped tumour. If a calculus be impacted in the cystic duct the gall-bladder may be distended with mucus, and occasionally it is enlarged from the presence of numerous calculi; these are exceptions to the general rule enunciated by Courvoisier, that in jaundice due to gall-stones the gall-bladder is collapsed.

*The spleen* is usually not enlarged, but if it is it points to biliary cirrhosis, some septic or toxic process, such as Weil's disease or infective jaundice, to syphilis, or to that extremely rare condition, alveolar hydatid.

*The temperature*, like the functions of the body generally in simple obstructive jaundice, is depressed. Fever, when present, is either due to the same cause that is responsible for the jaundice, as in toxæmic jaundice, Weil's disease, and cirrhosis, or is the result of some complication, such as cholangitis in gall-stone obstruction.

*Symptoms.*—The symptoms that may be met with accompanying jaundice are due partly to the presence of bile in the circulation, which acts as a depressing poison, partly to a secondary toxæmia due to the liver failing to perform its important protective function of stopping poison brought to it from the alimentary canal, and partly to the absence of bile from the alimentary canal. These distinctions in the causation of the symptoms must not be pressed too far, but it will be convenient to consider the symptoms under the following heads:—

*Symptoms due to Bile in the Circulation.*—Bilirubin is said to be ten times more toxic than the bile acids. It will be noticed that in cases of jaundice a considerable amount of the bile pigment is fixed in the tissues, which become thereby bile-stained; in this way the rest of the body is protected from the full toxic effects of all the bile pigment that has entered into the circulation. The tissues in which the bile pigment settles suffer; for example, in the skin the irritation of the cutaneous nerves shows itself by pruritus, which may be very troublesome and prevent sleep. Scratching thus induced may be the cause of pimples and traumatic eczema, while urticaria and lichen are sometimes seen.

A bitter taste in the mouth is often complained of, although the saliva does not contain bile pigment; this bitter flavour may depend on the presence of toxic bodies, which, owing to hepatic insufficiency, have escaped into the general circulation and passed into the saliva, and if so, would properly come in the next category.

There is often considerable impairment of appetite, with a special distaste for fatty foods.

Mental depression, low spirits, and incapacity for continued thought or application are commonly seen.

Yellow vision or xanthopsia, due to the media of the eyes being jaundiced, is sometimes found to be present on inquiry, but is hardly ever a prominent symptom. It is indeed better marked after the ingestion of santonin than in jaundice.

*Symptoms due to the Presence of Poisons other than Bile in the Circulation.*—Owing to the failure of the liver to stop poisons received from the alimentary canal, auto-intoxication results, and if the kidneys do not compensate for this by free diuresis, a toxæmic condition, analogous to that of uræmia, results. Drowsiness, delirium, and somnolence passing into coma may develop.

*Symptoms due to the Absence of Bile from the Intestinal Tract.*—Constipation is very common, and is usually explained as due to the absence of the natural purgative—the bile. Excessive fermentation with flatulence and extremely offensive motions may occur, while interference with the digestion and with the absorption of fatty food leads to an excessive amount of fat in the fæces (steatorrhœa).

Diagnosis.—As has already been pointed out, jaundice may be overlooked altogether if the patient is only seen by yellow artificial light, such as gas or candle illumination. No serious difficulty should arise in distinguishing jaundice from other pigmentary changes in the skin.

The yellow colour of patients with slight icterus is hardly likely to be confused with the bronzing of sunburn, with the natural hue of the yellow-skinned races of mankind, or with the tint of the skin in the advanced cachexia of malignant disease; but should any question arise, examination of the conjunctivæ, and of the urine for the presence of bile pigment, will quickly settle the matter.

The dark green colour of the skin in black jaundice of long-continued biliary obstruction might conceivably be confused with the pigmentation of the skin in Addison's disease, hæmochromatosis, malarial melanæmia, argyria, etc., but here again examination of the urine and conjunctivæ will prevent error.

The past effects of severe jaundice may give rise to some difficulty, but further investigation and the history of the case should make matters clear.

DIFFERENTIAL DIAGNOSIS OF JAUNDICE DUE TO VARIOUS CAUSES.—Since the distinction between obstructive jaundice and toxæmic (or hæmohepatogenous) jaundice has already been considered (p. 53), it is now only necessary to refer to the differential diagnosis of the various factors that may give rise to obstructive jaundice.

The causes of obstructive jaundice are very numerous, but for convenience they may be grouped into three classes:—

(1) Where the obstruction is due to something occluding the lumen of the bile ducts, such as a gall-stone, or parasites.

(2) Where the obstruction depends on changes originating in the walls of the larger bile ducts, *e.g.* catarrhal cholangitis.

(3) Where obstruction is due to processes arising outside the ducts and occluding them, either (i.) by direct pressure alone, or (ii.) by actually invading the tissues of the ducts.

(1) *Causes obstructing the Lumen of the Ducts.*—(*a*) A gall-stone in the common duct is the most familiar example. A history of biliary colic, often of repeated attacks, preceding the development of jaundice is the rule; sometimes, however, no history of colic is forthcoming, and the case may at first very closely resemble malignant disease; but as time goes on the jaundice is not uniformly or necessarily progressive; sometimes, as the duct dilates around the stone, the jaundice fades and may even disappear. Another point of distinction is that in cholelithiasis the gall-bladder is usually small and contracted from past inflammation of its walls, whereas in malignant disease it is often enlarged. It is sometimes found that gall-stones and malignant disease are combined, especially in the case of the gall-bladder.

When gall-stones remain long in the common duct infective cholangitis with intermittent hepatic fever may develop (*vide* "Disease of Gall-bladder and Bile Ducts," vol. iii.).

Inspissated mucus and small calculi may also give rise to slight jaundice and attacks of pain, often described as "spasms," and regarded as dyspepsia.

(*b*) *Parasites.*—A hydatid cyst may discharge into the ducts, and membrane or daughter cysts block the ducts, giving rise to biliary colic, jaundice, and often to infective cholangitis. The diagnosis depends on the history that the patient has either had a cyst which has disappeared, or that an existing one has grown smaller about the time that the symptoms appeared, or still better on the presence of bile-stained pieces of hydatid membrane in the fæces, or possibly even in the vomit.

Round worms (Ascaris lumbricoides) may enter the common bile duct from the duodenum and give rise to jaundice. Of this rare condition Mertens has collected forty-eight examples.

Distomum hepaticum, the liver fluke so fatal to sheep, has been found in the ducts of the human liver, as have in rare instances D. sinense and D. conjunctivum. In these cases the diagnosis depends on the recognition of the worms or their ova in the fæces.

(*c*) As pathological curiosities some authors have referred to the presence of foreign bodies, such as needles, in the bile ducts.

(2) *Changes originating in the Walls of the Ducts.*—(*a*) Catarrhal jaundice due to inflammatory swelling of the mucous membrane of the duct, at or extending some distance from the biliary papilla, and due to the spread of inflammation from the duodenum, is preceded by gastro-intestinal disturbance—diarrhœa and vomiting. It is of short duration, one to two weeks, and is not accompanied by the fever, enlarged spleen, and albuminuria seen in infectious jaundice. It should, however, be borne in mind that the jaundice of malignant disease and of acute yellow atrophy may begin exactly like benign catarrhal jaundice.

(*b*) The infective and suppurative forms of cholangitis are usually associated with gall-stones or the rupture of a hydatid cyst into the ducts; but the more severe form complicates a considerable proportion of the cases of that rare condition, primary carcinoma of the duodenal surface of the biliary papilla.

Possibly jaundice in the roseolous stage of syphilis is due to a specific cholangitis.

In the infective forms the symptoms are those of intermittent hepatic fever, while in suppurative cholangitis the clinical picture is that of suppuration in the liver, and closely resembles pylephlebitis. It may be incidentally pointed out that jaundice may be absent in the suppurative cholangitis.

(*c*) Growths of the mucous membrane of the ducts.

Carcinoma, which is sometimes met with, gives rise to deep progressive jaundice with enlargement of the gall-bladder. At first, since it is often accompanied by colic, it may resemble gall-stones, while later it resembles malignant disease of the head of the pancreas.

Innocent tumours, such as papillomata, are curiosities and cannot be accurately diagnosed.

Xanthelasma has been known to occur in the mucous membrane of the bile ducts, but if multiple its occurrence on the skin would probably, and in most instances rightly, be referred to jaundice due to some other cause.

(*d*) Simple stricture due to the cicatrisation of past ulceration in the ducts, such as might conceivably be set up by the passage of a gall-stone, is so extraordinarily rarely shown to exist that, clinically, cases of jaundice following hepatic colic should be regarded as due to fresh gall-stone impaction or to malignant disease.

In very rare instances cicatrisation of a duodenal ulcer involving the biliary papilla has been demonstrated, but duodenal ulcers are usually confined to the first part of the

duodenum, and are often latent. Jaundice following the symptoms of duodenal ulcer would therefore more reasonably be regarded as due to adhesions involving the common duct.

Congenital obliteration of the bile ducts, giving rise to persistent icterus in young children, has already been described (*vide* vol. iii. p. 372). The age of the patient and its persistent nature make its diagnosis easy.

(*e*) Spasm of the muscular fibres of the ducts explains emotional jaundice, such as that due to fright, satisfactorily; but inasmuch as it is difficult to prove, its occurrence is open to question.

(3) The conditions that may press on or invade the bile ducts from without, and so interfere with the flow of bile and give rise to jaundice, are very numerous. A tumour may merely press on the duct, or on the other hand it may actually invade the walls of the duct; a floating kidney may exert temporary traction or pressure on the ducts, or inflammatory adhesions spreading out from a gastric, duodenal ulcer, or other focus of inflammation, may involve the ducts and strangle them. The following list shows the great variety of conditions that may be met with:—

(*a*) Tumours projecting from the liver itself into the portal fissure may compress the bile ducts. Thus a carcinomatous mass may compress the ducts; exceptionally it occludes one hepatic duct and sets up jaundice, though bile from the other lobe enters the duodenum and colours the fæces. A hydatid cyst may, though it rarely does, press on the ducts. A gumma occasionally arises in this situation and sets up jaundice.

(*b*) Enlarged glands in the portal fissure may press on the ducts. The enlargement may be secondary to carcinoma of the stomach, colon, gall-bladder, liver itself, pancreas, kidney, etc. Enlargement may also be due to tubercle or possibly to syphilis.

(*c*) Malignant disease may spread by continuity up the lesser omentum from the pylorus and give rise to jaundice by invading the ducts. Jaundice is said to be met with in 5 per cent. of cases of gastric carcinoma, and may depend either on a direct extension of the growth or on a secondary growth in the portal fissure.

(*d*) As already mentioned, fibrosis spreading out from a gastric ulcer at the pylorus, or even from a duodenal ulcer, may involve and compress the ducts. It is rare, and when it does occur is likely to be regarded as malignant disease, since the matting together of the tissues may be palpable as a tumour. It is remarkable that jaundice is hardly ever seen in perihepatitis or in simple chronic peritonitis — conditions in which fibrosis and cicatricial contraction are very marked, and might be expected to compress the bile duct.

(*e*) Enlarged glands in the neighbourhood of the common bile duct near its entrance into the duodenum may be secondary to carcinoma in various organs. When the duct is thus obstructed the gall-bladder should be enlarged, and the condition closely resembles malignant disease of the head of the pancreas.

(*f*) *Tumours and Cysts of the Pancreas.*—Malignant disease of the head of the pancreas, which is practically always spheroidal-celled carcinoma of comparatively slow growth, forms a hard mass, rarely large enough to be felt, which compresses and may invade the common bile duct. The duct becomes dilated, and the gall-bladder often forms a tense, pear-like tumour. The jaundice is progressive and deep, and emaciation is rapid.

Hydatid cysts have been known to occur in the head of the pancreas, and to give rise to jaundice.

Pancreatic and peripancreatic cysts rarely come so far to the right as to compress the ducts, but exceptionally this does occur. Cysts of the pancreas are readily felt, and should be distinguished from a distended gall-bladder by the fact that the stomach, if inflated, is found to lie in front of the cyst.

(*g*) Retroperitoneal tumours arising from glands, connective tissues, and possibly from accessory adrenal bodies, may grow forward and involve the bile ducts. As in the case of pancreatic and renal tumours, coils of intestine may intervene between them and the abdominal wall, and give rise to a resonant note on percussion.

(*h*) *Renal Tumours.*—A primary tumour of the right kidney rarely presses on the bile duct, giving rise to jaundice; a secondary growth in the portal fissure is a more probable cause of jaundice in primary renal growths. The same is true of primary tumours in the suprarenal bodies.

Floating kidney on the right side may give rise to attacks of biliary colic and jaundice; the condition is therefore very likely to be regarded as cholelithiasis. The mechanism has been thought to be due to direct pressure of the kidney on the duct, but Macalister regards it as due to traction, the peritoneum covering the kidney being continuous with that around the bile duct.

(*i*) *Abdominal Aneurysms.*—An aneurysm of the aorta under the pancreas may compress the common bile duct and give rise to jaundice.

Aneurysms of the hepatic artery are rare, but when they occur are frequently accompanied by jaundice. The aneurysm may compress the common hepatic or hepatic ducts, in which case the gall-bladder will not be dilated as it is when an aortic aneurysm compresses the common duct.

Aneurysm of the commencement of the superior mesenteric artery has been known to give rise to jaundice.

Aneurysms can only be diagnosed as the cause of jaundice when they can be felt.

(*j*) *Glenard's Disease.* — In rare instances hepatoptosis may give rise to twisting of the bile duct and jaundice. Dilatation and ptosis of the stomach may also induce kinking of the bile duct, or by leading to kinking of the duodenum prevent the outflow of bile from the biliary papilla.

(*k*) *Uterine and Ovarian Tumours.* — Large fibro-myomata and cysts have been known to produce jaundice, but they must reach an enormous size before doing so, and their existence would be manifest long before.

Malignant tumours arising in the uterus or ovaries may, by means of secondary growths in or near the liver, give rise to biliary obstruction.

(*l*) Fæcal impaction in the colon has been said to give rise to jaundice, but the association is rare, and it is probable that when these two conditions are associated there is either some underlying cause, such as malignant disease, which accounts for them both, or the jaundice is due to catarrh of the ducts.

### Differential Diagnosis

The large number of conditions of which jaundice may be a symptom makes it essential that a careful examination should be made for evidence of disease elsewhere. Thus the existence of a tumour in the abdomen, breast, or rectum will suggest malignant disease, while the coexistence of syphilis, in either the secondary or tertiary stage, should be an indication for specific treatment.

The following points have a bearing on the nature of jaundice in a given patient:—

*Age.*—Transient slight jaundice noticed a few days after birth is physiological, but if the jaundice is well marked, and accompanied by fever, septic infection spreading from the umbilicus may have taken place. Persistent jaundice from birth is in favour of congenital obliteration of the ducts.

In early adult life catarrhal jaundice should be thought of. Between the ages of thirty and forty years gall-stones, especially in women, are the most probable cause, while later in life malignant disease and cirrhosis must also be taken into account.

*Sex.*—Women are more prone to gall-stones and to malignant disease; men to cirrhosis, and perhaps to the infectious forms of jaundice, such as Weil's disease, which have been noticed to attack soldiers and butchers.

Pregnant women seem more susceptible than others to acute yellow atrophy.

*Onset.*—If preceded by gastro-intestinal disturbance, catarrhal jaundice is indicated; if by severe colic, gall-stones. A gradual onset with no special accompaniments should suggest the pressure of a tumour, practically always of a malignant nature, on the ducts. Repeated and transient attacks are in favour of gall-stones.

*Occurrence of Pain.*—Constant pain is suggestive of malignant disease, intermittent attacks point to gall-stones. Attacks of biliary colic may also occur when hydatid membranes are passed through the ducts, and occasionally when malignant disease involves the ducts—pseudo-gall-stone colic.

Absence of pain is, however, no proof against malignant disease, though it is the rule in catarrhal jaundice, and common in cirrhosis.

*Duration and Progress.*—Jaundice of short duration is most commonly catarrhal, or due to the passage of gall-stones. If continued for more than six months it is highly unlikely that malignant disease is present, and biliary cirrhosis or impacted gall-stone should be thought of. Jaundice lasting for years is probably due to biliary cirrhosis.

Progressive and black jaundice suggests malignant disease, while jaundice that persists but varies from time to time is more compatible with a stone impacted in the common duct, or with biliary cirrhosis.

*Degree and Intensity of Jaundice.*—Slight icterus may be catarrhal or, if associated with fever and constitutional conditions, toxæmic. The commonest causes of deep jaundice are malignant disease, impaction of a gall-stone in the common duct, and biliary cirrhosis. Extremely deep jaundice almost necessitates malignant disease; that accompanying gall-stone impaction is deeper than that of cirrhosis, but never equals that seen in compression of the common bile duct by tumours, such as carcinoma of the head of the pancreas, or in malignant disease of the duct.

Outbreaks of jaundice in epidemics suggest some form of toxæmic jaundice.

Fever suggests toxæmic jaundice, or biliary cirrhosis when associated with considerable splenic enlargement; pyrexia, of course, occurs in calculous cholangitis, in hepatic suppuration such as abscess or pylephlebitis, and occasionally in malignant disease.

Considerable enlargement of the liver is met with in biliary cirrhosis, in abscess, and in malignant disease; in the latter it is progressive, and the surface will probably be nodular.

The association of ascites with jaundice points to malignant disease or to cirrhosis, but in the former jaundice is deeper. In fact, the darker the jaundice the more likely is the cause to be malignant growth.

The existence of disease elsewhere in the body has already been insisted on as of supreme importance in arriving at a diagnosis of the cause, and therefore of the prognosis and treatment of jaundice.

Prognosis.—The occurrence of jaundice in certain diseases is of importance as showing that hepatic complications have supervened;

thus when, as very rarely happens, jaundice is met with in the course of typhoid fever, cholangitis or cholelithiasis should be thought of. In puerperal eclampsia, jaundice is of extremely bad omen; death follows in a few hours or days. The onset of jaundice after phosphorus poisoning is a sign that the liver is affected, and must be regarded as of the most grave significance, since few cases recover when this stage is reached. In like manner the occurrence of jaundice in malaria is of very serious import, as pointing to a severe hæmic infection, such as occurs in hæmoglobinuria or "blackwater" fever.

The ultimate prognosis of any case of jaundice is, of course, dependent on the cause and not on the degree of the jaundice; thus the slight icteric tint seen in toxæmic jaundice due to pyæmia, and the black jaundice of complete obstruction due to malignant disease, do not differ very much with regard to their prospect. The prognosis of chronic jaundice is bad: it is worst in cases of complete obstruction by malignant disease, for in addition to the fatal nature of the growth, biliary toxæmia or cholæmia is likely to occur; it is rather better in hypertrophic biliary cirrhosis, for although complete recovery is very improbable life may yet be prolonged for a very considerable time. In chronic jaundice due to gall-stones the prognosis is fair, because operative measures can be undertaken with a fair prospect of success.

When chronic jaundice is accompanied by xanthelasma or by the bulbous or "Hippocratic fingers" it may be assumed that the cause of the jaundice is not malignant disease, inasmuch as this would have killed the patient before these changes could have had sufficient time to develop.

In chronic jaundice much depends on the functional activity of the kidneys being well maintained; if the amount of urine falls and waste products are less freely excreted, biliary toxæmia is likely to result. The presence of albuminuria points to the kidneys being damaged by the toxæmia accompanying the jaundice, and is therefore an index of a severe condition. The detection of leucin and tyrosin in the urine of a case of jaundice makes the prognosis very grave.

When jaundice is accompanied by hepatic insufficiency, so that poisons which should have been destroyed by the liver escape into the general circulation, and give rise to a general toxæmia as shown by nervous symptoms such as delirium, drowsiness, and coma, and by hæmorrhages, the prognosis is very grave indeed, since life cannot be long maintained after the development of cholæmia. The occurrence, therefore, of nervous symptoms in cases of jaundice should always arouse anxiety.

There are cases at first quite indistinguishable from simple catarrhal jaundice in which nervous symptoms somewhat rapidly develop, and the case then runs the course of acute yellow atrophy of the liver. Again, in other instances the jaundice associated with malignant disease of the liver may begin exactly like catarrhal jaundice. Caution is therefore necessary in forming a prognosis in the early stages of what appears to be simple catarrhal jaundice, especially in patients past middle life.

TREATMENT. — The radical and most satisfactory plan is, of course, the removal or cure of the cause of the jaundice; for this an accurate diagnosis of each case is essential. The results of operations for the removal of gall-stones show how successful this plan may be.

Even in cases of inoperative malignant disease, such as carcinoma of the head of the pancreas, surgical measures may still give some relief by removing the extreme jaundice. Cholecystenterostomy, or uniting the gall-bladder to the small intestine, has been performed, and by allowing the bile to enter the intestine has removed the jaundice, prevented the occurrence of biliary toxæmia or cholæmia, and greatly improved the patient's condition for a time. The operation must be performed early; if black jaundice has already developed, the patient is in a very unfavourable state for this procedure.

The Palliative Treatment of Jaundice.— Symptoms should be treated as they arise: constipation should be met by five grains of blue pill or calomel, followed by a saline; want of appetite by dilute acids and nux vomica; nausea and vomiting, if present, by bismuth, dilute hydrocyanic acid, and morphia. If there is much flatulence, creasote, salicylate of bismuth, liquor perchloridi hydrargyri, and salol may be employed as intestinal antiseptics.

Capsules of ox bile, or pills coated with keratin so as to prevent the bile being liberated in the stomach, may be given before food three times daily in order to replace the absent bile in the intestine.

For the pruritus, alkaline baths or sponging with carbolic lotion 1 in 40 should be tried. Pilocarpine $\frac{1}{6}$-$\frac{1}{4}$ of a grain injected hypodermically often gives good results. For itching of the skin and hæmorrhages the administration of chloride of calcium in 15 or 20 grain doses may be tried three times a day for two or three days, but not longer at a stretch, as its effect in promoting coagulation of the blood is lost after a comparatively short time.

Before an operation such as a cholecystotomy on a jaundiced patient, it is well to give chloride of calcium so as to diminish the risk of bleeding from the jaundiced tissues.

The patient should be encouraged to take plenty of water so as to avoid constipation, and in catarrh or cholangitis to wash out the ducts; in these cases salicylate of soda may also be

taken with advantage, inasmuch as it increases the secretion of bile and so tends to sluice out the bile ducts.

A milk diet should be adhered to since it is easily digested, tends to minimise intestinal fermentation and auto-intoxication, and acts on the kidneys as a diuretic. Fatty food, for which the patient often has a distaste, should be avoided.

### Jaundice of Phosphorus Poisoning


Morbid Anatomy . . . . . 60
Symptoms and Signs . . . . . 60
Diagnosis . . . . . 61
Prognosis . . . . . 61
Treatment . . . . . 61


Usually phosphorus poisoning is suicidal, and is effected by consuming rat pastes or making a meal of the heads of lucifer matches. It is rare in England—in the ten years ending 1892 there were 147 fatal cases in England and Wales (W. Blythe); but it is not infrequent abroad, especially in Vienna. It may be due to rat paste accidentally swallowed, or, as in Leonard Hill's case, be due to the application of phosphorescent paste to the skin.

It sometimes follows the medicinal use of the drug; the smallest fatal dose for an adult is $\frac{1}{8}$ of a grain; usually 1 to 2 grains, either taken in a single or in divided doses, is the quantity followed by a lethal result. In a baby the smallest fatal dose known is $\frac{1}{100}$ grain.

Results very similar to those of phosphorus poisoning may follow poisoning with iodoform, arsenic, antimony.

Morbid Anatomy.—The liver is, as a rule, much larger than natural, firm but friable, and of a pale yellow colour. In a few cases the liver has presented exactly the features of acute yellow atrophy; but this is exceptional, and the change in the liver is essentially one of increase in size due to acute fatty change, and resembles that of iodoform poisoning. It is said that as time goes on the liver diminishes in size.

Under the capsule and on section the yellow buff aspect of the liver substance shows here and there reddish spots due to hæmorrhage, which stand up against the bile-stained liver substance.

In dogs poisoned by phosphorus there is a large quantity of fat in the liver, and the nuclei of the liver-cells show fragmentation. There is, however, no fat or almost none in the myocardium, none in the voluntary muscles, and only a little in the kidneys (Ray, M'Dermott, and Lusk).

Microscopically the liver-cells show cloudy swelling and very advanced fatty change. The cells in parts of the lobules may contain granules of bile pigment. Leucin and tyrosin may be found in the liver. There is sometimes slight proliferation of the connective tissue elements of the portal spaces, and in cases that recover some cirrhosis may develop.

The heart and kidneys show advanced fatty change; the heart may be so soft as to be readily perforated by the fingers during its examination. The voluntary muscles also undergo fatty change.

The spleen may be much enlarged.

Hæmorrhages are found scattered throughout the body.

The *symptoms* due to the irritating effect of phosphorus on the gastric mucous membrane come on from a few minutes to three hours after taking the poison. With phosphorated oil or phosphorus in a soluble state the ill effects are soon manifested, while if the poison was taken in a solid form the onset is delayed. There is gastric pain and vomiting, which may be so constant as to lead to dangerous collapse. The vomited matters and eructations may be luminous in the dark. The vomit may become dark and grumous. There is usually intense thirst, tenderness over the stomach and liver, but no hepatic enlargement at this stage.

The patient may die from collapse; if he does not, and has been satisfactorily treated, permanent recovery may follow, but in a considerable number of cases there is a temporary improvement succeeded by the onset of grave symptoms due to the toxic effects of the absorbed poison on the liver and other internal organs.

These severe symptoms usually begin about four days after the poison was taken; they may come on sooner, or, on the other hand, be delayed for two, three, or even six weeks, as in S. West's case.

It begins with jaundice and recurrence of vomiting of dark, grumous matters; then follows great prostration, ending in coma and death, usually on the fifth or sixth day from the time the phosphorus was taken, and after a day or two of grave constitutional symptoms.

Hæmorrhages into the skin and from mucous membranes are a constant feature of the disease, but are not so marked as in deep jaundice due to other causes. A blood count shows an increase in the number of red blood corpuscles in the circulation.

Jaundice is by no means a constant symptom, and does not, when present, bear any relation to the severity of the changes taking place in the liver. From experiments on animals, and from some observations in man, there is reason to believe that, as in acute yellow atrophy, the jaundice is obstructive and due to inflammation and stagnation of viscid bile in the smaller ducts.

The temperature is usually below normal.

The liver is enlarged and tender, the spleen is also enlarged, and the abdomen may become distended.

The most marked difference between this stage of phosphorus poisoning and acute yellow

atrophy is in the size of the liver. But in exceptional cases of phosphorus poisoning the liver is not enlarged, and in the early stages of some examples of acute yellow atrophy it is enlarged, so that the clinical resemblance between the two affections may sometimes be very close.

The urine is somewhat diminished in quantity, but never suppressed; it is high-coloured and of rather high specific gravity. It may contain albumin, blood, and casts. There are generally bile pigments and bile acids, while sarcolactic acid is very frequently present. It was formerly said that leucin and tyrosin were not present in the urine, and stress was laid on this in the diagnosis from acute yellow atrophy; it is now known that leucin and tyrosin may be present in the urine in phosphorus poisoning, but are far from being constantly found. Leucin is less frequently found than tyrosin. The amount of urea was formerly thought to be greatly diminished; it now appears that though it is diminished in the early stages when all food is refused by the stomach, it is absolutely increased in the later stages as a result of increased metabolism. Experimentally it has been shown that the rise in the proteid metabolism in phosphorus poisoning is only equalled by that in phloridzin diabetes. It is said that alimentary glycosuria is more readily induced in phosphorus poisoning than in health, but it is very seldom found in practice; Hunter says that only three cases are on record.

Diagnosis.—The history that phosphorus has been swallowed, or that symptoms justifying this conclusion have recently occurred, is a most important if not essential point. The presence of phosphorus in the vomit, or the fact that the vomited matters are luminous in the dark, of course settles the question.

In the differential diagnosis from acute yellow atrophy the following points should be borne in mind:—(i.) The large size of the liver. (ii.) The amount of urea is not diminished except in the earliest stage. (iii.) The greater prominence of gastro-intestinal symptoms.

Prognosis.—In cases where jaundice and enlargement of the liver come on, the outlook is very grave; most cases die.

Treatment.—When the poison has been recently taken the stomach should be emptied and washed out.

As an antidote, French or old or oxidised oil of turpentine should be given every quarter of an hour for the first hour, 40 minims in an emulsion, and afterwards three or four times daily.

Permanganate of potash and sanitas have also been recommended. Mucilaginous drinks should be given; but oils or fats should be avoided, as they render the phosphorus more soluble. Purgatives should be given.

When the grave constitutional symptoms have developed, no special treatment can be relied on.

**Jaw-Clonus.**—A phenomenon (series of rhythmical contractions of the muscles of the jaw) occasionally seen, in cases of progressive muscular atrophy, on pulling down the lower jaw. *See* Jaws; Tendon-Jerks.

**Jaws.** *See* Mouth, Injuries and Diseases of the Jaw; *see also* Acromegaly (*Pathological Anatomy, Jaw*); Actinomycosis (*Primary Actinomycosis of Mouth and Pharynx*); Brain, Affections of Blood-Vessels (*Paralysis from Vascular Lesions, Reflexes, Jaw-Jerk*); Labour, Management of (*Podalic Presentations, Jaw Traction*); Labour, Accidental Complications (*Fœtal Injuries, Jaw*); Leontiasis Ossea (*Megalocephaly*); Meningitis, Posterior Basic (*Symptoms*); Nose, Accessory Sinuses, Inflammation of (*Maxillary Sinus*); Rheumatism, Rheumatoid Arthritis (*Clinical Features, Temporo-Maxillary Joint*); Teeth; Toxicology (*Phosphorus Poisoning, Necrosis of Jaw*); Trades, Dangerous (*Phosphorus and Lucifer Match-Making*.—When we consider the important rôle played by the upper and lower jaws in health, the importance of their rôle in disease is obvious. According to popular belief the set of the lower jaw, by which is meant the state of development and tenacity of its bony and muscular structures, is an important index of character—that is, an index of the state of mental development of the individual. In this connection it is interesting to refer to the views entertained by some alienists, that a similar imperfect state of development of the upper jaw, as revealed by a characteristic arch of the palate, is not infrequent in many cases of mental deficiency, more especially in those which first manifest symptoms about the end of the first decade. This view is by no means fully confirmed by the experience of dental practitioners, and the subject will be again referred to. (See "Teeth.")

The diseases of the lower jaw are almost entirely surgical, including various forms of inflammation, tumour growths, and cystic formations, involving the mucous membrane of the gums, the periosteum, or the bone. The main points of more medical interest are those relating to affections of the temporo-maxillary joint (see "Rheumatism" and "Rheumatoid Arthritis"), the different forms of neuralgia, and the later stages of paralysis when the "jaw-jerk" is a marked clinical symptom. (See "Neuralgia" and "Paralysis.")

Diseases of the upper jaw have special significance because of the various irregular cavities that lie amongst the bones of the face and the intercommunications between these different cavities.

When we bear in mind that the upper jaw is the principal bone of the face, entering into the

formation of the floor of the orbit, the floor and lateral wall of the nasal cavity and the hard palate, in addition to constituting the site for the attachment of the upper teeth, with their important functions and delicate nervous supply, we need not wonder that the exact diagnosis of the position and nature of the different diseases encountered is sometimes very difficult to determine. This is specially true of the various suppurative diseases of the antrum of Highmore and the various accessory cavities communicating with it. This will be discussed in the section on "Nose," where the differential diagnosis of disease in the different accessory cavities will be fully considered. The other general surgical articles will be mainly considered in the article on "Mouth," to which the reader is referred.

**Jecorin.**—A substance obtained from the horse's liver (Lat. *jecur*, the liver), containing sulphur and phosphorus as well as carbon, oxygen, hydrogen, and nitrogen; it is found also in blood serum. *See* PHYSIOLOGY, BLOOD (*Plasma and Serum*).

**Jecur Uterinum.**—The placenta.

**Jejuno-.**—In compound words jejuno- (Lat. *jejunus*, fasting) means referring to the portion of the small intestine named the jejunum; thus *jejuno-colostomy* means the making of a communication between the jejunum and the colon.

**Jejunostomy.**—The operation by which an artificial opening is made into the jejunum, with the object of setting the stomach at rest, in cases of inoperable cancer; but it is of doubtful utility. *See* STOMACH, SURGICAL (*Cancer, Palliative Operations*).

**Jellies.** *See* DIET (*Animal Food, Jellies*); INVALID FEEDING (*Prepared and Predigested Food, Jellies*).

**Jelly-Fish.** *See* DERMATITIS TRAUMATICA ET VENENATA (*Causal Agents*).

**Jelly of Wharton.**—The mucoid tissue or gelatine of the umbilical cord. *See* FŒTUS AND OVUM, DEVELOPMENT OF (*Umbilical Cord*); PHYSIOLOGY, TISSUES (*Connective, Mucoid*); PREGNANCY, DISEASES OF PLACENTA AND CORD (*False Knots*).

**Jenner, Edward.** *See* MEDICINE, HISTORY OF (*Vaccination*).

**Jensen's Pump.**—A pneumatic force-pump used for detecting faults in drains; this pneumatic test is preferable to the hydraulic one, for it can be applied in all weathers, the pressure at all points is uniform, and it can be used for vertical pipes.

**Jensen's Tumour.**—A malignant growth found in the mamma of the mouse, and capable of transplantation beneath the skin of another mouse and of growth and the formation of metastatic tumours there; it has been doubted whether Jensen's tumour is a carcinoma, but the weight of opinion is in favour of the view that it is a malignant growth and a true carcinoma.

**Jequiritin.**—The active principle of jequirity seeds (*Abrus precatorius*), regarded as a mixture of legumin and jequirity-zymase; poisonous if injected into the blood.

**Jequiritol.**—A preparation obtained from the seeds of *Abrus precatorius* by Merck; it is used in affections of the eye, for clearing corneal opacities, but is contraindicated if disease of the lachrymal ducts be present.

**Jequirity Seeds.** *See* ABRUS; JEQUIRITIN; JEQUIRITOL.

**Jerk.** *See* TENDON-JERKS.

**Jeyes' Fluid.**—A disinfecting fluid, containing cresol or methyl phenol. *See* DISINFECTION (*Chemical Disinfectants, Phenol*).

**Jigger.**—The pulex penetrans, chigoe, or sand-flea, found in the West Indies, China, South America and elsewhere. The adult female penetrates the skin, especially of the feet, produces her eggs, and so leads to inflammation and even ulceration; its attacks may be prevented by the use of the essential oils locally.

**Jiu-Jitsu.**—The Japanese art of self-defence on scientific principles, enabling a comparatively feeble person with knowledge of the art to overcome a much heavier and stronger individual who is ignorant of it; it means literally "the gentle art"; it teaches how to fall without injuring one's self, how to parry attacks, and how to utilise a knowledge of the anatomy of the body to obtain an advantage over an assailant.

**Johannisbad.** *See* BALNEOLOGY (*Austria*).

**Johimbine.**—An alkaloid from the Johimbehe, an African tree; it is said to have aphrodisiac qualities.

**Joints, Diseases of.**

DEFINITION OF TERMS APPLIED TO MORBID CONDITIONS OF JOINTS—

*Synovitis* . . . . . 63
*Hydrops* . . . . . 63
*Arthritis* . . . . . 63
*Empyema* . . . . . 63
*Ulceration of Cartilage* . . . 63
*Articular Caries* . . . . 63
*Disorganisation* . . . . 64
*Rigidity* . . . . . 64
*Contracture* . . . . . 64
*Ankylosis* . . . . . 64

CLASSIFICATION OF DISEASES OF JOINTS—
I. ERRORS OF DEVELOPMENT . . 65
II. BACTERIAL DISEASES—
*Pyogenic* . . . . 65
*Tuberculous* . . . . 69
*Syphilitic* . . . . 79
*Acute Rheumatism* (*q.v.*)
III. DISEASES ASSOCIATED WITH CERTAIN CONSTITUTIONAL CONDITIONS—
*Gout* . . . . . 80
*Chronic Articular Rheumatism* 80
*Arthritis Deformans* . . 81
*Hæmophylia* . . . . 82
IV. DISEASES ASSOCIATED WITH NERVE LESIONS—
*Neuro-Arthropathies* . . 82
V. HYSTERICAL OR MIMETIC JOINT AFFECTIONS . . . . . 84
VI. TUMOURS AND CYSTS . . . 84
VII. LOOSE BODIES IN JOINTS . . 84
VIII. DISEASES OF SPECIAL JOINTS (*q.v.*)

*See also* AMPUTATIONS; ANKLE-JOINT, REGION OF, INJURIES AND DISEASES AND OPERATIONS; ASPIRATOR, USES OF (*Effusion in Joints*); BALNEOLOGY (*Balneo-Therapeutics in Gout, Rheumatism*, etc.); BONE, DISEASES OF (*Deformities from Osteomyelitis*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Trophic Changes in Joints*); ELBOW-JOINT, REGION OF, INJURIES AND DISEASES; FINGERS; GOUT (*Morbid Anatomy*); HÆMOPHILIA (*Symptoms, Joint Affections*); HIP-JOINT, INJURIES AND DISEASES OF; HYSTERIA IN CHILDHOOD (*Symptoms*); KNEE-JOINT, DISEASES AND INJURIES; LABOUR, INJURIES TO THE GENERATIVE ORGANS, etc. (*Pelvic Articulations*); LARYNX, AFFECTIONS OF THE CARTILAGES (*Anchylosis of Crico-Arytenoid Joint*); LEUCOCYTHÆMIA (*Symptoms, Hæmorrhage into Joints*); MALINGERING (*Joints*); MENINGITIS, TUBERCULOUS AND POSTERIOR BASIC (*Complications, Periarthritis*); MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*Complications, Arthritis*); MOUTH, INJURIES AND DISEASES OF THE JAW (*Temporo-Maxillary Joint*); MUMPS; OSTEO-ARTHROPATHIES; PREGNANCY, PHYSIOLOGY (*Changes in Pelvic Joints*); PURPURA (*Symptoms*); SCURVY, INFANTILE (*Clinical Features*); SHOULDER, DISEASES AND INJURIES OF; STERNO-CLAVICULAR JOINT; SYPHILIS; SYRINGOMYELIA; TABES DORSALIS (*Tabo-Arthropathies*); TUBERCULOSIS (*Joints*); URETHRA, DISEASES OF (*Gonorrhœa, Complications*); WRIST-JOINT, INJURIES.

DEFINITION OF TERMS APPLIED TO MORBID CONDITIONS OF JOINTS.—*Synovitis*, while implying inflammation of the synovial membrane, presents different features according to its etiology. It presents many analogies with peritonitis. The effusion into the joint, which is a frequent accompaniment of synovial inflammation, may be serous, sero-fibrinous, or purulent. It is much to be desired that one should avoid the use of the term synovitis without some qualifying adjective, which will indicate its pathological nature, *e.g.* rheumatic, gouty, gonorrhœal, pyogenic, or tuberculous.

*Hydrops, hydrarthrosis*, or *chronic serous synovitis*, are terms employed when the effusion of fluid into the joint is the most prominent clinical feature. It presents analogies with ascites or hydrocele of the tunica vaginalis, and is to be regarded rather as a symptom than as a separate entity. It may occur apart from disease, *e.g.* in the knee-joint, from repeated and neglected sprains (football knee); it is chiefly met with in the chronic and intermittent forms of synovitis resulting from chronic staphylococcus osteomyelitis of one of the adjacent bones, from gonorrhœa, tuberculosis, syphilis, arthritis deformans, arthropathies of nerve origin, and when there are loose bodies in the joint.

*Arthritis* is the term applied, when not only the synovial membrane, but all the joint structures are involved in the disease, viz., the ligaments, articular surfaces, and it may be also the ends of the bones. While it may be anatomically possible to differentiate between synovitis and arthritis, it is often impossible to do so clinically, so that in practice the two terms are often used indiscriminately. One may confidently speak of the existence of arthritis whenever there are marked symptoms of involvement of the articular surfaces.

There are as many different pathological forms of arthritis as of synovitis, so that it is desirable in using the term to add a qualifying adjective which will indicate its nature, *e.g.* rheumatic, gouty, pyogenic, or tuberculous. The arthritis, according to its etiology, may assume a dry form, or it may be attended with effusion into the joint; this may be serous, as in arthritis deformans, or may be sero-fibrinous or purulent, as in certain forms of pyogenic and tuberculous arthritis. Wasting of the muscles in the vicinity of the joint is a constant accompaniment of arthritis; it especially affects the extensor muscles, and is quantitative rather than degenerative; the muscles affected do not show the reaction of degeneration. From the involvement of the articular surfaces it is unusual to have complete recovery from arthritis; it is apt to result in one or other form of ankylosis.

*Empyema* is the term occasionally employed to indicate that the cavity of the joint is full of pus; it is chiefly observed in chronic suppurative disease of pyogenic or tuberculous origin, and is usually attended with the formation of abscesses outside the joint.

"*Ulceration of cartilage*" and "*caries of the articular surfaces*" are common accompaniments of the more serious and progressive forms of joint disease, and especially those of bacterial origin. They represent successive stages in the same destructive process, the disappearance of

the cartilage being frequently followed by exposure and disintegration of the subjacent bone. The changes which precede and follow upon the ulceration of the cartilage vary with the joint disease of which it is an accompaniment; their consideration is beyond the scope of the present article. The occurrence of ulceration of cartilage and of articular caries is always attended with characteristic clinical features, viz., the joint is held rigid by the involuntary contraction of muscles, the wasting of muscles is more pronounced, and there are "starting pains" at night. Advanced articular caries is usually associated with some deformed attitude, with shortening, and sometimes with dislocation. It may be possible under anæsthesia to make the exposed and crumbling bony surfaces grate upon one another. Should recovery take place, repair will usually be attended with fusion of the opposing articular surfaces by fibrous tissue or by bone.

*Disorganisation of a joint* is a convenient description of the condition in which all the constituent parts are damaged or destroyed. It results from the more severe and destructive forms of joint disease, and especially those of pyogenic or tuberculous origin.

Conditions of Impaired Mobility of Joints. —(1) *Rigidity* implies the fixation of a joint by the involuntary contraction of muscles; it is sometimes called false ankylosis, because it entirely disappears under anæsthesia. (2) *Contracture* is the term applied when the fixation of the joint is due to permanent pathological changes in the soft parts surrounding it, chiefly consisting in the shortening of muscles, tendons, tendon sheaths, ligaments, fasciæ, and skin; the parts on the flexor aspect are more liable to shortening, hence contracture is nearly always associated with flexion. Contracture results from a number of conditions, apart from disease of the joint concerned, *e.g.* disease in one or other of the adjacent bones, lesions of the motor nervous mechanism, hysteria, etc. (3) *Ankylosis* is the term applied to the stiffness or immobility of a joint when it results from changes involving the articular and other surfaces which normally move or glide upon one another. It is frequently combined with contracture and with thickening and induration of the capsular and other ligaments. Three anatomical varieties of ankylosis are distinguished: (*a*) the *fibrous*, in which there are fibrous adhesions between the opposing surfaces; these adhesions may be loose or tight, may be localised in the form of bands, or diffuse, altogether obliterating the cavity of the joint; the stiffness may vary, therefore, from restriction of the normal range of movement up to close fibrous union of the bones which may prevent any movement whatsoever. Fibrous ankylosis may result from injury, especially dislocations and fractures implicating a joint, or from disease, *e.g.* pyogenic, gonorrhœal, tuberculous, rheumatic, gouty, or other form of arthritis. (*b*) *Cartilaginous ankylosis* implies the fusion of two opposed cartilaginous surfaces; it is best seen between the patella and trochlear surface of the femur, and between the femoral condyles and articular facets of the tibia in certain forms of tuberculous disease of the knee. Clinically it is associated with absolute rigidity of the joint. (*c*) *Bony ankylosis* (synostosis) implies an osseous union between articulating surfaces; it may be a sequel of the preceding forms, or it may result from a more direct fusion of two opposing surfaces subsequent to their having been bared of their cartilage. In the majority of cases it is to be regarded as a reparative process, and as presenting analogies with the union of fractures. It may be a sequel of almost any one of the diseases known to affect joints. Its occurrence is not necessarily dependent upon antecedent suppuration in the joint, as was formerly believed; it has been observed to follow the pyogenic, gonorrhœal, tuberculous, syphilitic, gouty, and neuropathic affections of joints. It is doubtful if it occurs in the spinal arthropathies apart from superadded infection; in arthritis deformans it is also questionable if the articular surfaces ever become united by bone, although it is common to have complete fixation of the vertebral and other joints by the ossification of ligaments and other extra-articular structures (external or peripheral ankylosis). While in most cases the occurrence of true bony ankylosis is readily explained by changes resulting from antecedent disease, the pathology of certain rarer forms is quite unknown. Ankylosis may certainly occur apart from any recognised reparative process, and may coexist with other trophic changes in the skeleton of unknown origin. The name *arthritis ossificans* has been applied by Griffiths to a certain group of these exceptional cases. The origin of ankylosis from simple disuse of a joint has not been corroborated.

It is important to bear in mind that in any example of bony ankylosis there are associated changes in the soft parts which, if the limit be fixed in a vicious attitude, will render futile any operative interference solely directed to the bones concerned.

Ankylosis of a joint before the skeleton has attained maturity, has very little influence on the growth in length of the bones affected; any arrest of growth is more likely to depend on changes in the epiphysial junctions resulting from the original disease.

In the *diagnosis* between false and true ankylosis it may be necessary to anæsthetise the patient. The nature and extent of true ankylosis may be learned from manipulations of the limb or by skiagraphy. In fibrous ankylosis mobility may be elicited, although only to a very slight degree; in osseous

ankylosis the joint is rigidly and immovably fixed; in the fibrous variety any attempt to forcibly move the joint causes severe pain, while in the osseous variety such attempts are painless.

*The treatment* is influenced by the nature of the original disease, the variety and attitude of the ankylosis, and the normal functions of the joint concerned. If the aim be a movable joint in a case of fibrous ankylosis, treatment is directed towards elongating or rupturing the fibrous union between the bones. The gradual stretching of adhesions, by exercises, manipulations, douching, extension, and special forms of apparatus, has much to recommend it, given the required perseverance and fortitude on the part of the patient, and the encouragement afforded by indications of yielding on the part of the adhesions. The forcible rupture of adhesions under an anæsthetic (nitrous oxide) may be necessary, especially when there are one or more strong fibrous adhesions or bands; these give way with an audible crack; the procedure must be carried out with caution in view of such risks as fracture of the bone (which is often rarefied), separation of epiphysis, fat embolism, and restarting of the original disease; in any case it is followed by considerable pain and effusion into the joint, which necessitate rest for some days before exercises, massage, and other manipulations are resumed.

In selected cases of fibrous ankylosis, with or without contracture, it may be advisable to attempt to secure a movable joint by open arthrotomy, dividing or removing adhesions and other contracted tissues; this procedure, which has been specially named *arthrolysis*, has been chiefly practised in the elbow, and has yielded results which are distinctly encouraging.

*If the ankylosis is osseous and a movable joint is desired, e.g.* at the elbow, a sufficient amount of bone, and it may be also of periosteum, must be resected to allow of the formation of a false joint.

On the other hand, *if it be desired that the joint disease should result in rigid ankylosis, e.g.* in certain cases of tuberculous disease of the knee, treatment may be directed towards favouring its occurrence, and in such an attitude as will secure the maximum usefulness of the limb concerned. To this end prolonged immobility in plaster-of-Paris or other apparatus is employed. This will not suffice in other forms of joint disease, *e.g.* arthritis deformans, spinal arthropathies; in these the articular surfaces must be removed with the saw in order to bring about osseous ankylosis.

*When bony ankylosis has occurred in an undesirable attitude, e.g.* flexion at the hip or knee, it can only be remedied by an osteotomy or wedge resection of the bone, with or without such additional division of the contracted soft parts as will permit of the limb being placed in the attitude desired. The fixation of the bones to each other by means of pegs may hasten the occurrence of osseous union, and afford an additional security of the correct attitude being maintained after operation.

I. Errors of Development.—These include congenital dislocations and other deformities of intra-uterine origin, *e.g.* abnormal laxity of joints, absence, displacement, and defective growth of one or other of the essential constituents of a joint, etc. They are chiefly described under "Deformities," vol. ii.

II. Bacterial Diseases. — In those which arise apart from wounds the bacteria concerned are carried directly to the joint in the blood stream, or they are lodged in the first instance in one of the structures (one of the bones) adjacent to the joint. In the former, *i.e.* the direct infections, the tendency is for all the structures of the joint to be involved simultaneously and diffusely, whereas in the indirect infections the disease is often localised to the area first infected, and only becomes generalised at a later period.

Bacterial affections resulting from infection of a wound implicating the joint are described under Injuries of Joints.

*Pyogenic Diseases.*—1. Those due to common pus organisms (staphylococci and streptococci).

2. Those related to acute articular rheumatism, pneumonia, typhoid, smallpox, scarlet fever, measles, diphtheria, erysipelas, dysentery, etc.

3. Those associated with gonorrhœal urethritis and gonorrhœal ophthalmia.

*The commoner pyogenic diseases* are the result of infection of one or other of the joint structures with staphylococci or streptococci, which may be demonstrated in the exudation into the joint, and especially in the substance of the synovial membrane. The method of infection is the same as has already been described in diseases of bone (see vol. i.). The organisms concerned having effected an entrance into the body, are carried to the joints by the arteries. Their localisation in particular joints is determined by injury, exposure to cold, antecedent disease of the joint, and other factors whose nature is not always apparent. A distinction may be made between *primary infections of joints*, in which the organisms involve articular structures from the outset, and *secondary infections* in which the initial lodgment and disease is in one of the bones belonging to the joint concerned. The former are more often met with in adults, and are illustrated by the joint suppurations in pyæmia and allied conditions. The latter are more frequent in children, and are illustrated by the well-known "acute arthritis of infants," in which the joint lesion owes its origin to an osteomyelitis in one of the bones adjacent to the joint. The clinical diagnosis between primary and secondary joint suppurations is rarely

possible, because their features are so very similar, and in the secondary infections it is usual for the joint disease to so overshadow the bone lesion from which it originates that the latter element may be only recognised on operating, or on post-mortem examination.

*The clinical features* vary with the gravity of the infection. They may assume the form of an *acute serous synovitis* which may recover, or become *chronic*, or may relapse after apparent cure. The *relapsing or intermittent synovitis* or hydrops, which closely resembles that of gonorrhœal or tuberculous origin, has been shown to depend in certain cases on staphylococcal disease of one of the adjacent bones, so that treatment of the latter is essential for permanent recovery.

In a certain number of cases the clinical features of pyogenic infection are remarkably *latent*, especially when it occurs in the course of some general illness, such as scarlet or other fever. It has been known to escape notice until the occurrence of some striking development, such as dislocation in the case of the hip-joint, or the occurrence at a later period of ankylosis.

*In the graver infections* the suppurative element is more prominent; the effusion into the joint is purulent; there is general illness, often ushered in with a rigor. The local signs and symptoms are those of an *acute arthritis*, in which all the joint structures participate, and which, if left to itself, may result in disorganisation. The synovial membrane is converted into granulation tissue; the ligaments and inter-articular cartilages share a similar fate. The articular cartilages, which are at first dulled and macerated, undergo fibrillation and necrosis, and separate in visible fragments. The subjacent bone which is thus exposed becomes the seat of inflammation and granulation, so that it disintegrates, the so-called articular caries. These changes in the articular surfaces add materially to the gravity of the lesion and to the suffering of the patient. The joint is held rigid by the involuntary contractions of muscles. The least attempt at movement causes severe pain. The slightest jar, even the shaking of the bed, may cause agony. Sleep is impossible, or is disturbed with "starting pains." The distension of the joint and fluctuation may be evident, or may be obscured by œdema of the overlying soft parts. Sometimes the entire limb is swollen and œdematous. In untreated cases the joint is usually allowed to become flexed. At the knee the angle of flexion may be so acute that the heel touches the buttock. The pus in the joint may perforate the capsule and spread in the surrounding tissues up and down the limb; sooner or later it ruptures on the surface and discharges externally through one or more sinuses. The final disorganisation of the joint with destruction of the ligaments, may be indicated by abnormal mobility, by grating of the articular surfaces, or by dislocation. In the acute arthritis of infants the epiphysis may be separated and displaced. The progress of the local disease is associated with aggravation of the general symptoms, and the patient is exhausted with suffering and poisoned with toxines.

In the course of *pyæmia* joints may become distended with pus without any pronounced changes in the joint structures, without local signs except those indicating the presence of fluid, and without much complaint on the part of the patient.

When the joint is the seat of a *direct infection through an external wound*, either accidental or operative, the condition is commonly spoken of as a *septic arthritis*. Its morbid anatomy and clinical features are similar to those described when the infection has been carried to the joint by the blood stream, but the lesion is usually more severe and destructive, and is more likely to persist and to result in osseous ankylosis.

*The terminations* vary with the gravity of the infection and with the stage at which it comes under surgical treatment. In the milder forms recovery is the rule, with more or less complete restoration of function. In the more severe forms, and especially when several joints are involved, death may result at an early stage from general pyogenic infection or toxæmia, or at a later period with symptoms of hectic fever, waxy degeneration, and exhaustion. If the patient recovers, the joint or the entire limb may be permanently damaged. There may be fibrous or bony ankylosis, and this may be in a good or in a bad position. There may be deformity from displacement or dislocation. From changes in the periarticular structures there may be contraction of the limb in the flexed or other undesirable position, and in the case of young subjects there may be interference with the future growth of the limb. The persistence of sinuses is usually associated with disease in one or other of the bones belonging to the joint.

*The diagnosis*, while easy and straightforward in the graver suppurative forms, may be difficult in the milder varieties, for these may resemble very closely the serous effusions in syphilis, gonorrhœa, and tuberculosis, or that caused by injury where there is no question of infection.

*The treatment* is governed by the same principles as guide us in the treatment of other pyogenic infections. The limb is immobilised and elevated. The altitude preferred will be that in which, should stiffness occur, there will be least interference with function. Extension by means of the weight and pulley may relieve symptoms and counteract any tendency to flexion.

While the application of ice or leeches to the joint is recommended by some authorities, others prefer an antiseptic compress of 2½ per cent carbolic lotion or 1 per cent formalin. A more important question is that of evacuating the fluid in the joint. If the latter is in sufficient quantity to cause tension, or if it tend to persist, or if from the temperature and other indications there is reason to suspect that it is purulent, it should be evacuated without delay. A trocar and cannula may suffice in the serous variety; the suppurative forms demand incision and drainage. In addition, the joint may be gently washed out with salt solution, with or without a preliminary washing with an antiseptic (1 in 2000 corrosive sublimate). It is a common experience that many forms of acute suppuration in joints (*e.g.* the acute arthritis of infants, the suppurations in pyæmia) yield at once to incision and drainage, if carried out sufficiently early and before any destructive changes have taken place.

On the other hand the results of simple drainage may be unsatisfactory. The temperature and other indications of progressive mischief may call for further interference. Continuous irrigation, with multiple openings for drainage, may be given a trial, or the joint must be laid freely open so that every pocket and recess will be exposed to view. In certain joints this is only attainable by resecting one or other of the bones belonging to it.

Amputation is to be had recourse to, if life is threatened by general infection, or if the limb is likely to be useless.

It goes without saying that the occurrence of suppuration in the periarticular soft parts, or in one of the adjacent bones, must be looked for and promptly dealt with.

When convalescence is established attention is directed to the restoration of the functions of the limb, to the prevention of stiffness and deformity by movements, massage, hot air and other baths (see p. 65).

At a later stage, and especially in neglected cases, operative and other measures may be required for deformity or stiffness.

For details of treatment see the individual joints.

In *typhoid fever* joint lesions may result from infection with the typhoid bacillus, or with ordinary pyogenic organisms. They have been observed, especially in the hip-joint, in the shape of an arthritis, with or without suppuration. They are sometimes remarkably latent, and may result in spontaneous dislocation (on slight movement, or on lifting the patient), or in ankylosis. They are very amenable to treatment.

In *acute pneumonia* different forms of arthritis occur, due to the pneumococcus. They are sometimes serous, sometimes of an acute suppurative character. The prognosis is described as unfavourable, because of the frequent occurrence of similar lesions in other serous membranes, viz., pleurisy, pericarditis, meningitis.

In *smallpox* it is not known whether the joint lesions are due to the specific virus of that disease or to ordinary pyogenic organisms. They may be serous in character like those of acute rheumatism, and may pass from one joint to another. The purulent forms are met with in relation to the suppurative stage of the skin eruption.

In *scarlet fever* joint lesions are comparatively common. They were formerly described as *scarlatinal rheumatism*. Our knowledge of their bacteriological nature is very imperfect. The most frequent clinical type is that of a serous synovitis, occurring within a week or ten days from the onset of the fever, more common in persons over fifteen years and in females. Its favourite seat is in the hand and wrist, involving the sheaths of the extensor tendons as well as the joints. It does not tend to migrate to other joints, and it rarely lasts longer than a few days; it is probably due to the specific virus of scarlatina. Joint lesions more closely resembling those from ordinary pyogenic infection are much less frequent than the preceding type; they occur more often in children, at a later stage of the fever, and in cases in which the throat lesion is severe. The arthritis may be acute and suppurative, may affect several joints, and may exhibit a grave septicæmic or pyæmic character. Authorities also describe a "true rheumatic arthritis" occurring when convalescence from scarlet fever is well advanced, favourably influenced by anti-rheumatic remedies, and sometimes complicated with endocarditis and with chorea.

In *measles* joint lesions are much rarer, and are said to be less serious than in scarlet fever.

In *diphtheria* they are also very rare. A hydrops of the knee has been recorded during the second week of the disease in which Loeffler's bacillus was present. Probably the majority of the joint complications in diphtheria are related to streptococci, which enter the body by way of the throat lesion.

In *erysipelas* effusion into joints is very exceptional. It may be purulent. Streptococci have been found in the fluid. Sometimes the joint is infected when erysipelas passes over it.

In *dysentery* the joints are occasionally affected. Two varieties of lesion are described—a dry form in which there is polyarticular pain affecting different joints with great rapidity and unaccompanied by effusion; the second, which is especially met with in the knee, is attended with an abundant exudation, strongly fibrinous in character, but in which no organisms, amœbic or otherwise, have been found. From the tendency of the fluid to persist it is usually necessary to evacuate it through a cannula.

The joint lesions which accompany *acute rheumatism* may be provisionally included with

the other members of this group, although their infective nature has not been established. For a description of them the reader is referred to the article on acute rheumatism.

Joint affections associated with puerperal fever, otitis media, etc., are not in any sense specific, for they are the result of infection with the common pyogenic organisms.

Gonorrhœal Affections of Joints.—These include all forms of joint lesions associated with gonorrhœal urethritis or gonorrhœal ophthalmia. They may develop at any time during a gonorrhœa, but are usually met with when the infection has reached the deeper urethra. They have been observed after the discharge has ceased. There is no connection between the severity of the gonorrhœa and the liability to joint disease. The gouty and rheumatic are supposed to be more liable. The sexes are affected with equal frequency.

As a complication of ophthalmia the joint lesion occurs more commonly towards the end of the second or during the third week.

The joint lesions may be the only evidence of metastatic infection, or they may be part of a gonorrhœal pyæmia, involving the endocardium, pleura, tendon sheaths, etc. The gonococcus is nearly always present in pure culture; it is found with most certainty in the synovial membrane, in which it is first deposited from the blood; it may be possible to find it in the exudation in the joint unless at the first onset of the disease. In the purulent forms of joint lesion the gonococcus may alone be present, or it may be associated with staphylococci or streptococci, the latter being derived either from the urethra or from elsewhere (throat, intestines, etc.).

The order of frequency in which the joints are affected is as follows:—knee, elbow, ankle, hip, foot, wrist, shoulder, fingers.

The joint affection is more often mono-articular than polyarticular.

The following clinical types may be differentiated; they may, however, merge into one another.

1. *A dry polyarthritis*, like chronic rheumatism, sometimes trifling and evanescent, and it may be recurring with each attack of gonorrhœa, or persistent and progressive, resulting in partial or complete stiffening of the joints affected, and permanent crippling of the patient.

2. *A form of chronic synovitis or hydrops*, in which the joint, nearly always the knee, on one or both sides, quietly fills with fluid of a serous or sero-fibrinous character; it closely resembles the hydrops from other causes; it is indolent, may readily subside under rest and then relapse, or may be very persistent and disabling. When recovered from, the joint may be expected to return to its normal condition.

3. *A more acute general inflammation of the joint* (arthritis) may begin as such, or follow on a milder form of the disease; there is sudden onset of severe pain, swelling, inability to use the limb, and considerable fever; the swelling may extend well beyond the limits of the joint, and may be associated with œdema of the soft parts, the skin may be red and hot as in erysipelas; the adjacent tendon sheaths and bursæ, especially at the ankle, wrist, and knee, may be simultaneously involved. While resolution is possible the tendency towards stiffness and ankylosis is considerable. The ankylosis, at first fibrous from close adhesions between the surfaces, may become bony, and may be associated with flexion or other deformity.

This type of gonorrhœal joint disease may be mistaken for acute rheumatism. The points in diagnosis are: its sudden onset without apparent cause, there is less tendency to wander from joint to joint once it has settled down, it is little influenced by salicylates, and it is frequently, if not always, mono-articular. In the author's experience it is more often met with in the elbow.

4. *A suppurative form*, or empyema, like that from ordinary pus microbes; it is usually single, but may be multiple; it is fortunately rare, because it is very serious, endangering the joint, or the limb, or life itself. It may be the result of gonococcal infection alone, or of a mixed infection. Abscesses may form outside the joint. Recovery is attended with ankylosis.

*The diagnosis* of gonorrhœal affections of joints is often missed, because gonorrhœa is not suspected by the practitioner; the denial of the patient is not to be accepted, especially in the case of women; sometimes the patient is really ignorant. The points in diagnosis from acute rheumatism have been already indicated.

*The prognosis* should always be guarded, because the disease may relapse, or may prove tenacious and persistent; the patient may be laid up for weeks or months, and may be finally crippled in one or in several joints.

*The treatment* (besides that of the urethral disease) consists in complete rest until all symptoms have disappeared. Salicylates may relieve suffering, but are not curative.

Iodide of potash may also relieve symptoms. Locally, the joint is immobilised by means of a splint, or by cotton wool and an elastic bandage; extension is employed in the case of the hip.

Great relief may be obtained from very hot baths, to which turpentine and black soap may be added. König recommends the use of tincture of iodine applied several times a day. In the persistent dry forms the hot-air bath or Bier's method by venous congestion may be employed. In hydrops, when the fluid persists, tapping should not be too long delayed; the joint may be washed out with a 1 per cent

solution of protargol. The purulent form is to be treated on general principles.

After all symptoms have settled down, but not till then, for fear of exciting relapse or metastasis, the joint may be massaged and exercised; stiffness from adhesions is most intractable, and may, in spite of every attention, terminate in ankylosis; the latter is to be dealt with on general principles.

## Tuberculous Diseases of Joints


General Facts . . . . . 69
Pathological Anatomy—
*Relative Frequency of Synovial and Osseous Disease* . . . . . 69
*Joint Lesions from Disease in adjacent Bone* . . . . . 69
*Tuberculous Lesions of Synovial Membrane* . . . . . 70
*Changes in Articular Cartilages* . 70
*Caries of Articular Surfaces* . . 70
*Caries Sicca* . . . . . 70
*Pathological Dislocation* . . . 70
*Contents of Tuberculous Joints* . . 71
*Periarticular Abscesses and Sinuses* . 71
*Reactive Changes in Vicinity of Tuberculous Joints* . . . 71
*Terminations and Sequelæ* . . . 71
*Relative Frequency in different Joints* 71
Clinical Features . . . . . 72
*Of Tuberculous Joint Disease in general* . . . . . 72
*Of certain Clinical Types—*
*Hydrops* . . . . . 74
*Empyema* . . . . . 74
*White Swelling* . . . . . 74
*Arthritis* . . . . . 74
*Caries Sicca* . . . . . 75
*The General Health* . . . . 75
*Causes of Death* . . . . . 75
*Diagnosis* . . . . . 75
*Prognosis* . . . . . 75
*Treatment—*
*Conservative* . . . . . 75
*Operative* . . . . . 78


Tuberculous diseases result from bacillary infection of the synovial membrane, or of the marrow of one or other of the adjacent bones.

The infection occurs under similar conditions to those which have been already described in Diseases of Bone (*vide* vol. i.).

The tuberculous lesions of joints, although having a common origin in infection with the tubercle bacillus, differ widely in their anatomical and clinical features. From the anatomical point of view they may be divided into *those in which the disease originates in the synovial membrane and those which originate in disease of one or other of the adjacent bones.*

The relative frequency of these two types has been variously estimated. The sources of disagreement are to be found in the difficulty in distinguishing them from one another, and in the fact that only the more serious and more advanced forms of the disease are subjected to operation; the milder forms of primary disease in the synovial membrane so commonly recover without operation that they do not figure in the records upon which the estimate of the relative frequency is based. It is probable that the frequency of primary disease in the bone has been exaggerated; Krause, for example, estimates the proportion observed in Volkmann's clinique as 23 per cent of synovial origin to 77 per cent originating in the bones.

The relative frequency varies with the age of the patient and with the joint affected; in children, the number of cases originating in the bones is approximately that given by Krause; on the other hand, primary disease in the synovial membrane is relatively more frequent in adults. The predominance of bone lesions in childhood and youth is to be ascribed to the conditions associated with the growth of the skeleton, especially at the ends of the long bones.

As regards the joint affected, the maximum frequency of osseous lesions is found in the hip (26 synovial to 129 osseous, Krause); the proportion is about equal in the case of the knee (266 synovial to 281 osseous, König), and probably also in the case of the elbow, wrist, shoulder, and ankle.

We may preface the consideration of the morbid anatomy of tuberculous joint diseases by referring, in the first place, to the *non-specific lesions met with in joints when tuberculous disease is present in the interior of one or other of the adjacent bones.*

They are comparatively common and are often misunderstood in practice. They resemble those which result from staphylococcal disease in the adjacent bone (*q.v.*). When a tuberculous focus, especially a large one with cascation and a sequestrum, is seated near the articular cartilage or the attachment of the capsular ligament, it gives rise to reactive changes in the adjacent joint, characterised by exudation and by the prolongation of the synovial membrane over the articular surfaces. Adhesions may result, which may obliterate the cavity of the joint, or divide the cavity into different compartments. These phenomena are best observed in the knee. They are analogous to the changes in the pleura in disease of the subjacent lung and in the peritoneum in disease of the abdominal viscera. They are of importance because they interfere with the functions of the joint; and in the event of rupture at a later period of the osseous focus into the joint, they may limit the articular infection to a small area, and may altogether prevent the development of the graver forms of tuberculous joint disease.

*The infection of the joint from disease in the*

*adjacent bone* may take place *at the periphery* from the osseous focus reaching the surface of the bone at the site of the reflection of the synovial membrane; the infection begins at this point, and then spreads to the rest of the membrane; or it may take place *in the central area*, by a flood of tuberculous pus escaping into the joint through a hole in the articular cartilage, or by the projection of tuberculous tissue into the joint following upon the gradual erosion of the cartilage.

*Tuberculous Lesions of the Synovial Membrane and of the Articular Surfaces.*—The nature of the changes in the synovial membrane depends upon whether the disease originated in the synovial membrane or in the bone, and in the latter case, whether the osseous focus has erupted directly into the cavity of the joint or has only infected the synovial membrane at the line of its reflection on to the bone, and whether the joint was normal or not at the moment of infection.

In the majority of cases the first evidence of disease in the joint is *diffuse thickening of the synovial membrane*; this thickening is chiefly due to the formation of granulation tissue or young connective tissue in the substance of the membrane. It may be described as being arranged in two layers; the outer layer is composed of more fully-formed connective or fibrous tissue, while the inner consists of embryonic tissue, usually studded or permeated with miliary and other tubercles. The tubercles are met with in all stages in the same joint, some in course of active formation, others quiescent, others again in course of retrogression and cicatrisation. They may be seen shining through the moist shining layer on the free surface, or the inner layer of the synovial membrane may undergo fibrinous degeneration followed by caseation and disintegration, so that the free surface is covered with a thin layer of fibrinous or caseous pus, and a similar material may accumulate in the cavity of the joint. Where there is greater resistance on the part of the tissues there is active formation of young connective tissue circumscribing or encapsulating the tubercles, so that they remain embedded in the substance of the synovial membrane, and are only revealed when it is cut in sections; the surface of the membrane then retains its smooth shining character, and there may be no fluid in the cavity of the joint.

The new formation of tissue in relation to the synovial membrane is rarely confined to its normal limits; it tends to infiltrate the ligaments and to be projected into the cavity of the joint, filling up its pouches and recesses and *growing over the surface of the articular cartilages* like ivy growing over a wall. Wherever the synovial tissue covers the cartilage it becomes adherent to it and fused with it, for covered cartilage always undergoes a retrograde metaplasia into ordinary connective tissue. The morbid process may be arrested at this stage, and may cure with fibrous adhesions between the opposing articular surfaces, or it may progress, in which case further changes occur which result in *destruction of the articular cartilages and exposure of the subjacent bone.*

The synovial connective tissue covering the cartilage may at first present no structural evidences of the presence of tubercle, but in time it acquires the characters of a tuberculous infiltration and exhibits aggressive qualities; it causes pitting and perforation of the cartilage, it makes its way through the cartilage, and often spreads widely between the cartilage and the subjacent bone so as to separate the cartilage in portions of considerable size. These changes are commonly spoken of as "*ulceration and exfoliation of the articular cartilage.*" They usually commence and are most marked at the points of junction of synovial membrane and cartilage, viz., at the margins of the articular surfaces, and at the points of attachment of such intra-articular ligaments as the round ligament in the hip and the crucials in the knee. The cartilage is also destroyed more rapidly and extensively when it overlies a caseating, sclerosed, or other focus in the bone; the latter being then exposed in the joint contributes to the progress and aggravation of the disease.

To a certain extent the cartilage may be regarded as a barrier to the spread of tubercle, protecting the joint where the disease originates in the bone, and protecting the bone where the disease begins in the synovial membrane.

*Carious changes in the subchondral bone* usually follow upon the destruction of the articular cartilage, and are associated with tubercular infiltration of the marrow in the surface cancelli, and breaking up of the spongy framework of the bone into minute irregular fragments; this disintegration of the surface bone is known as *caries.*

The mutual pressure of articular surfaces against one another, resulting from the contraction of muscles and other factors, favours the progress of ulceration of cartilage and of articular caries; these are usually more advanced in areas most exposed to pressure, *e.g.* on the superior aspect of the head of the femur and on the posterior and upper segment of the acetabulum.

When the destructive changes in the articular surfaces are very pronounced, and at the same time there is an absence of caseation and suppuration, the condition has been called *caries sicca.*

The occurrence of *pathological dislocation*, while possible in any joint, has been specially observed at the hip. It implies softening and stretching of the ligaments which retain the bones in their normal position and some ex-

citing factor causing displacement; this may be the accumulation of fluid in the joint or of granulations filling up the socket, or the involuntary contraction of muscles or some movement or twist of the limb. In some cases the occurrence of dislocation is favoured by destructive changes in the bones, *e.g.* diminution in the size of the head of the femur, and enlargement or actual destruction of a portion of the socket of the acetabulum. The dislocation may be complete or incomplete. It may take place gradually and insidiously, or suddenly, especially when it results from some slight form of external violence, or the spasmodic contraction of muscles acting on the joint.

*Rarer Forms of Synovial Tuberculosis.*—While the diffuse thickening of the synovial membrane, above described, is the most common form of synovial tuberculosis, there are others worthy of mention. The synovial membrane may present *nodular masses or lumps*, resembling the tuberculous tumours met with in the brain; they project into the cavity of the joint, may be pedunculated, and may give rise to the symptoms of loose body. In rare instances the fringes of the synovial membrane may undergo a remarkable development, like that observed in arthritis deformans, and may deserve the name *arborescent lipoma*. Both these types are met with in the knee-joint.

*The Contents of Tuberculous Joints.*—In a large proportion of cases of synovial tuberculosis the joint cavity is entirely occupied by the diffuse thickening of the synovial membrane, and there is an absence of fluid in the joint. In a small number there is an *abundant serous exudation*, as in a pleurisy, and the condition is known clinically as *hydrops*. There may be a considerable *formation of fibrin within the joint*, covering the free surface of the membrane, and floating in the fluid as shapeless flakes or masses; under the influence of joint movements they may assume the shape of *melon seed bodies* (*corpora oryzoidea*). More rarely the joint contains tuberculous pus, and the surface of the synovial membrane resembles the wall of a cold abscess (*empyema of joints*).

*Periarticular Tubercle and Periarticular Abscesses.*—These may result from the eruption on the periosteal surface of foci in the interior of the bones, or from the extension of foci in the synovial membrane into the surrounding cellular tissue, either by direct continuity or by way of the lymphatics. A collection of pus within the joint may perforate the capsule and infect the tissues outside the joint. The periarticular abscesses, after spreading in various directions, finally reach the skin surface and give rise to tortuous sinuses; the more distant sinuses may be the result of the spread of tuberculosis along the tendon sheaths in the vicinity of the joint.

*Reactive changes in the vicinity of tuberculous joints* are of common occurrence, and play a considerable part in the production of what is known clinically as *white swelling*. New connective tissue forms amidst the periarticular fat and between muscles and tendons; it may be fibrous and tough, or it may be soft, vascular, and œdematous; the periarticular fat may become swollen and gelatinous, constituting a layer of considerable thickness, in which tubercle may be entirely absent. This is commonly known as *gelatinous degeneration*. It is supposed that the fat disappears and is replaced by a mucoid effusion between the fibrous bundles of connective tissue, these changes resulting from interference with the circulation and nutrition of the tissues concerned. In the case of the wrist the newly-formed connective tissue may fix the tendons in their sheaths, and may seriously interfere with the movements of the fingers. In relation to the bones there may be reactive changes, resulting in the formation of spicules or scales of new bone on the periosteal surfaces and at the attachment of the capsular and other ligaments.

*Terminations and Sequelæ of Tuberculous Diseases of Joints.*—The disease may cure at any stage, the tuberculous tissue being replaced by healthy connective and scar tissue. Recovery is apt to be attended with impairment of movement even in mild forms of the disease. This may depend upon limited adhesions, or upon ankylosis, or upon contraction of the periarticular structures. Encapsulated caseous foci in the interior of the bones may remain latent indefinitely, or may be the cause of a relapse of the disease at any future period. Elongation of the shafts of the bones of the affected limb may result from the stimulation of growth at the epiphysial junctions, but it is much rarer than in the staphylococcus osteomyelitis of young children. Interference with growth is more common; it may involve only the epiphysial junctions in the immediate vicinity of the joint affected, or it may involve all the bones of the limb: this is well seen in adults who have suffered from disease of the hip in childhood—the entire limb, including the foot, may be shorter and smaller than the corresponding parts of the opposite side.

Atrophic conditions are also met with from prolonged disuse of the limb; the bones may undergo fatty atrophy, with enlargement of the medullary canal, and marrow spaces, and thinning of the rigid framework; in extreme cases the bones may be cut with a knife or may sustain spontaneous fracture. This is to be borne in mind in forcible manipulations of stiff joints. These atrophic conditions are recovered from when the limb resumes its normal functions.

*Relative Frequency of Tuberculous Disease in different Joints.*—Available statistics enable us to place the various joints in the following order of frequency: spine, knee, hip, ankle and tarsus,

elbow, wrist, shoulder. It is probable that the frequency in the joints of the upper extremity is understated, because the subjects thereof are often treated as out-patients and do not figure in the statistics.

*Relative Frequency of different Joints at different Ages.*—While the wrist and shoulder are rarely affected in children, disease of the hip and spine is essentially a disease of childhood and youth, and rarely commences after the skeleton has attained maturity. Disease of the knee, while very common in children, may be met with at any period of life. The elbow and ankle exhibit little age preference, but are chiefly affected during childhood and youth.

Clinical Features.—These vary indefinitely with the different anatomical forms of the disease, with the joint affected, and with the individual tendencies of the patient. The symptoms do not always correspond with the nature and severity of the tuberculous lesions.

The onset is usually insidious, its date uncertain, and often misstated by the patient. Sometimes the disease is ushered in with fever and with pains in several joints before settling down in one or other joint. This method of onset was described by John Duncan under the title of "*Tuberculous arthritic fever.*" It has frequently been mistaken for rheumatic fever, from which it may usually be distinguished by the absence of any real migration from joint to joint, the absence of sweating, of visceral complications, and the failure of salicylates to influence the progress of the disease. The formation of a cold abscess or the presence of a sinus may afford valuable corroboration of the tuberculous nature of the lesion.

While it is the rule for tuberculosis to affect one joint, it may involve several, either simultaneously, as above described, or one after another.

The initial symptoms may be those associated with the presence of a focus in the neighbouring bone; such an osseous focus may be hidden for years, perhaps causing neuralgic pains in the joint, and suggesting a diagnosis of hysteria, or the complaint may be of weakness, tiredness, stiffness, and inability to use the limb; the symptoms improve with rest and relapse after exertion. These symptoms may be erroneously interpreted until the diagnosis is cleared up by the rupture of the focus into the joint. Buried foci in the trochanter and in the neck of the femur may give rise to most of the symptoms of hip disease without actual infection of the joint. Even large caseous foci may exist for long periods without infecting the joint, from which they may be only separated by the articular cartilage. It is rarely possible to recognise these buried foci in the vicinity of joints by external examination; if they are near the surface in a superficial bone, such as the head of the tibia, there may be local thickening of the periosteum, œdema, pain, tenderness on pressure and on percussion; large soft foci might be revealed by the X-rays; the patient may not be seen until the formation of an abscess and of a sinus, or, still more unfortunately, until the focus has ruptured into the joint. It is of great practical importance to recognise such buried foci, for by treating them promptly and radically joint disease may be prevented.

Tuberculous joint diseases are nearly always insidious in development and chronic in progress; they *occasionally follow an acute course*, resembling that of the "acute arthritis of infants" of pyogenic origin; this has been observed in very young children, especially in the knee, the lesion being synovial in origin and attended with the accumulation of pus in the joint; if treated promptly by incision, recovery is rapid, and free movement of the joint may be preserved.

The onset and initial progress of the disease is more often insidious, and is attended with so few symptoms that it may have obtained a considerable hold over the joint before it attracts definite notice. After some extra use of the limb or some slight injury the disease becomes more active; it is customary for patients or their friends to attribute the disease to such an injury. The symptoms may subside under rest, only to relapse again with use of the limb; there may be successive improvements and relapses in the course of months or years. The milder forms of synovial tuberculosis may entirely recover; the severer bone lesions tend to cause persistent, relapsing, and more aggravated forms of joint disease. In the absence of other evidences of the presence of bone lesions their existence may be inferred from the mere persistence of the disease. In addition to the well-known symptoms of joint disease, such as *pain*, *swelling*, and *heat*, attention must be specially directed to the wasting of muscles, the impairment or loss of the normal movements of the joint, and the development of abnormal attitudes of the limb. *The wasting of the muscles* is a constant accompaniment of tuberculous joint disease; it is attributed to want of use and to an influence reflected from the trophic centres in the spinal cord; it is especially well seen in the extensor muscles of the thigh in disease of the knee, and in the deltoid in disease of the shoulder; the affected muscles become soft and flaccid, and exhibit tremors on attempted movements and a diminution of reaction to the faradic current; the muscular tissue may be largely replaced by fat. *The impairment or loss of the normal movements* varies in degree according to the nature and seat of the disease. In the early stages of synovial tuberculosis the movements may be merely restricted in range and in quality. In the case of the joints of the lower extremity there is

usually a limp in walking. When the articular surfaces are involved, all movements, whether active or passive, are usually abolished, and the condition presented is one of *fixation* or *rigidity*; this results from involuntary contraction of the muscles; it disappears under an anæsthetic and returns again on waking. Its recognition is of great diagnostic value, especially in such deeply-seated joints as the shoulder, hip, and in those of the spine. *Abnormal attitudes of the limb* may precede other symptoms of joint disease, but are more frequently of later development; they are best illustrated by the well-known attitudes assumed in disease of the hip and knee (*q.v.*). Their production was ascribed by Bonnet to increased pressure within the joint and distension of the capsular ligament; their real cause is the reflex or involuntary contraction of the muscles acting on the joint, with the object of placing it in an attitude in which there will be least suffering. Certain groups of muscles, *e.g.* the flexors at the knee, the flexors and abductors or adductors at the hip, assume the upper hand, either because they are more powerful or because they are specially thrown into contraction. These attitudes disappear under chloroform unless secondary changes have occurred causing contracture or ankylosis. In very indolent and mild cases these abnormal attitudes may be absent altogether. They occur earlier, and are more pronounced in cases in which pain and other irritative symptoms of articular disease are well marked. With the lapse of time these attitudes may not only become exaggerated, but may become permanent deformities from changes within the joint in the direction of ankylosis, and from changes in the surrounding soft parts, *e.g.* shortening or contracture of the ligaments, muscles, fasciæ, skin, and it may be also in the vessels and nerves. The occurrence of *startings at night*, which are frequently met with in the stage of muscular fixation, are the result of the sudden contact and jarring of the diseased articular surfaces when the muscles are relaxed during sleep; they are to be regarded as indications that the disease of the articular surfaces is progressive. They are more often met with in cases which go on to suppuration.

*The formation of abscess* is one of the commonest accompaniments of tuberculous joint disease; it may appear early and play a prominent part in the clinical features, or it may develop long after the original disease has settled down; it usually indicates the existence of a persistent lesion, and very often an osseous focus of some importance. It is said to be met with more often in patients with an inherited predisposition to tuberculosis, in those with multiple lesions of the skeleton, and in those who are run down and emaciated. The formation of the abscess is often attributed to a slight injury; it develops so insidiously that it may not attract the attention of the patient until it has attained a considerable size; this is especially the case with the abscesses which are associated with disease of the spine, pelvis, and hip. The abscess presents itself at definite situations in relation to the different joints, the selection being influenced by the anatomical relationships of the capsule and of the synovial membrane to the surrounding tissues. The bursæ and tendon sheaths in the vicinity may influence the direction of spread of the abscess and the situation of the resulting sinus or sinuses. It will be referred to under treatment that the formation of abscess in the course of tuberculous joint disease may sometimes be an advantage, for it may render a tuberculous focus more amenable to treatment, especially by the injection of iodoform. When left to itself, however, or when opened without precautions, abscess formation implies the risk of pyogenic infection, of persistent discharge, aggravation of the associated joint disease, progressive impairment of the general health, and greater liability to tuberculous meningitis. It was formerly possible to observe how often the course of the disease was altered for the worse, the inflammatory symptoms became more acute, the pain greater, the fever higher, the swelling increased, the skin over the joint red, hot, and œdematous, and the discharge coming to resemble ordinary pus.

When sinuses have been allowed to form, their course is often so tortuous that it may be difficult or impossible to pass a probe down to the focus from which the abscess took its origin. Infection of the lymphatic glands of the limb is exceptional; it may, however, follow upon infection of the skin around the orifice of a sinus.

*The occurrence of pyrexia* in tuberculous joint disease is usually an indication of the local progress of the disease in the direction of suppuration, or of the development of complications elsewhere in the body; including dissemination of the tubercle, *e.g.* to the lungs, membranes of the brain, etc. A little rise of temperature in the evening may be induced in quiescent joint lesions by any mechanical disturbance of the tissues involved, by travelling or other exertion, by injury, by movement of the joint under chloroform for purposes of diagnosis, or for the correction of some abnormal attitude or deformity. The development and progress of an abscess may also be attended with an evening rise of temperature; when the abscess is quiescent the temperature usually remains normal. A carefully taken temperature chart may afford useful information as to the formation or spread of abscess. When sinuses have formed and have been allowed to become septic, there may be a diurnal variation in the temperature of the type known as hectic fever.

### Clinical Types of Tuberculous Joint Disease

1. *Tuberculous Hydrops.*
2. *Cold Abscess or Tuberculous Empyema.*
3. *White Swelling.*
4. *Tuberculous Arthritis.*
5. *Caries Sicca.*

1. *Hydrops Tuberculosis* is the name given to that form of tuberculous joint disease in which the outstanding feature is the accumulation of serous fluid within the joint. It is analogous to the ascitic type of peritoneal tuberculosis. It is most often met with in the knee of young adults. Inasmuch as it frequently terminates in recovery with a useful joint, it may be regarded as the least serious form of tuberculous joint disease. It will be further described under "Knee-Joint, Diseases of."

2. *Cold abscess; empyema of joints* is the name given by König to that form of tuberculous joint disease in which the outstanding feature is the accumulation of pus in the joint. It is analogous to the purulent type of peritoneal tuberculosis. Its clinical features will be described under "Knee-Joint, Diseases of."

3. *White Swelling of Joints (Synovial Fungus).—Tumor albus* is the name originally applied by Wiseman in 1676 to that form of tuberculous disease which is characterised by the gradual development of a solid swelling in the area of a joint. The swelling is to a considerable extent the result of reactive and mucoid changes in the fat and connective tissue surrounding the capsule of the joint, as well as of tuberculous thickening of the synovial membrane. It is not to be regarded as a distinct pathological type of tuberculous joint disease, for it may originate from primary tuberculosis of the synovial membrane as well as from disease in the bone, while the changes within the joint and the course of the disease necessarily vary within very wide limits; at the same time, the appearances of white swelling bulk so largely in the clinical features of a large number of cases of tuberculous joint disease that it is probably the best known clinical type. It is only recognised in joints which are superficial, viz., the knee, ankle, elbow, and wrist. White swelling of the hip or shoulder is not described. The initial symptoms are those of swelling rather than those of implication of the articular surfaces, even although the disease may have originated in the bone. The swelling develops gradually and painlessly, obliterating the bony prominences and outlines by filling up the natural hollows; the overlying skin is white; the swelling appears greater to the eye than is borne out by measurement, because of the wasting of the muscles above and below the joint; in the early stage the swelling is elastic, doughy, and non-sensitive, and corresponds very accurately to the superficial area of the synovial membrane involved; appearing at first over the *cul de sac* or recess of the synovial membrane, and later over the interval between the bones. At this stage there is comparatively little complaint on the part of the patient, for the articular surfaces and ligaments are still intact; there may be a feeling of weight in the limb; in the case of the knee and ankle the patient may tire on walking, and drag the leg with more or less of a limp; passive movements are comparatively free and painless, although usually limited in range. The disability of the joint is increased by use and exertion, and improves under rest, for a time at any rate. As the disease progresses, the signs and symptoms become slowly exaggerated; the skin over the joint becomes tense and hot, the swelling, which was at first solid, may show areas of softening, and later of fluctuation, and a cold abscess may form, may burst, and result in one or more sinuses. The wasting of muscles becomes more marked, the joint becomes more rigid as the articular cartilages become affected, and the attitude of flexion is very commonly assumed, more especially in the case of the knee. Startings at night indicate the occurrence of destructive changes in the articular surfaces. The final condition is one of disorganisation of the joint, with deformity and septic sinuses.

4. *Tuberculous Arthritis.*—It is convenient to group under this heading those cases of tuberculous joint disease in which the outstanding clinical features are the result of implication of the articular surfaces. Although, as already indicated, these symptoms commonly develop in the later stages of white swelling, it is a matter of everyday experience that symptoms of implications of the articular surfaces may be the *first* evidences of tuberculous joint infection, and may exist without white swelling or any other clinical evidence of disease in the synovial membrane. These remarks specially apply to such deeply-seated joints as the hip, shoulder, and spine, which never present the phenomena of white swelling, but they are also applicable to other joints. The recognition of this arthritic form of tuberculous joint disease depends rather upon inferences founded upon certain symptoms and signs than upon direct examination of the joint concerned. The patient complains of pain at the site of the disease, or he refers it to some other part with which it is connected through the nerves; the pain is aggravated by movement and by the manipulations of the surgeon; in the case of the joints of the lower extremity the patient will limp in walking; the movements of the joint are restricted, very often to the extent of fixation or rigidity. The recognition of rigidity is one of the most valuable evidences in the diagnosis of disease at this early stage, especially in deeply-seated joints.

The wasting of muscles is more marked than in the early stage of white swelling. Very commonly the limb is placed in an abnormal attitude by the contraction of special groups of muscles; these attitudes are well illustrated in disease of the hip. If the disease is left to itself and progresses, all the other well-known signs of joint disease may make their appearance, *e.g.* startings at night, the formation of abscess and of sinuses, displacement or even dislocation of the bones; until under an anæsthetic the joint may be found to be completely disorganised, with destruction of ligaments, abnormal mobility, and grating of the articular surfaces.

5. *Caries sicca* is the name given by Volkmann to a very chronic form of tuberculous arthritis, chiefly met with in the shoulder and hip of adults between the ages of fifteen and thirty-five. There is an entire absence of swelling. The wasting of all the structures in the vicinity of the joint is characteristic; the bony prominences, such as the acromion and coracoid in the case of the shoulder and the trochanter at the hip, stand out prominently. Passive movements are very restricted and are attended with severe pain. The general health usually remains unimpaired in spite of the long duration of the disease. In exceptional cases an abscess may form; it is usually small and extra-articular, and is related to a sequestrum.

Krause has observed cases in which, after many years of indolent progress, there has developed without any apparent cause the most acute suppurative and destructive changes in the joint, necessitating operation without delay; in excising the joint it is found that the head of the bone may have almost or entirely disappeared, and that the tissue of the neck is dense and sclerosed, proving conclusively that the disease was really one of long standing.

*Influence of the Joint Disease on the General Health. Causes of Death.*—While experience shows that tuberculous joint disease may be compatible with good general health, the tendency is for it to be affected when the disease is serious and persistent. Sherman has observed in children a diminution of the red blood corpuscles and of the hæmoglobin. Dane has observed leucocytosis when an abscess is forming and when septic infection is superadded. The appetite is impaired. The patient is easily tired and complains of loss of strength, especially when there is fever. The skin is dry. The loss of flesh may amount to emaciation. *Albuminuria* is a frequent accompaniment of joint lesions which are suppurating; it usually results from waxy disease of the kidneys, but may be a sign of parenchymatous nephritis; general dropsy is a more unfavourable indication of the interference with the renal functions. A considerable number die from exhaustion with all the accompaniments of hectic fever. *Tuberculous disease of the lung* is very frequent in the case of adults; it may be present at the time when the joint disease begins, or it may appear later. *Tuberculous disease of the intestine* is not uncommon. In the case of children *acute miliary tuberculosis* is more common than either, and is usually rapidly fatal; it may occur without apparent cause, or it may follow immediately on operative interference (comparatively trifling operations, *e.g.* scraping of sinuses); the clinical features are frequently those of basal meningitis.

*The Diagnosis of Tuberculous Joint Disease.*—The family history, the presence or history of tuberculous lesions elsewhere in the body, the insidious onset, the fact that there is usually an interval between the appearance of symptoms and the receipt of an alleged injury, all suggest the tuberculous origin of any given case of joint disease. The diagnosis is usually quite easy in typical cases. One should not be misled by the age of the patient or by the appearance of excellent health The X-rays are chiefly of value in the recognition of osseous lesions. The details of differential diagnosis are beyond the scope of the present article. The use of Koch's tuberculin is not recommended.

*Prognosis.*—This is not easily stated in general terms, since it varies with the seat, extent, and severity of the local disease, and with the resisting powers of the patient as influenced by the general health, age, and social circumstances. Recovery is never impossible. The existence of tuberculous lesions elsewhere and the formation of septic sinuses are unfavourable factors. While tuberculous lesions in children tend to become circumscribed, in adults the tendency is in the opposite direction. The absence of any response to conservative treatment is unfavourable. The locality of the disease is an important factor; in the limbs the hip-joint is the most serious of all, because there is greater difficulty in treatment, and the disease is often of a serious type, and may be attended with pelvic complications.

The patient and his friends must be informed of the length of time required for complete recovery; it necessarily varies, but it may be stated generally as being from one to three years. The risk of relapse at some future period of life must not be forgotten.

*Treatment.*—In addition to that applicable to all forms of tuberculosis (hygienic, dietetic, etc.), we are here concerned with the local measures directed towards the disease in the joint and its concomitants. These may be described under two heads, the conservative and the operative.

*Conservative Treatment.*—This is almost always to be employed in the first instance, and only when it fails is recourse to be had to operative methods, since with the former a large proportion of cures are obtained with a less mortality, and the functional results are better than those obtained by operation. The essentials of con-

servative treatment include the placing of the joint at *complete rest*, the *correction of abnormal attitudes*, the production of *venous congestion*, and the injection of *iodoform*; all these may not be applicable in every case.

*The treatment by rest* implies the immobilisation of the diseased limb until pain and tenderness have disappeared. It is carried out by means of bandages, plaster-of-Paris splints, or other apparatus according to the joint affected. The attitude in which the limb is immobilised should be that in which, in the event of subsequent stiffness, it will be most serviceable to the patient. *Extension* with the weight and pulley is a valuable adjunct, especially in disease of the hip or knee; it eliminates muscular spasm and the pressure of the articular surfaces against one another; it relieves pain and startings at night; it prevents abnormal attitudes of the limb, and may bring about their disappearance, provided they are not associated with organic changes which render them permanent. The question of abnormal attitudes is discussed in the special articles on the hip and knee, but it may be stated generally that if the limb is in a deformed attitude when the patient first comes under observation, and it does not readily yield to extension, it should be corrected straight away under an anæsthetic. The permanent deformed attitudes which are due to contraction of the soft parts around the joint or to ankylosis will be considered later.

*The injection of iodoform*, if carried out efficiently, is of great value. The preparation employed is a 10 per cent emulsion of powdered iodoform in glycerine, which becomes "sterile" soon after it is made. Its curative effects would appear to depend upon its antiseptic properties, which, although slight, continue in action for weeks or months, and upon its capacity for irritating the tissues and stimulating the formation of scar tissue. The usual antiseptic precautions are imperative. An anæsthetic is rarely called for.

*If it is proposed to inject the cavity of an abscess* the contents are first evacuated through a medium-sized trocar, introduced obliquely, avoiding any part where the skin is thin or red. If the trocar is blocked with caseous material it must be cleared with a probe. The iodoform is injected by means of a glass barrelled syringe which will screw securely on to the trocar. The amount injected varies from 4 cc. in small children to 30 cc. or more in adults. The puncture is covered with a pad of gauze, and a dressing is applied which will exert a certain amount of compression.

*If it is preferred to make an incision into the abscess* (and this may be necessary where the contents are semi-solid, or may be indicated when it is intended to clear out a localised focus in the bone) the cavity is emptied, and the iodoform is injected through a rubber tube attached to the syringe, simultaneously with the closure of the wound by sutures, so that the cavity when closed will be distended with the emulsion. If the abscess wall has been scraped with the spoon a less amount of iodoform is injected, as the drug is more likely to be absorbed. The method is unsatisfactory unless the wound heals by first intention.

The iodoform injection may require to be repeated after an interval of three to six weeks; the author has, however, repeatedly cured retropharyngeal, lumbar, and psoas abscesses by a single injection, sometimes through the trocar, sometimes through an incision.

*If it is proposed to inject the iodoform into the joint* the procedure varies with the nature of the lesion. In cases of hydrops and of empyæma the method is the same as in abscess, and is easily carried out, especially in the knee. The sites to be selected for injecting the different joints are as follows:—(Krause) wrist, just below the radial or ulnar styloid process; elbow, just above the head of the radius; shoulder, either outside the coracoid or at the junction of the acromion with the spine of the scapula; ankle, below either malleolus, then direct the trocar upwards; knee, by way of the suprapatellar pouch, or between the bones at the inner side of the ligamentum patellæ; hip (see "Hip-Joint, Diseases of"). When the joint is not distended with fluid, *e.g.* in white swelling, the conditions for distributing the iodine are less favourable. In the knee, the joint may be divided up into compartments by septa of connective tissue derived from the synovial membrane, and the iodoform must be introduced into all of them. The trocar must be thrust in all directions, successive parts of the joint being attacked at different sittings, and the injections must be repeated more frequently and at shorter intervals (ten to fourteen days). Opinion is divided on the question of massage and gentle passive movements with the object of distributing the iodoform; some believe that this entails the risk of disseminating the tubercle bacilli. *Injections into the substance of the synovial membrane* (parenchymatous injections) are less certain, are more painful, and require greater pressure on the piston of the syringe. Mikulicz and others attempt to inject the iodoform *into the adjacent bones* when they are soft enough to allow of the entrance of the trocar. Wherever the iodoform is introduced it remains for long periods, and may be seen as a dark shadow in skiagraphs.

After any form of iodoform injection, one must be prepared for considerable reaction, attended with fever (101° F.), headache, malaise, and it may be sickness for from one to three days, and considerable pain and swelling of the joint. The reaction diminishes with each subsequent injection.

*When an abscess has ruptured and left a sinus*,

an olive-shaped nozzle must be attached to the syringe which will completely close the orifice of the sinus and prevent the immediate escape of the emulsion, which must be forced into the sinus; the orifice of the latter is closed with the finger for ten minutes; it will then be found that only a little clear glycerine escapes; pads of gauze are applied, and the procedure repeated two or three times a week. The results are very good if the sinus is not already septic. Septic sinuses are better laid open, sterilised, and made to heal from the bottom by the open method. It is maintained that even the existence of sequestra is not an obstacle to the success of the iodoform treatment, for if they are thoroughly soaked in iodoform and glycerine, the bacilli may be exterminated and the sequestra may become encapsulated by connective tissue.

*The artificial production of venous congestion*, introduced by Bier, is an important adjuvant to the iodoform treatment. To be successful it must be efficient and carefully supervised. An elastic webbing bandage (two or three turns) is applied outside a layer of lint immediately above the affected joint, sufficiently tightly to constrict the veins and produce a bluish-red tinge of the skin. Below the joint the limb should be bandaged to prevent œdema. The site of application of the elastic bandage should be changed frequently so as to avoid maceration of the skin and diminish the tendency to wasting of the muscles. It may be worn continuously, but the intermittent application is better, from fourteen to eighteen hours each day; in the interval the œdema around the joint may be dispelled by a bandage. The venous congestion appears to act beneficially by stimulating the formation of connective tissue. It has been objected to by some surgeons because under its influence latent tuberculous foci have been transformed into cold abscesses; this is no drawback, since the cold abscess is more amenable to iodoform treatment than the latent focus. The congestion should be persevered with, in suitable cases, until a month or two after the joint disease appears to have been cured. The congestion should be omitted for two or three days after iodoform has been injected. The congestion treatment is not applicable to the hip or shoulder.

*Under the combined influence of rest, iodoform injections, and venous congestion*, if the disease of the joint undergoes cure the pain and tenderness subside, passive movements become possible, and the swelling gradually subsides; it is a favourable sign if the swelling becomes harder and firmer. In the later stages of treatment the patient is encouraged to remain in the open air; in the case of the lower extremity the limb must be maintained in a good attitude, and should not be allowed to touch the ground. In the evening, the limb should be washed, massaged, gently exercised, and the wasted muscles may be stimulated with electricity.

The results obtained by the foregoing methods of conservative treatment, along with attention to the general health, are in the majority of cases extremely satisfactory. The best and most certain results are obtained in children. Apparently permanent cures are obtained in cases which were formerly subjected to all kinds of severe operative interference. It is unfortunate that we can rarely tell beforehand whether it is certain to succeed, or how long the cure will take. An exception must be made in *disease of the knee-joint in adults*; opinion is becoming unanimous that if there is no prospect of obtaining a movable joint by conservative measures, it is better to have recourse to excision in the first instance, for thereby one may guarantee the best obtainable functional result with the minimum expenditure of time. In other joints the conservative treatment is only abandoned if the disease continues to progress in spite of it, if improvement does not show itself after a thorough trial, or if the disease relapses after apparent cure (*vide* indications for operative interference). The external application of iodine or of mercurial ointment (Scott's dressing) is of doubtful value; the fly blister and the actual cautery have largely gone out of fashion, but they may be employed with benefit for the relief of pain when this is a prominent feature.

*Operative treatment: the indications for operative interference* vary in each case; they are more restricted than was the case during the era when Listerian methods first eliminated the septic complications of wounds. So far as *the general condition of the patient* is concerned, age is an important factor. Other conditions being equal, operation is more often required in adults, because after the age of twenty there is less prospect of spontaneous recovery, there is more tendency to relapse and to tuberculous disease of the internal organs, and there is no fear of interfering with the growth of the skeleton. The *general health* may necessitate the removal of the disease by the most rapid method, viz., by operation.

*The social status* must, unfortunately, be taken into account; the bread-winner, under existing social conditions, may be unable to give up his work for a sufficient time to give conservative measures a fair trial.

*The local conditions* which decide the question for or against operations are differently regarded by each individual surgeon. They may be expressed in general terms, for those who have no personal experience to guide them, as follows: — Operative interference is indicated (1) in cases where, in spite of a fair trial of conservative measures, the disease continues to progress; (2) in cases unsuited for conservative treatment, *e.g.* where there is dislocation,

separation of epiphysis, or deformity incapable of being rectified otherwise, when there are sinuses with septic infection, and the operation affords a reasonable prospect of getting rid of both the tuberculosis and the sepsis, and when the disease is associated with severe bone lesions (*e.g.* large sequestra, central abscess of bone), or threatened with infection of the lymphatics; (3) in cases where the results of operative interference will be as good or better than those likely to be obtained by conservative measures; this has been already discussed in relation to the knee, and the advice given that if in the adult the joint is likely to be stiff, then this result is more certainly and rapidly obtained by excision. The same indication applies to the knee in children; the operation performed, however, must not entail any interference with the epiphysial discs; the articular cartilages are pared with a strong knife instead of removing the ends of the bones with the saw. The same indication also applies to the elbow both in adults and in children; if the joint is likely to be stiff, and this result will not comply with the requirements of the patient, much time will be saved by an immediate excision, thereby securing a movable joint and getting rid of the disease at the same time. In other joints the functional results obtained by conservative measures (excepting under the conditions mentioned above) are usually superior to those following operation; they are therefore persisted in so long as there is any prospect of their leading to a cure of the disease.

*The nature of the operative interference* varies with the patient, the joint affected, and the type and extent of the disease. In many cases it can only be decided after exploration of the joint. The operative treatment of the present day is different from the old method of excising joints in which the bones were removed with the saw and the diseased soft parts left behind. The modern tendency is not to proceed on stereotyped lines, but to perform atypical operations directed to the special features of each individual case. Experience is therefore an important adjunct to pathological knowledge.

The chief aim is to remove all the disease with the least impairment of function. The sacrifice of healthy tissues is reduced to a minimum. The more *open* the method of operating the better, so that all parts of the joint are available for inspection, and the principal incision must be so planned as to achieve this object without unnecessary damage to the essential structures of the joint and of the overlying soft parts; the methods introduced by Kocher comply with these conditions, especially those which permit of dislocating the joint, since this procedure affords the freest possible access for inspection and for removal of the disease. Cold abscesses or sinuses should be cured, if possible, before operating on the joint. Diseased synovial membrane is removed with the scissors or knife, sparing its fibrous layer if possible. If the cartilages are sound they may be left (excepting always the knee if a rigid joint is the aim of the operation). If the cartilage is diseased at any point, it should be removed so as to permit of investigating the bone beneath; if extensively separated it should be removed entirely, and special attention directed to the bones. The most minute sinus in bone must be followed up in case of its leading to a caseous focus or sequestrum. If the surface-bone is diseased, a thin slice of it should be removed with the knife or saw. If foci are then revealed it is often better to dig them out than to remove further slices of bone, thereby sparing the cortex and the periosteum. The uninitiated must not mistake fatty marrow for disease.

Further details belong to the surgery of the individual joints. The limb should be rendered bloodless before commencing the operation. The technique should be antiseptic, rather than aseptic, so as to diminish the chances of tuberculous infection of the wounded surfaces; with the same object in view, as well as to overcome any minute tuberculous foci which may have escaped detection and removal, a small quantity of sterilised iodoform should be rubbed into the raw surfaces and recesses of the joint.

Closure of the entire wound without drainage may be successful in selected cases; inasmuch, however, as an accumulation of blood-clot affords an admirable soil for the development of any tubercle bacilli which have been left behind, it is safer to employ some means of preventing the accumulation of blood in the wound; the most reliable is to pack the wound with iodoform worsted or gauze, bringing out the end of the strand or strands at one point of the main wound (or through a small wound made for the purpose). If the temperature remains normal the packing is left for a week; it is then moistened with iodoform-glycerine to allow of its being removed without bleeding; a less amount of packing is then introduced, or the whole wound is filled up with the iodoform-emulsion by means of the injection syringe and a rubber tube, the end of which is inserted into the deepest part of the wound; a suture or gauze pad is then applied to prevent the escape of the emulsion. If there is septic infection either in the first instance or subsequently, the whole wound should be stuffed and treated by the "open method." If a rubber drainage tube is employed to prevent the accumulation of blood, it should be removed in twenty-four or forty-eight hours.

Where there are sinuses they must be treated as already described. They are often an indication for treatment by the "open method." It will be observed that nothing has been said of *the respective merits and spheres of arthrectomy*

*and of excision*; the original distinction between these procedures has largely disappeared; the modern atypical operation for the cure of tuberculous joint disease sometimes partakes of the characters of an arthrectomy, sometimes of an excision, but in many cases neither of these terms would accurately describe the operation which best meets the requirements of the case. A formal excision is more often employed in the knee and elbow than in other joints, modified in the case of children in view of the functional importance of the epiphysial junctions concerned. For details the reader is referred to the articles on the individual joints. In the *after-treatment* of cases subjected to operation, it is essential that they should be under direct supervision for several years in case of a relapse of the disease, to promote mobility where the joint is intended to be movable, and to prevent deformities and abnormal attitudes where it is intended to be stiff or rigid. Massage, electricity, exercises, and hydrotherapy promote the recovery of function. When the functional result is good, the wasting and arrest of growth of the muscles and of the limb as a whole are more likely to be recovered from.

*The operative treatment of deformities resulting from tuberculous joint disease* has almost entirely replaced the former attempts by forcible reduction, because of the unsatisfactory results and of the risks involved (fracture, separation of epiphysis, fat embolism, lighting up of quiescent encapsulated foci, etc.). The modern procedure is to divide the contracted soft parts by open operation, and to divide or resect the bone where there is undesirable osseous ankylosis (see individual joints).

*The treatment of relapse or recrudescence of the disease* at the site of operation is carried out on the same lines as for the original disease, and should be had recourse to as soon as it is recognised. The same remark applies to tuberculous disease in the associated *lymphatic glands*.

*Amputation or disarticulation of the limb* for tuberculous joint disease is becoming one of the rare operations in surgery. It is only employed where recovery is otherwise hopeless. The general health and age of the patient, and the occurrence of local and general septic complications, are the chief determining factors. Amputation should never be performed unless it secures a complete removal of the disease both in the bones and in the soft parts. Other things being equal, one has less hesitation in having recourse to amputation in the lower than in the upper limb.

### Syphilitic Diseases

These are decidedly rare as compared with tuberculous diseases, syphilis being much more a disease of bone than of joints. It is probable that their rarity has been over-estimated, because they are not always correctly diagnosed. As in tuberculosis, they may be primary in the joint, or secondary to disease in the adjacent bones. *In acquired syphilis* the joint affections may be described as early and late.

(i.) *The early lesions* occur in what is conveniently described as the secondary period. They may assume the form of an *arthralgia*, corresponding to the bone pains, and affecting the shoulder, knee, wrist, or ankle; the joint becomes sensitive and painful, and the pain is worst at night. There are no organic changes in the joint.

They may assume the form of a *serous synovitis*, sometimes called syphilitic rheumatism, from its resemblance to polyarticular rheumatism; the joint or joints become swollen, hot, and painful, and there may be a certain amount of fever, or of a *hydrops*, which is met with almost exclusively in the knee; it is frequently bilateral; it is very insidious in its onset and progress; the patient may be able to go about; if untreated, it may last for months.

Both the synovitis and the hydrops may closely resemble the corresponding lesions resulting from gonorrhœa; they rapidly and completely disappear, however, under syphilitic treatment.

(ii.) *The late or tertiary lesions* of joints are much more persistent and destructive; they result from the formation of gummata in the extra-articular tissues, either in the deeper layers of the synovial membrane or in the adjacent bone or periosteum; this explains the absence of articular symptoms in the early stages. In the majority of cases severe joint symptoms do not develop unless as a result of breaking down of the gummatous tissue and the addition of septic infection.

*Perisynovial and peribursal gummata* are most often met with in relation to the knee-joint of adults of middle age, and especially of women; the gummata are usually multiple, they develop very slowly, and may be unattended with any symptoms; they are rarely sensitive or painful; in the working classes the patient may not apply for advice until the gumma has broken down and given rise to a tertiary ulcer. The simultaneous presence over the knee-joint of indolent swellings, of ulcers, and of depressed scars is very characteristic. When the gummata do not break down, the resemblance to the white swelling of tuberculous origin may be considerable; attention should be directed to the nodular, uneven, irregular character of the gummatous affection; sometimes the skin is red and tender over a gumma without the decided liquefaction and fluctuation which would accompany reddening of the skin in a tuberculous lesion.

Effusion into the joint is rarely a prominent feature. The gummatous nodules when close

to the synovial lining may project into the interior of the joint, it may be like the fringes in arthritis deformans, and have been known to give rise to the symptoms of "loose body."

Recovery may be attended with considerable stiffness and contracture deformity.

*Gummata in the periosteum or marrow of the adjacent bones* may result in a form of joint disease known as *syphilitic osteo-arthritis*. There is a gradual enlargement of one or other of the bones, attended with neuralgic pains which are worst at night; at this stage the diagnosis from sarcoma may be difficult or impossible; the gummatous disease may extend to the synovial membrane, and may be attended with effusion into the joint, or it may erupt on the periosteal surface and break through the skin, forming one or more sinuses. The further progress in untreated cases is complicated by the occurrence of septic infection and of necrosis of bone. In the knee-joint, the patella or one of the condyles of the femur or tibia may furnish a sequestrum, which may involve the articular surface and impart to the disease a persistent and destructive character. In such cases one should not expect recovery from antisyphilitic treatment alone, it must be supplemented by operative measures directed to the removal of the damaged tissues; excision of the knee is rarely called for, even in the most aggravated cases; in the elbow it may be practised in order to obtain a movable joint.

*In inherited syphilis* the earliest joint affections are associated with the epiphysitis (or syphilitic osteo-chondritis) of young infants; there may occur some effusion into the adjacent joint (knee, elbow); in exceptional cases pyogenic infection may be superadded, and the joint may fill with pus. In children a *serous synovitis* or *hydrops* may develop in the knee of one or of both sides, sometimes in the earlier period, along with iritis, or at a later period along with interstitial keratitis; it is very chronic, and scarcely causes any symptoms. It disappears under treatment without any impairment of the functions of the joint. The *tertiary or gummatous lesions* of joints are the same as have been described as met with in the subjects of acquired syphilis; they are most often met with in relation to the joints of the fingers in syphilitis, but are also met with in the knee and elbow.

### III. Joint Diseases accompanying certain Constitutional Conditions

*Gout.*
*Chronic Articular Rheumatism.*
*Arthritis Deformans.*
*Arthritis Ossificans.*
*Hæmophylia.*

*The gouty affections of joints* are considered in the general article on "Gout" (vol. iv.). Their surgical importance relates to the differential diagnosis and to the occasional necessity for operative interference.

*Chronic rheumatism* is an ill-defined affection of joints which is chiefly remarkable for the amount of suffering to which it may give rise, and the great disturbance in the functions of the joint which may result from it. Its claims to be called rheumatic rest upon the following facts: it usually follows upon acute articular rheumatism; it may show exacerbations or relapses, attended with pyrexia and relieved by salicylates; it is met with in patients who present a family history of acute rheumatism or of inflammation of serous membranes: there may be a history of chorea, or of erythema nodosum, or of rheumatic nodules, or other undoubted evidences of rheumatism.

It is usually polyarticular. It may be met with in childhood and youth as well as in adults. The primary changes in the affected joints almost exclusively involve the synovial membrane, the ligaments, the surrounding tendon sheaths, and bursæ; they consist in inflammatory infiltration and exudation, resulting in the formation of new connective tissue, which encroaches on the cavity of the joint and gives rise to adhesions. The newly-formed connective tissue tends to contract, causing deformity and stiffness. Changes may occur in the articular cartilages secondary to adhesions between opposing surfaces, or as a result of their displacement, so that they are no longer in contact with one another; they consist in the conversion of the cartilage into connective tissue The bones are only affected in so far as they undergo fatty atrophy from disuse, or alteration in their configuration as a result of displacement (subluxation). Suppuration does not occur. Osseous ankylosis may be observed, especially in the small joints of the hand and foot.

*Clinically* the disease is chronic and often incurable. Pain may be so prominent a feature that the patient resists the least attempt at movement. In other cases the joints, although stiff, may be moved, and exhibit pronounced crackings. The joints are enlarged or swollen when there is much new connective tissue formed in relation to the synovial membrane; the swelling becomes more noticeable as the muscles waste above and below the joint. Subacute exacerbations occur from time to time, with fever and with aggravation of the local symptoms and signs. While recovery may take place with ankylosis and deformity, the patient becoming a helpless cripple, the tenure of life is very uncertain because of the tendency to visceral complications.

From the nature of the disease *treatment* is very rarely curative. Salicylates are only of service during the exacerbations attended with pyrexia. Temporary improvement may result from the general and local therapeutics available at such places as Bath, Buxton, Wiesbaden,

Wildbad, Aix, etc. Forcible attempts to remedy stiffness or deformity are to be avoided. A certain measure of success has followed operative interference in selected cases, consisting in a modified arthrectomy, and the injection of an emulsion of iodoform or guaiacol in glycerine. Deformities resulting from chronic rheumatism are treated on the usual lines.

*Arthritis Deformans, Osteo-Arthritis, Chronic Rheumatic Arthritis, Rheumatoid Arthritis, Rheumatic Gout, Malum Senile, Traumatic or Mechanical Arthritis.*—It is impossible within the limits of the present article to attempt to give an account of the group of joint affections which are at present included under the above vague and misleading nomenclature. Excluding those which are definitely gouty or rheumatic, there are provisionally included under the name arthritis deformans or osteo-arthritis a number of joint lesions which, in their etiology and clinical features, differ from each other to such a degree that we can only explain their inclusion in a common group by confessing that we are ignorant of their essential nature. Among the list of names given above, we must be especially suspicious of those which aim at giving a clue to the origin of the disease. Rheumatism and gout are only related to the diseases under consideration in so far as they may precede the latter, and that arthritis deformans is more often met with in families who are tainted with rheumatic or gouty tendencies. The term *malum senile*, implying as it does an association with the changes resulting from advancing years, is singularly inappropriate as a general name for a disease which may be met with in childhood. The suggestion of Arbuthnot Lane's, that the lesions under consideration are the result of a single or repeated trauma, while ingenious and instructive, can scarcely be accepted as conclusive.

The reader will probably agree with the author that it is easier to express the negative in regard to arthritis deformans, than to formulate positive views which are of any real value.

The anatomical changes are so well known that their description may be omitted.

The Clinical Features vary indefinitely; the following are the chief types:—

1. *Hydrops* is frequent in the knee, but may be met with in the elbow, shoulder, ankle, etc.; the patient complains of a feeling of weight, of insecurity, and of tiredness in the joint; pain is occasional and evanescent, and is usually the result of some extra exertion. As the joint fills more and more with fluid the ligaments become stretched, so that the limb becomes weak and unstable; it may be associated with hydrops of the adjacent bursæ. The affection is extremely chronic, and may last for an indefinite number of years. It is to be diagnosed from the other forms of hydrops already considered, viz., the purely traumatic, the pyogenic, gonorrhœal, tuberculous and syphilitic, and from that associated with Charcot's disease.

The symptoms may be relieved by hydrotherapy and massage, and by the support of an elastic bandage; great benefit or even cure may follow the withdrawal of the fluid and the injection of iodoform glycerine.

2. *The presence of fringes and of pedunculated and other loose bodies* may give rise to characteristic clinical features, especially in the case of the knee; they often coexist with hydrops; the fringes, which may assume the luxuriance of what has been described as an arborescent lipoma, project into the cavity of the joint, and may fill up all its recesses and distend the capsule. The joint is swollen and slightly flexed. Pain is not a prominent feature; the functions of the joint are but little impaired, so that the patient may walk fairly well. On grasping the joint while it is flexed and extended by the patient the fringes may be felt moving under the fingers.

The patient may first apply for advice on account of the symptoms of loose body, viz., sudden severe pain with temporary fixation or locking of the joint, disappearing as suddenly as it came. The attack may recur at irregular intervals. If the loose body is attached, the pain is located to a particular area of the joint, if its pedicle has given way it may wander about the joint; in either case it may be identified by the patient, or on examination by the surgeon. The treatment applicable to this type is the removal of the hypertrophied fringes or of the loose body by open arthrotomy, and is usually very successful.

3. *The dry arthritis deformans* (arthritis sicca), although especially common in the knee, is met with frequently in all the large joints, either as a solitary or multiple disease, and it is also very common in the joints of the spine and of the fingers, and in the temporal maxillary joint. In the joints of the fingers in older patients the disease is remarkably symmetrical; it tends to assume the nodular type (Heberden's nodes), whereas in younger individuals it assumes the more crippling and painful and progressive fusiform type. In the larger joints, *e.g.* knee, hip, shoulder, the subjective symptoms usually precede any palpable evidences of disease. The patient complains of stiffness, cracklings, and aching, aggravated by changes in the weather and by rest. The roughness (fibrillation) of the articular cartilages may be appreciated by the coarse friction or rubbing, on movement of the joint. It may be many months or years before the lipping and other hypertrophic changes in the ends of the bones are recognisable, and before the joint assumes the deformed features which have given the disease its name. These are referred to under the individual joints.

The three types described may occur in combination.

As regards the progress of the disease, it is usually observed that in patients who are still young the tendency is to advance with considerable rapidity, so that in the course of a few months it may cause serious crippling of several of the joints. In older patients its progress is much more gradual and intermittent, and in them the disease is compatible with long life.

*Treatment*, in the absence of definite knowledge of the etiology of the disease, is chiefly directed towards the relief of symptoms. On no account should the affected joints be kept at rest. Passive movements, exercises of all kinds, massage, and douching are to be steadily persevered with. When pain is a prominent feature it may be relieved either by douches of iodine and hot water, or by application of lint saturated with chloral gr. v., glycerine ʒj, water ℥j, or with equal parts of menthol and parolein, and covered with oil-silk. Operative interference (arthrectomy, arthrodesis, excision) is indicated in the large joints of the limbs when the disease is of an aggravated type, is mono-articular, and the patient is neither old nor unhealthy.

A course of treatment at one of the reputed baths, *e.g.* Aix, Bath, Buxton, Gastein, Wiesbaden, Wildbad, is often of great service.

The patient should be well nourished. There should be no restriction, such as is required in gouty patients, so long as the digestion is not impaired. Benefit is also derived from the administration of cod-liver oil and of tonics.

*Arthritis ossificans* is the name applied by Griffiths of Cambridge to a condition in which the joints affected become obliterated as a result of fusion of the bones with one another. The cancellous tissue of the one becomes directly continuous with the other without any trace of separation across what was originally the joint cavity. The disease usually begins in the early years of adult life. It is more often met with in men. It is slow in its progress, inasmuch as years elapse before the joints become rigid. It is polyarticular, one joint being affected after another. It may involve all the joints of the body. Its origin is unknown.

### Hæmophylia—Hæmarthrosis—Bleeders' Joint

Although described in the article on "Hæmophylia," in vol. iv., it may be useful to refer to the clinical features of bleeders' joint so as to bring out the contrast between it and other diseases of joints. The subject is usually a boy or youth, who, without any definite injury, presents a rapid effusion into a joint, usually the knee. There is little pain. The temperature may be considerably elevated (102° F.). The patient frequently exhibits ecchymoses or swellings on other parts of the body, so that he should be completely stripped for purposes of examination. After a single hæmorrhage into the joint the blood is reabsorbed, especially under the influence of gentle massage and passive movements. After repeated attacks, however, secondary changes occur, associated with the persistence of blood-clot and its partial organisation, and the joint may become uniformly swollen and stiff, so that the resemblance to white swelling may be so close that a mistaken diagnosis has been made by experienced observers, and an operation has been performed which has cost the patient his life. The treatment consists in the maintenance of rest, the application of cold and of compressing bandages when the hæmorrhage is recent. After an interval the use of massage and of gentle passive movements promotes the absorption of the blood and hinders or prevents the occurrence of stiffness.

### IV. Joint Diseases associated with Lesions of the Nervous System—Neuro-Arthropathies — Spinal Arthropathies — Charcot's Disease.

In the absence of any proof of the existence of special trophic nerves distributed to joints, the diseases under consideration are to be regarded as related to a disturbance of the sensory nerves which pass from the joints to the spinal cord, whereby they are cut off from the reflex vasomotor influence which is necessary for their proper nutrition and for their capacities of recuperating from the effects of injury. The joints present a diminished resistance to trauma and other external influences very similar to that exhibited by the skin in its liability to pressure sores, perforating ulcer, and other trophic disturbances. It may be said, therefore, that while the nerve lesion prepares the way for the joint disease, its onset and progress are largely dependent upon external factors, of which trauma in its various forms is the most important. A patient whose knee-joint is anæsthetic and analgesic is not only more exposed to minor forms of injury, but he continues to use the joint, whereas under normal conditions he would place it under conditions favourable for repair.

(1) *In Lesions of the Peripheral Nerves.*—Affections have been observed in the joints of the hand, and more rarely in those of the foot, when one or other of the main nerve trunks has been divided or compressed. The affected joints become swollen and painful, and may afterwards become stiff and deformed. Bony ankylosis has been observed in exceptional cases.

(2) *In lesions of the spinal cord*, excepting locomotor ataxia and syringo-myelia, arthropathies are very rare indeed. In relation to stab-wounds and crushes of the cord their rarity is probably the result of the rest and immunity from injury of the paralysed limbs. Joint lesions are also very rare in cases of myelitis, progressive

muscular atrophy, infantile paralysis, insular sclerosis, etc.

*In locomotor ataxia* the occurrence of joint lesions was first described by Charcot, hence the popular term "Charcot's disease." They occur in from 5-10 per cent of the recorded cases. Although usually developing in the ataxic stage, one or more years after the initial spinal symptoms, they may appear before any other evidence of tabes. Their association with injury is generally accepted. The joints of the lower extremity are much more commonly affected, and the disease is bilateral in a considerable proportion of cases, *e.g.* both knees, both hips, etc.

The disease may assume *a mild form*, in which the joint and its vicinity become swollen, either spontaneously or after some extra exertion or slight injury. The swelling is chiefly due to fluid within the joint, and the latter cracks or grates on movement. The affection may disappear under rest, or persist, or relapse, or merge gradually into the more severe form.

In *the severe type* the onset of the disease may be extraordinarily rapid. Within a few days or weeks the entire joint may be disorganised. An atrophic and a hypertrophic type may be distinguished according to whether the wearing away and disappearance of bone, or the extravagant new formation of bone, is the more prominent feature. Sometimes, and especially in the knee, the clinical features are those of an enormous hydrops, with fibrinous and other loose bodies and hypertrophied fringes, like an exaggeration of that met with in arthritis deformans, only there is usually great œdema of the periarticular tissues, the joint is wobbly or flail-like from the stretching and destruction of the controlling ligaments, and there is no sensation in the joint. Sometimes, and especially in the shoulder, the wearing down and total disappearance of the ends of the bones is the prominent feature, this being also attended with flail-like movements and with coarse grating of the opposed surfaces. Dislocation is chiefly observed at the hip; it is rather a gross displacement, with exaggerated mobility, than an ordinary dislocation, for it is usually possible to draw the bones apart. An occasional and very striking feature is the extensive formation of new bone in the capsular ligament and surrounding muscles, resulting in the presence of large masses and plates which may add materially to the already existing deformity of the joint. In certain cases the enormous swelling of the joint and its rapid development may suggest the growth of a malignant tumour.

The most useful factor in diagnosis is the entire absence of pain, tenderness, and common sensibility. The freedom with which a tabetic patient will allow his disorganised joint to be handled, moved, and the bones grated on each other, requires to be seen to be appreciated.

*In syringo-myelia* ("gliomatous arthropathy") joint affections are more frequent (in 10 per cent of cases) than in tabes, and more often involve the upper extremities in correspondence with the seat of the lesion in the lower cervical and upper dorsal segments of the chord. Except that the joint disease is rarely symmetrical, it closely resembles the arthropathy of tabes. The complete analgesia of the joint structures and of the overlying soft parts, is well illustrated by cases in which the joint has been painlessly excised without an anæsthetic, and by one case in which the patient himself was in the habit of evacuating the fluid from his elbow by means of a pair of scissors. The painless whitlows of the fingers known as "Morvan's disease" are similarly the result of the analgesia, for the patient neglects breaches of the skin surface which allow of the entrance of pyogenic infection.

Suppuration, apart from superadded infection through a breach of the surface, does not occur in any of the forms of spinal arthropathy.

Spontaneous fracture may occur as a complication, both in tabes and in syringo-myelia.

The *prognosis* is uncertain as to progress, and is unfavourable as regards treatment, for in the majority of cases it is at the most capable of retarding or arresting the progress of the disease.

*Treatment* is usually directed towards supporting and protecting the joint by means of bandages, splints, and special apparatus. In the lower extremity the use of crutches may assist in taking the strain off the affected limb. When there is much distension of the joint, considerable relief may follow the evacuation of fluid. The best possible result being rigid ankylosis in a good position, it may be advisable to bring this about artificially by arthrodesis or excision where only one joint is affected, and where the cord lesion is such as will permit of the patient moving about. Although the victims of tabes are unfavourable subjects for operative interference on account of their liability to uncontrollable vomiting or diarrhœa, and to intercurrent complications, the wounds heal remarkably well. When the limb is quite useless, and there is danger from superadded septic infection, if one is to interfere at all, it should be by amputation.

(3) *In cerebral lesions* attended with hemiplegia (from hæmorrhage, tumour, etc.) joint lesions are occasionally met with in the paralysed limbs attended with evanescent pain, redness, and swelling. The secondary changes in joints which are the seat of paralytic contracture are considered elsewhere.

*An intermittent neuropathic hydrops* has been observed, especially in the knee, in cases of epilepsy, hysteria, general paralysis of the insane, etc., but it is of little clinical importance.

### V. Hysterical or Mimetic Joint Affections

Under this heading Sir Benjamin Brodie in 1822 described a rare affection of joints, characterised by the prominence of the subjective symptoms and the absence of any pathological changes in the joint. Although chiefly met with in young adult single women and widows, with impressionable nervous systems, and more often in those of good social circumstances, it occurs occasionally in robust women, and even in men. The onset may be referred to injury or exposure to cold, or it may be associated with some disturbance of the emotions or of the generative organs, or it may result from an involuntary imitation of the symptoms of organic joint disease presented by another patient.

It is characteristic that the features develop abruptly without sufficient cause, that they should be exaggerated and wanting in harmony with one another, and that they do not correspond with the typical features of any of the known forms of organic disease. In some cases the only complaint is of severe neuralgic pains; more often these are associated with excessive tenderness and with impairment of the functions of the joint. On examination, the joint presents a normal appearance, but the skin over it is remarkably sensitive: the slightest touch is more likely to excite pain than deeper and firmer pressure over those points which are usually tender in ordinary forms of organic joint disease. Stiffness is a variable feature: in some cases it may amount to absolute rigidity, so that no ordinary force will elicit movement at the joint. It is characteristic of this, as of other neuroses, that the symptoms come and go without apparent reason. When the patient's attention is diverted the pain and stiffness may disappear. There is never any actual swelling of the joint, although there may be an appearance of this from wasting of the muscles above and below. If the joint is kept rigid for long periods secondary contracture may occur, in the knee with flexion, in the hip with flexion and adduction. Attempts at movement may then cause cracking noises. Months or years may elapse without any further developments.

*The diagnosis* is often a matter of considerable difficulty, for there are organic lesions, *e.g.* a tuberculous focus in the bone close to a joint, which may cause vague neuralgic pains for months or years before rupturing into the articulation. Examination with the Röntgen rays, and of the joint under chloroform, may assist in difficult cases, but there are cases on record in which an experienced surgeon has been obliged to perform an exploratory operation in order to make a definite diagnosis. The greatest difficulty is met with in the knee, where the condition may closely resemble tuberculous disease.

*The treatment*, besides that directed to the constitution of the patient, chiefly consists in improving the nutrition of the affected limb by means of massage and baths, and electricity. Splints are to be avoided. In refractory cases considerable benefit may follow the application of Corrigan's button or the actual cautery. Complete recovery is the rule.

### VI. Tumours and Cysts

*Innocent tumours of the synovial membrane*, whether fatty, fibrous, or cartilaginous, are not recognised as distinct from the overgrowth of the corresponding tissues in certain chronic forms of joint disease, *e.g.* arthritis deformans.

*Sarcoma of the synovial membrane* has been chiefly met with in the knee, and has been nearly always mistaken for synovial tuberculosis. One case is recorded in which a localised sarcoma of the synovial membrane gave rise to the symptoms of loose body in the knee. The usual treatment has been to cut away the synovial membrane, and so far as the recorded cases go it has been quite successful. The spindle and round-celled sarcomata are much more malignant than the myeloid.

*Cysts of joints* constitute an ill-defined group. They include *ganglia* which form in relation to the capsular ligament, most commonly on the outer aspect of the knee-joint in the interval between the bones and in front of the tendon of the biceps (see "Knee-Joint, Diseases of"). *Cystic distention of the bursæ* which communicate with the joint is most often met with in relation to the knee in cases of long-standing hydrops. It has been maintained that similiar cystic swellings may result from the hernial protrusion of the synovial membrane between the stretched fibres of the capsular ligament, and the name "Baker's cysts" has been applied to them, after Morrant Baker who first described them.

In the majority of cases these cysts give rise to little inconvenience, and may be left alone. If interfered with at all, they should be excised.

### VII. Loose Bodies

While there is probably no more controversial subject in surgical pathology than the origin and nature of loose bodies, their clinical aspects and treatment are quite clear and straightforward. It is convenient to group them anatomically into two great classes: those composed of fibrin, and those composed of organised connective tissue.

I. Fibrinous Loose Bodies (*corpora oryzoidea*).

These are homogeneous or concentrically laminated masses of fibrin, sometimes quite irregular in shape, sometimes resembling rice grains, melon seeds, or adhesive wafers; usually present in large numbers, they are sometimes solitary and may then attain considerable dimensions. They are not peculiar to joints

for they are met with in tendon sheaths and bursæ; their origin from the synovial membrane may be accepted as proved. Their presence is almost invariably associated with chronic effusion from the synovial membrane (hydrops) in tuberculosis, arthritis deformans, or Charcot's disease. While they may result from the coagulation of fibrin-forming elements in the exudation, their occurrence in tuberculous hydrops would appear to be the result of coagulation necrosis or fibrinous degeneration of the surface layer of the diseased synovial membrane. However formed, their characteristic shape is the result of mechanical influences, and especially of the movements of the joint. *Clinically* they constitute an unimportant addition to the features of the disease with which they are associated; they never give rise to the classical symptoms of loose body; their presence may be recognised, especially in the knee, by the crepitating sensation imparted to the fingers when the bodies are moved to and fro in the fluid. The *treatment* is concerned with the disease underlying the hydrops; if it is desired, however, to empty the joint by means of a trocar and cannula, one must be prepared for the cannula becoming blocked with the bodies; should this occur, the alternative is to evacuate the fluid and bodies by means of a suitable incision.

Extravasation of blood into a fringe of the synovial membrane of the knee has been known to give rise to the symptoms of loose body; such a condition is quite capable of spontaneous recovery.

II. Bodies composed of Organised Connective Tissue, *e.g.* fatty, fibrous, cartilaginous, bony, or combinations of these, are met with under the following conditions:—

A. *In association with some general disease of the joint*; loose bodies composed of connective tissue or of its derivatives are comparatively common in arthritis deformans; they are also met with in certain rare forms of synovial tuberculosis and in Charcot's disease. They are derived almost exclusively from hypertrophic changes in the synovial fringes; they may consist of fat, *e.g.* the arborescent lipoma; more commonly the connective-tissue cells of the fringes proceed to form fibrous tissue, cartilage, and bone in varying proportions and combinations, after the manner commonly observed in innocent new growths. Like other hypertrophies on a free surface, they tend to become polypoidal and pedunculated, and exhibit a limited range of movement. The pedicle or stalk may, however, give way and the body becomes free; in this condition it may wander about the joint, or lie snugly in one of its recesses until disturbed by some exaggerated movement or twist; in the free state it is alleged to be capable of continued growth, deriving the necessary nutriment from the surrounding fluid. The number and size of the bodies vary indefinitely; they have been known to attain the size of the patella, and to number considerably over a hundred. A rarer type of loose body in arthritis deformans is met with when a portion of the "lipping" of one of the articular margins is detached by injury. It may also be mentioned that in Charcot's disease large loose bodies composed of bone may be formed in relation to the capsular and other ligaments, and may be made to grate upon one another.

In this group of organised loose bodies, the disease which underlies their formation is the predominating element in the clinical features and in the treatment. The characteristic symptoms of loose body (*vide infra*) are often absent; when present, they are to be regarded rather as a complication of the existing disease than as a separate entity.

B. *Loose bodies in joints which are otherwise healthy*; these constitute the majority of cases causing the classical symptoms of loose body, and the majority also of cases which call for operative treatment. Confining our statements to established facts, it may be said that they are chiefly met with in the knee and elbow of healthy males under the age of thirty. The complaint may be of vague pains in the joint (usually ascribed to rheumatism), of occasional cracking on movement, or of impairment of function, usually an inability to extend or flex the joint completely. In many cases a clear account is given of the characteristic symptoms which arise when the body is impacted between the articular or other closely applied surfaces of the joint, viz., sudden and intense pain, loss of power in the limb, and locking of the joint, followed by effusion and other accompaniments of a severe sprain. On some movement of the joint, the body is disengaged, the locking disappears, and recovery takes place as after an ordinary sprain or twist. These symptoms may continue, and the attacks of impaction may be repeated at irregular intervals during a period of many years. On examining the joint it may be found to contain fluid, and there may be points of special tenderness; the patient himself, or the surgeon, may succeed in palpating the loose body, and in making it roll beneath the fingers, especially if it be lodged in the suprapatellar pouch in the case of the knee, or on one or other side of the olecranon in the case of the elbow. In most instances the patient has carefully observed his own symptoms, and is aware, not only of the existence of the loose body, but of its situation when "attached," or of its erratic appearance at different parts of the joint when "free." When the body contains bone it may show in a skiagraph. While in some cases the patient attributes his symptoms to some definite injury (rightly or wrongly), exactly similar phenomena

may occur apart altogether from traumatic influences. The treatment consists in opening the joint and removing the body; the patient recovers with an absolutely healthy joint; if at the operation the opportunity is taken of inspecting the articulation, it is usually found to be normal, the important point being that there is no general disease such as attends the presence of loose bodies in the preceding groups.

*The characters of the loose bodies removed by operation*, as above described, are remarkably constant; they are usually solitary, about the size of a bean or almond, concavo-convex in shape, the convex aspect being smooth like an articular surface, the concave aspect more often uneven, nodulated, suggesting the healing over by fibrous or fibro-cartilaginous tissue, of an irregular fracture of spongy bone. Such bodies when still attached may be lodged in a kind of compartment, nest, or excavation, in one of the articular surfaces, usually one or other condyle of the femur, from which they may be readily shelled out by means of an elevator. They usually present on section a nucleus or core of spongy bone or calcified cartilage.

The origin of these loose bodies is too controversial to allow of its being discussed in the present article; some maintain their origin entirely from injury, others regard them as originally derived from the synovial membrane, while König regards them as portions of the articular surfaces which have been detached by a morbid process which he calls "osteochondritis dissecans." The subject of loose bodies in joints may be concluded by mentioning the traumatic *displacement or detachment of one or other of the semilunar cartilages* in the knee, which give rise to the characteristic symptoms of loose body, modified by the accurate localisation of the offending body and the conditions under which it is met with.

**Jores' Fluid.**—A preserving solution for tissues containing formalin, sodium sulphate and chloride, and magnesium sulphate. *See* Post-Mortem Methods (*Preservation of Tissues*).

**Jothion.** *See* Iothion.

**Joule's Law.** *See* Physiology, Food and Digestion (*Determination of Energy Value*).

**Jugular.**—Relating to the throat (Lat. *jugulum*, the throat), *e.g.* the jugular glands (*see* Lymphatic System) or the jugular veins (*see* Neck, Region of, *Wounds*).

**Juices.** — Expressed or secreted fluids obtained from animal or vegetable tissues, *e.g.* the gastric or the pancreatic juice (*see* Physiology, Food and Digestion); in pharmacy there are six official *Succi* or *Juices*, five of which (the Succi of Belladonna, Hyoscyamus, Conium, Broom, and Taraxacum) contain alcohol while one (Succus Limonis) does not. *See* under the various drugs.

**Jujubes.**—Lozenges of gelatin or gum-arabic flavoured with the jujube fruit (*Zizyphus*) or supposed to be so; used as demulcents in cases of pharyngitis, etc.

**Julep.**—A drink, sweetened with syrup or sugar, used to disguise the unpleasant taste of some drugs; a medicated drink (the Persian word *gullab* means rose-water).

**Jumpers.**—A variety of saltatory spasm or myriachit, occurring sometimes in epidemic form (*e.g.* the "jumping Frenchmen" of Maine and New Hampshire); there is a jumping movement which takes place only when the patient tries to stand.

**June Cold.**—Hay fever, or rose-cold, or catarrhus æstivus. *See* Hay Fever.

**Juniper.** *See also* Dermatitis Traumatica et Venenata (*Causal Agents, Juniperus sabina*). The official oil of juniper (*Oleum Juniperi*) is obtained from the *Juniperus communis*; it has a characteristic odour and contains *pinene* ($C_{10}H_{16}$), *cadinene* ($C_{15}H_{24}$), and a crystalline substance called *juniper camphor*; it is given internally in doses of ½ to 3 m. for the same purposes as oil of turpentine, but chiefly as a diuretic in chronic Bright's disease, ascites (due to liver disease), and in heart disease. There is a preparation, *Spiritus Juniperi*, of which the dose is 20 to 60 m.; it gives a violet odour to the urine. It is a flavouring ingredient in *Gin* or *Hollands* (*see* Alcohol).

**Junker's Apparatus.** *See* Anæsthesia, General Physiology (*Regulating Chloroform Inhaler*).

**Jurisprudence, Medical.** *See* Medicine, Forensic.

**Jury Mast.**—An apparatus for the purpose of keeping the cervical or upper dorsal portion of the spinal column at rest, used in cases of Pott's disease of the spine. *See* Spine, Surgical Affections (*Spinal Caries, Treatment*).

**Justo-Major Pelvis.** — A pelvis equally, symmetrically, or proportionately enlarged in all its diameters; a giant pelvis; pelvis æquabiliter justo major. *See* Labour, Precipitate.

**Justo-Minor Pelvis.** — A pelvis equally, symmetrically, or proportionately diminished in all its diameters; a generally contracted or dwarf pelvis; pelvis æquabiliter justo minor. *See* Labour, Prolonged (*Pelvic Deformities*).

**Juvenile General Paralysis.** *See* Mental Deficiency (*Developmental Cases, Syphilitic*).

**Kahler's Disease.**—Myelopathic albumosuria; there is persistent excretion of Bence-Jones' proteid in the urine, along with the presence of multiple myelomata.

**Kairine.** — An alkaloidal derivative of quinoline, somewhat resembling quinine; another artificially prepared alkaloid is *Kairoline*.

**Kakke.** *See* BERIBERI.

**Kala-azar.**—Dum-dum fever, piroplasmosis, or tropical splenomegaly. *See* MALARIA (*Sequelæ, Malarial Cachexia*); TROPICS, UNCLASSED FEVERS OF (*Fevers of Mixed Origin, Kala-azar*).

**Kaladana.**—Pharbitis Nil or the dried seeds of *Ipomœa hederacea*, official in the Indian and Colonial Addendum to the British Pharmacopœia of 1898; its dose is from 30 to 50 grains in powder or in the form of *Pulvis Kaladanæ Compositus* (dose, 20 to 60 grs.) or of *Tinctura Kaladanæ* (dose, ½ to 1 fl. dr.); *Kaladanæ Resina* or Pharbitisin has a dose of 2 to 8 grains; Kaladana and Kaladanæ Resina have the same action, and are used for the same purposes as jalap and jalapæ resina (*Ipomœa purga*). *See* IPOMŒA; JALAP; etc.

**Kalium.** *See* POTASH; IODINE AND IODIDES; etc.

**Kalmuck Idiocy.**—A form of idiocy with facial appearances resembling those seen in Mongolian idiocy. *See* MENTAL DEFICIENCY (*Congenital, Mongoloid*).

**Kalomelol.**—Colloidal calomel of Merck, a form of calomel soluble in water, given internally (dose, ⅙ gr.) or externally (as dusting powder or ointment) in the treatment of syphilis.

**Kamala.**—An anthelmintic powder, containing a crystalline resin (rottlerin, $C_{33}H_{30}O_9$), obtained from the fruit capsules of *Rottlera tinctoria*; official in the United States and in Germany.

**Kamloops.** *See* THERAPEUTICS, HEALTH RESORTS (*America, British Columbia*).

**Kangaroo Tendon.** *See* ASEPTIC TREATMENT OF WOUNDS (*Disinfection of ligatures*).

**Kaolinum.**—A native aluminium silicate which in the form of a powder is used as a dusting powder and as a basis for pills, containing potassium permanganate, phosphorus, etc.

**Kaposi's Disease.**—Xeroderma pigmentosum. *See* SKIN, PIGMENTARY AFFECTIONS OF (*Xeroderma pigmentosum*).

**Kaputine.**—"Kaputine" powders are said to consist mainly of antifebrin or acetanilide. *See* MORPHINOMANIA AND ALLIED DRUG HABITS (*Antifebrin*).

**Kariyat.**—A synonym of Indian Chiretta. *See* ANDROGRAPHIS.

**Karlsbad.** *See* BALNEOLOGY (*Austria*).

**Karyochrome Cells.**—Nerve cells of the cortex cerebri which do not contain chromophile substance in their protoplasm; they are smaller than the cells which contain it, and which are known as *somatochrome*. *See* INSANITY, PATHOLOGY OF (*Pathological Anatomy, Cortical Nerve Cells*).

**Karyokinesis and Karyomitosis.** *See* CARYOCINESIS.

**Katabolism.** *See* CATABOLISM.

**Katadicrotism.**—"The occurrence of dicrotism in the downward stroke of a sphygmographic tracing"; dicrotism, as opposed to anacrotism.

**Kataphoresis.**—The effect produced on tissues, *e.g.* cancers, by electricity, the positive pole being coated with mercury, which forms an oxychloride of mercury, supposed to act lethally on the cancer cells but not on the normal ones.

**Katatonia.**—A form of insanity in which there are melancholic and cataleptic phenomena and morbid somnolence. *See* SLEEP, NORMAL AND MORBID (*Morbid Somnolence*).

**Katharsion.** *See* BALNEOLOGY (*Greece, Lesbos*).

**Kauri.**—A coniferous tree of New Zealand from which Kauri-gum (a resin) is obtained, which is used as a disinfectant and as a substitute for collodion.

**Kava.**—*Kavæ rhizoma* is the dried rhizome of *piper methysticum*; it is officially used in Australia, and is included in the Indian and Colonial Addendum to the British Pharmacopœia of 1898. The *extractum kavæ liquidum* is given in doses of ½ to 1 fl. dr.; if injected hypodermically the liquid extract produces local anæsthesia (due to the resin, *Kawine*). A liquid made from the root ("yangona root") by the natives of the Sandwich Islands, and called "Kava" or "Ava," produces intoxication of a drowsy character. The liquid extract of Kava is used as a diuretic, and in gonorrhœa and leucorrhœa.

**Kawine.** *See* KAVA.

**Kedgeree.** *See* INVALID FEEDING (*Fish*).

**Kefir.** *See* KEPHIR.

**Kehrer's Operation.**—An operation for the relief of depressed or invaginated nipples; mammillaplasty. *See* PUERPERIUM, PATHOLOGY (*Nipples*).

**Kelene.** *See* ANÆSTHETICS, ETHYL CHLORIDE.

**Keloid.** *See* CICATRICES (*Keloid or Cheloid*); MORPHŒA; SCLERODERMIA (*Circumscribed, Keloid of Addison*); SKIN DISEASES OF THE TROPICS (*Leucodermia and Keloid*); TUMOURS OF THE SKIN (*Keloid*).

**Kemmern.** *See* BALNEOLOGY (*Russia*).

**Kentucky.** *See* BALNEOLOGY (*America, Blue Lick and Paroquet Springs*).

**Kephir.**—A product obtained from the milk of the cow by combined vinous and lactic fermentation; it seems to have been first used as a nutrient drink in the Caucasus, and the ferment which is believed to be the causal factor is named *bacillus caucasicus*. *See* DIET (*Milk and its Products*); MILK (*Therapeutic Uses, Kephir*). Kephir has recently been employed in tuberculosis, in rickets, and in infectious fevers (*e.g.* scarlet).

**Keratectasia.** — Protrusion of the cornea. *See* CORNEA.

**Keratectomy.**—Excision of a piece of the cornea, or incision into the anterior chamber of the eye through the cornea.

**Keratin.**—A substance, closely resembling a proteid, of which the superficial squamous epithelial cells of the skin are composed; it also forms the nails, hair, horns, and hoofs; it contains sulphur, and can therefore be stained by lead, etc. (*see* PHYSIOLOGY, TISSUES, *Epithelium*). Although keratin has generally been used in medicine only as coating for pills to prevent them being acted on by the gastric juice, it is now claimed for it that it has medicinal properties; and it has been used in such interstitial affections as tabes dorsalis and chronic myelitis, where it is supposed to do good by inhibiting the formation of connective tissue and facilitating its resorption (by uniting with glutin). *See also* SKIN, ANATOMY AND PHYSIOLOGY (*Epidermis, Stratum Corneum*); SKIN, PIGMENTARY AFFECTIONS OF (*Pigments, Keratin*).

**Keratinisation.**—The change of the epidermal cells into a horny substance, or the coating of pills with keratin. *See* CICATRICES (*Pathological Conditions, Weak Cicatrices*).

**Keratitis.**—Inflammation of the cornea. *See* CORNEA (*Inflammation*); *see also* CONJUNCTIVA, DISEASES OF (*Chronic Ophthalmia, Complications*); IRIS AND CILIARY BODY (*Iritis and Cyclitis, Complications*); NERVES, NEURALGIA (*Operations on Gasserian Ganglion, Sequelæ*); SYMPATHETIC OPHTHALMITIS (*Keratitis punctata*); SYPHILIS, TERTIARY (*Eye and its Appendages, Cornea*); SYPHILIS IN CHILDREN (*Hereditary, Later Signs*); TYPHOID FEVER (*Complications and Sequelæ*).

**Keratocele.**—Hernia of the membrane of Descemet through a corneal ulcer. *See* CORNEA.

**Keratoconus.** — Conical cornea. *See* CORNEA.

**Keratodermia.** *See* ICHTHYOSIS (*Local Hyperkeratosis, Keratodermia erythematosa symmetrica*).

**Keratoglobus.**—Spherical bulging of the cornea of the eye (usually affecting also the sclerotic); hydrophthalmus.

**Keratohyalin.** *See* ELEIDIN.

**Keratolysis.**—A state of morbid loosening of the horny layers of the epidermis, as seen in pityriasis, in dermatitis exfoliativa neonatorum, and in fœtal keratolysis (*Ballantyne*). *See* NEW-BORN INFANT (*Ritter's Disease*).

**Keratoma.**—A morbid state of the skin, often congenital, characterised by excessive growth of the stratum corneum; fœtal ichthyosis. When it is limited to the palms of the hands and soles of the feet, it is called *Keratoma Palmare et plantare hereditarium*. *See also* SCLERODERMIA.

**Keratomalacia.**—A morbid state of the cornea characterised by softening and degeneration. *See* CORNEA (*Keratomalacia or Infantile Ulceration of the Cornea*).

**Keratonosus.**—A disease of the stratum corneum of the skin and of the nails and hair.

**Keratonyxis.**—The breaking up of a soft cataract by a needle passed in through the cornea.

**Keratorrhexis.**—Rupture of the cornea.

**Keratoscopy.** *See* RETINOSCOPY.

**Keratosis.** — A diseased state of the stratum corneum of the skin and of other horny structures; thus *hyperkeratosis* is an excessive development of the stratum corneum, as is seen most markedly in fœtal ichthyosis, and, locally, in tyloma and clavus; *hypokeratosis*, again, is defective formation of the horny layer; *keratosis follicularis* is a variety of ichthyosis. *See* ICHTHYOSIS; PHARYNX, EXAMINATION OF (*Pharyngomycosis or Keratosis Pharyngis*).

**Kerion.**—Inflammation of the tissues of the scalp, characterised by pustular folliculitis, and due to *trichopyton megalosporon ectothrix*, a form of ringworm. *See* SKIN, PARASITES (*Tinea Tonsurans*).

**Kernig's Sign.**—In cases of meningitis, if the patient sits on the edge of the bed with his legs hanging down, the lower leg cannot be completely straightened owing to rigidity of the

flexors of the knee; in other words, when the thigh is flexed on the abdomen the leg cannot be extended on the thigh. *See* MENINGES OF THE CEREBRUM (*Acute Simple Meningitis, Symptoms*).

**Ketones.**—Oxidation products obtained from the secondary alcohols (*e.g.* secondary propyl alcohol), to which they therefore stand in the same relationship as the aldehydes to the primary alcohols. *See* PHYSIOLOGY (*Organic Chemistry*).

**Khosam.**—The kernel of a small fruit found in China, the Straits Settlements, and Australia, identical with the *Brucea Sumatrana* of Borneo. The active principle seems to be a glucoside (*Khosamine*) which acts as a cathartic and cholagogue; it has been used in dysentery (dose, 10 grs. of the powdered seeds) and other maladies as a hæmostatic.

**Kidinga Pepo.** *See* DENGUE.

## Kidney.

PHYSIOLOGY OF . . . . . 89
SURGICAL AFFECTIONS OF . . . . . 93
MORBID AFFECTIONS OF. *See* articles "Nephritis," "Uræmia," "Urine," etc.

*See also* ABDOMINAL TUMOURS, DIAGNOSIS OF (*Kidney*); ADRENAL GLANDS (*Malignant Tumours, Diagnosis*); ALCOHOL (*Morbid Changes in Chronic Alcoholism, Kidney*); ANÆMIA, PERNICIOUS (*Morbid Anatomy, Kidneys*); ANÆSTHESIA, GENERAL PHYSIOLOGY OF (*Effect on Kidneys*); BED-SORES (*Causes, Kidney Affections*); BLACKWATER FEVER (*Morbid Anatomy*); BLADDER, INJURIES AND DISEASES OF; COLIC (*Diagnosis, Renal Colic*); CYSTOSCOPE; ECLAMPSIA (*Pathological Anatomy, Ætiology*); EMBRYOLOGY; FŒTUS AND OVUM, DEVELOPMENT OF (*Fœtal Kidneys*); HÆMATURIA; HEART, AFFECTIONS OF MYOCARDIUM AND ENDOCARDIUM (*Effects of Cardiac Disease, Cyanotic Kidneys and Renal Infarcts*); HYDATID DISEASE (*Echinococci of the Urinary System*); LARDACEOUS DEGENERATION (*Morbid Anatomy, Amyloid Kidney*); LEUCOCYTHÆMIA (*Morbid Anatomy, Kidney*); LIVER, DISEASES OF (*Constricted Lobe, Diagnosis*); LUNG, TUBERCULOSIS OF (*Complications, Uro-Genital*); MEDICINE, FORENSIC (*Rupture of Kidney*); NEPHRITIS; PANCREAS, DISEASES OF (*Pancreatitis, Diagnosis*); PHYSIOLOGY, EXCRETION (*Secretion of Urine*); POST-MORTEM METHODS (*Kidneys, Examination*); PREGNANCY, INTRA-UTERINE DISEASES (*General Fœtal Dropsy*); PYURIA; SPLEEN, SURGERY OF (*Wandering Spleen, Diagnosis*); STOMACH AND DUODENUM, DISEASES (*Secondary to other Diseases*); SYPHILIS (*Visceral*); THERAPEUTICS, HEALTH RESORTS; TRADES, DANGEROUS (*Lead-Poisoning*); VISCERAL PAIN.

### Physiology of the Kidney

*STRUCTURE.*—The kidney possesses two capsules: externally, a loose covering containing a large number of fat cells embedded in loose connective tissue, and within this a strong tunica fibrosa, composed of ordinary white and yellow elastic fibres, which forms a close covering for the organ, turning in at the hilum where it is continuous with the sheaths of the vessels. The latter covering can be comparatively easily and completely peeled off. Underneath this and lying on the surface of the kidney there is a network of smooth muscle fibres. If a longitudinal section be made through the organ from the outer border to the hilum, the glandular tissue can be easily recognised as consisting of two parts, cortical and medullary, the former reddish brown in colour, covering the latter and sending prolongations between the bases of the pyramids (Bertin's columns), the latter presenting a paler striated appearance, and arranged in the form of pyramids with the apices of the latter towards the hilum. Each pyramid is seen to be made up of a papillary part with indefinite striations and a boundary zone where the striæ are well marked. Each papilla projects into a short tube, the calyx; and these, of which there are eight to ten, unite to form two or three infundibula, and these finally form the pelvis of the ureter. As the striations pass out towards the bases of the pyramids they become more widely separated by blood-vessels, and are continued outwards as the pyramids of Ferrein almost but not quite to the surface of the cortex.

*Course of the Urinary Tubules.*—The convoluted tubules constitute the principal part of the cortex, in most animals forming about $\frac{1}{4}$ to $\frac{1}{3}$ of the length of the entire urinary tube. They constitute the true secreting as opposed to the collecting part of the glandular tissue, and arise from Bowman's capsule which surrounds the glomerulus. There is a short and narrow constriction where the capsule passes into the proximal tubule, the direction of the latter being at first towards the surface of the organ, so that, as one may see on microscopic examination, the superficial zone is free from glomeruli. The tube becomes very convoluted and gradually passes downwards towards the medulla. Some distance before the medulla is reached the tube becomes straighter, although still maintaining a spiral course, and when the limiting layer of the pyramid is reached it narrows down to a thin tube which pursues a straight course, in some cases almost completely down the papilla, usually, however, bending round either in the deeper parts of the marginal layer or in the superficial papillary region. It then proceeds upwards parallel to the descending limb as the ascending limb of Henle's loop. This limb widens out either at the bend or shortly before or after the loop, attaining about double the diameter of the descending limb, and after a more or less irregular course passes into the distal convoluted tubule, which is much shorter

than the proximal one. This communicates with a collecting tube through a short connecting branch, and the collecting tubes unite to form larger ducts, papillary ducts, which open by means of pores on the apices of the papillæ.

*Microscopic Structure of the Tubules, etc.*—Bowman's capsule is composed of a structureless membrana propria with an inner lining of flattened epithelial cells. The membrana passes almost entirely down the urinary tube, thinning towards the end and disappearing when the principal branches of the papillary ducts are reached. The epithelium maintains its squamous character as far as the neck of the tubule, becoming then higher and presenting a very distinctive appearance in the convoluted tubules. The cells lining these tubules show no definite cell outlines, but present a peculiar granular appearance in the outer basal part where the granules are arranged in linear fashion as rods, these being limited to this part and not passing into the cytoplasm of the inner portion of the cell. If the tissue be well fixed another peculiar feature of these cells may be made out, namely, a striated cuticular hem (*Bürstenbesatz*) about 2 to 3 $\mu$ in thickness, the striæ of which are exceedingly fine, certainly not more than ·5 $\mu$ in thickness, and are affirmed by some authors to possess vibratile movements; but this is more than doubtful. There are no secretory capillaries, and probably no intercellular bridges between these cells. A clearer epithelium lines the descending limb of Henle, the cells of which are flattened and contain nuclei which project into the lumen of the tube, giving the latter a wavy appearance. These tubes might be confused with capillaries if it were not for the characteristic nuclei, the thick membrana propria, and the absence of blood corpuscles. The epithelium which lines the thick limb of Henle's looped tubule, the spiral and the distal convoluted tubule, is similar to that lining the first part of the convoluted tube, only the epithelium is not so high, the rods are not so long, and the lumen appears wider. As the tube passes into the connecting and then into the collecting ducts the protoplasm of the cells becomes much clearer, staining more faintly with eosin, while in the papillary ducts the epithelium becomes more cylindrical and very clear.

*Structure and Arrangement of the Vascular Parts.*—The Malpighian bodies consist of two parts: (1) a capsule which, with the exception of the place of entrance and exit of the afferent and efferent vessels, completely invests a tuft of capillaries; (2) the glomerulus, a space being left between the capsule and the capillaries which communicates with the lumen of the convoluted tubule. A small artery, vas afferens, immediately after its entrance through the capsule breaks up into a bundle of fine capillaries, from which the blood is removed by an efferent arteriole with non-striped muscle in its wall, the vas efferens, a vessel of smaller calibre than the afferent one. This efferent vessel in its turn breaks up into capillaries which surround the convoluted tubules. The glomerular capillaries contain no muscle fibre, and so far as can be made out by the silver method, show no cell outlines such as would appear if they possessed an endothelial coat, while the afferent and efferent vessels and the capillaries around the convoluted tubules undoubtedly possess one. The walls of the glomerular capillaries seem to consist of a protoplasmic mass containing no definite cells, while covering the glomerulus and passing into the hollows on the tuft there is what Kölliker terms a syncytium, containing numerous nuclei, but showing no distinct cell outlines, in this respect differing markedly from the covering of the embryonic glomeruli. The glomeruli vary in size, and some have distinguished a large from a small variety. Where the vessels penetrate the capsule there is direct continuity between the latter and the syncytium, or, in the case of embryonic Malpighian bodies, the cylindrical cells covering the glomerulus. It is not necessary in this short article to describe the development of the kidney, but it is advisable to refer shortly to the two important views that are at present held by scientists on the mode of development of the glandular part, and it is perhaps best to refer to it in this place after the glomerular structure has been discussed. The most widely-held theory is that associated with the names of Toldt, Kölliker, and Golgi, who supported the view that all the renal tubules are developed as outgrowths from the ureter, which pass out as solid cones of cells, forming ampullæ which divide into two coiled branches under the capsule or close to the interlobular septa. These columns of cells become hollowed out, and capillaries, developing locally or penetrating as branches of the renal artery, go to form the glomerulus in the lower curve of the ampulla branches which run an S-shaped course. The other view, and one which is gradually becoming more widely accepted, is that the collecting tubules are derived from ureter outgrowths, but that the Malpighian bodies, the convoluted tubules, and Henle's loop are developed from a solid clump of mesoblastic cells at the periphery of a lobule lying in close relationship to the termination of the dilated extremity of a collecting tube. This mass of cell becomes hollowed out and coiled in the form of an S, its lower limb going to form a Malpighian body, the glomerular capillaries developing *in situ*, the rest of the coiled S-tube going to form convoluted tubules, Henle's loop—part of the junctional tubule probably. The junction of the collecting with the convoluted tubule takes place at the periphery of the lobule. This view brought

forward by Thayssen is supported by the work of Bornhaupt, Hamburger, and Herring.

The afferent vessels of the glomeruli are derived from interlobular arteries which pass out through the cortex after arising from the renal arterial arches which radiate out between the cortex and medulla. The interlobular arteries also send branches to the capsule, and from their lower part near their origin from the arch a few straight branches to the medulla—*arteriolæ rectæ veræ*. The efferent arterioles from glomeruli lying close to the Malpighian pyramids divide up to a slight degree, and form false straight vessels in the medulla—*arteriolæ rectæ spuriæ*. The true straight vessels arise not only in the way above mentioned, but also in part from the concavity of the arches and from adjacent afferent glomerular arterioles. The veins arise from the capillary sources which have been described, and the blood is returned by the venous arches to the renal vein, no valves being present in their course. It is important to remember that the renal capsules have a blood-supply from many sources, *e.g.* the renal artery before its entrance into the hilum, suprarenal, and lumbar branches and interlobular arteries, and that the veins communicate with those of the neighbouring organs, and partly also with the portal system (Tuffiers and Lejars).

*Lymphatics.*—Between the blood-vessels and the convoluted tubules there are freely anastomosing lymph spaces, while in the medulla and the medullary rays they are poorly represented. The capsules have a rich lymph-supply.

*Nerves* are derived from the cœliac plexus of the sympathetic, and accompany the vessels at their entrance, some fibres forming a close network around the blood-vessels. The modes of termination of these nerves have been the subject of much discussion. Vaso-motor fibres have been traced to the adventitia, and muscular coats of vessels and other fibres to Bowman's capsule, but a connection with the convoluted tubules has not yet been made out with certainty.

The *connective tissue* is sparse in amount, being richest relatively in the pyramids, especially in the papillary zone.

*Mechanism of Renal Secretion.*—A fairly complete account has been given of the structure of the normal kidney, because it is impossible to speak about the probable functions of an organ without an exact knowledge of its structure. Of course one must avoid drawing conclusions as to function from structural arrangements alone as experimental; pathological and clinical evidence must all be brought to bear on the subject. Still, and this is especially true of the kidney, the very strongest proofs as to the functions of an organ are often derived from a careful preliminary study of its structural relationships. The observer is struck by the marvellous arrangement and character of the blood-vessels and urinary tubules, leading one immediately to surmise that the vascular arrangements in and around the glomerulus must play an important part in the mechanism of renal secretion. The afferent vessel breaks up into a number of fine divisions which offer a large surface for filtration, while the narrowness of the efferent arteriole and the introduction of another set of capillaries all offer high resistance to the blood-flow, so that the blood flowing through the glomerular capillaries must be under comparatively high pressure. Again, the fact that the urinary tubule is directly continuous with the space between Bowman's capsule and the tuft of capillaries is strong proof that the channel is one for collecting material that has been obtained from the capillary blood, the pressure in the circulating blood being considerable, while that in the secreting tubules, if there be no hindrance to outflow of urine, is inappreciable. The purely mechanical explanation that Ludwig gave of urinary secretion is supported by many well-established data. Ludwig's theory is that a very dilute urine, containing all the constituents, filters through the glomerular capillaries under the influence of blood-pressure, and that the urine subsequently becomes more concentrated on its downward course through the convoluted tubules by diffusion of water from urine to blood. If this view were correct, then every rise or fall in pressure in the glomerular capillaries ought to be succeeded by a corresponding increase or decrease in the amount of urine, and in many cases this is true. If the local blood-pressure fall, as, for example, after stimulation of the renal nerves or mechanical obstruction to the flow of blood through the renal artery, the amount of water secreted becomes correspondingly diminished, while a rise in local pressure, such as would result from section of the renal nerves, especially if after this the spinal cord or the splanchnics be stimulated, or if large blood-vessels elsewhere be ligatured, is followed by an increased flow of urine. There are certain facts, however, which make it difficult to accept this view in its entirety, and one is that any obstruction to venous outflow diminishes the amount secreted. This in itself is no argument against the process being one of filtration, because in such an organ as the kidney venous congestion results in arterial anæmia; but if in such a case of venous congestion with diminished excretion, nitrates of the alkalies be transfused through the blood, then immediately there is a great increase in the flow of urine. Such diuretics may act even when secretion has stopped, and the results cannot be explained satisfactorily by action on a peripheral vaso-motor mechanism. An hydræmic plethora is very short-lived after injection of these sub-

stances, the composition of the blood remaining very constant. The action of caffein as a diuretic has been carefully investigated with the oncograph, and it has been shown that secretion varies directly with the shrinkage or expansion of the kidney. It is difficult, on the filtration hypothesis, to explain why two crystalloids like urea and glucose, although their percentages in the blood are approximately the same, should pass into the urine in such different proportions. Again, as Max Hermann pointed out, if the renal artery be occluded for 1½ minutes only, the secretion of urine will not begin again until three-quarters of an hour have elapsed. It must be remembered also that the water has not only to pass through the capillary walls, but also through the glomerular covering, be that epithelial or syncytial, and that must offer a great resistance to filtration (*cp.* Leber's experiments on Descemet's membrane in the cornea).

There can be no doubt that venous congestion may cause obstruction to urinary outflow, either directly, or as a result of œdema by compression of the collecting tubules in the medullary rays, as Ludwig pointed out, and also that hindrance to urinary excretion may cause venous congestion.

But if there are difficulties in the case of a physical explanation of the glomerular functions, they are but insignificant in importance compared to those which immediately present themselves when an attempt is made to prove the accuracy of the second part of Ludwig's hypothesis, namely, the concentration of the dilute urine in the convoluted tubules. Long ago Hoppe-Seyler pointed out that water passed from blood serum to urine across an animal membrane, and more recently it has been shown that normally the osmotic pressure of the urine is decidedly greater than that of defibrinated blood or serum. These experiments proved that Ludwig's theory in its original form was untenable, for in order that the process of concentration might be carried on, work must be performed by the cells of the convoluted tubules, that is to say, the process must be regarded as one of active secretion. Von Sobieranski adopted Ludwig's views in the main, but regarded the cells of the convoluted tubules as actively absorbing water. His conclusions were based upon certain experiments dealing with the course of coloured substances through the kidney after injection. He noticed that pigment granules tended to be deposited in the part of the cell next the lumen, and not in the basal part, as one would expect, if the cells of the convoluted tubules took them up from the blood. Munk and Senator hold with the first part of Ludwig's theory, but accept Heidenhain's view of the specific secreting properties of the cells of the convoluted tubules and ascending limb of Henle's loop.

*The Specific Secretory Hypothesis* was put forward by Bowman in the first place, but he based it largely on his interpretation of the arrangement of glomeruli, tubules, and blood-vessels, rather than on any experimental evidence. It is to Heidenhain that we are indebted for most of the experimental proofs that render the theory a feasible one. As a result of experiments carried out by himself and others, for the purely physical hypothesis he was led to substitute one in which the specific activity of gland cells played the important rôle. In his classical article on renal secretion in Hermann's *Handbuch* he sums up his views under five brief headings:—

1. As in all other glands, secretion in the kidneys depends upon the active participation of special secretory cells.

2. In the first place, the cells covering the glomerulus secrete water and the salts which as a rule accompany it, *e.g.* common salt.

3. The other set of cells, lining the convoluted tubules and the broad part of the ascending limb of Henle's loop, serves for the secretion of the special urinary solids, urea, uric acid, etc., and also a certain quantity of water.

4. The degree of activity of the two types of cells depends upon

(*a*) Percentage of water and urinary solids in the blood.

(*b*) Rapidity of blood-flow in renal capillaries so far as that affects food-supply for the special cells.

5. The great variability in the composition of the urine is explained by variations in the secretory activity of the two types of cells.

Thus, according to Heidenhain, the water of the urine is most largely obtained from the glomerular capillaries, and he only differs from Ludwig in emphasising the importance of the rate of blood-flow rather than alterations in the pressure. Nussbaum's experiments on exclusion of the glomeruli of the amphibian kidney by tying the renal artery and studying the effects before and after injection of urea, sugar, egg, albumin, and peptone, are not conclusive, because it has been shown that it is impossible to cut off the glomerular blood-supply by tying the renal artery, seeing that there are anastomoses between branches of the renal portal (which was supposed to supply the convoluted tubules alone) and the renal artery.

It is also unnecessary to describe fully Heidenhain's experiments on the injection of indigo-carmine, because the results that he obtained have been shown to be by no means so definite as was at first imagined. Even if it were proved beyond doubt that indigo-carmine was secreted by the rodded cells of the convoluted tubules and the broad part of the ascending limbs of Henle's loop, and that the glomerular secretion merely washed it down into the collecting tubules, it by no means

follows that urea, uric acid, etc. pursue the same course. It is unfortunate that urea, owing to its great solubility, is so quickly excreted that its detection in the tubules is impossible, and uric acid also is excreted without, as a rule, leaving any trace of its passage through the rodded cells. Recently, however, crystals of uric acid have been detected in those cells by Minkowski after adenin has been given. They are often found in the lumen of the convoluted tubules, never in Bowman's capsule. Hæmoglobin seems without doubt to pass through the glomeruli (Adami), and in all probability serum albumin also.

Ribbert and Bradford's experiments on the effects of removal of larger or smaller portions of the kidney substance may be interpreted in so many ways that their discussion in this article is unnecessary. Although most of the important work on renal secretion was done by older investigators, recent workers have been able—thanks to the ingenious invention of Roy—by means of the oncograph, to register alterations in the volume of the kidney. This instrument is simply a plethysmograph of a suitable shape for the kidney, with oil as the medium, and a recording piston with attached lever for recording changes in the volume of the organ.

*Influence of the Nervous System.*—Reference has already been made to the influence exerted by the vaso-motor nerves, the fibres of which leave the cord by the anterior roots of the 11th, 12th, and 13th dorsal nerves (Bradford). According to Bradford, vaso-pilator fibres accompany the constrictors, as stimulation of the above-mentioned anterior roots by induction shocks at the rate of one per second produces active dilatation of the vessels without a sufficient rise in blood-pressure to explain the enlargement of the kidney. Stimulation of the posterior roots produces reflexly a similar dilatation. There has always, however, been doubt expressed as to the existence of special secretory nerves to the kidney, although Eckhard showed that polyuria might be produced by mechanical stimulation of that portion of the superior vermes of the cerebellum adjacent to the medulla if the hepatic nerves had been previously cut, or if the stimulation were a superficial one. He could not obtain the results which C. Bernard did on puncture of the floor of the 4th ventricle. It is hardly necessary, however, to accept the view of special secretory nerves for the kidney, as the existence of vaso-dilator fibres which can be stimulated either directly or reflexly explains the results obtained by Eckhard, and also the polyuria that occurs in hysteria, epilepsy, etc. The histological work of Berkeley on the nerve-endings in the kidney requires further confirmation.

The subject of physiological albuminuria is a difficult one. It is perhaps most easily explained on Heidenhain's hypothesis, the permeability or secretory activity of the cells being affected by any cause which tends to produce slowing of the local circulation, and as a result disturbance of the intracellular metabolism from want of oxygen, etc. (See article "Urine.")

It is impossible at present to speak with any certainty about the internal secretion of the kidney, although a vast amount of work has been done by French and Italian scientists on this subject.

*Specific Renal-Functions.*—Although in this article special reference has been made to the kidney as an organ of excretion, at the most selecting from the plasma preformed urinary constituents, it is necessary also to bear in mind that it possesses the power of altering plasma constituents, and in some cases even carrying out important syntheses. Thus the alkaline phosphates of the plasma appear in the urine as acid ones, and in at least some animals (*e.g.* the dog) the synthesis of benzoic acid and glycocoll to form hippuric acid takes place in its tissues. Although some at present hold that the kidney is the seat of uric acid formation, the proofs that have been brought forward in support of the view do not warrant one in coming to that conclusion, and the same holds with regard to the renal origin of urea.

## Surgical Affections of the Kidney


Movable and Floating . . . . 93
Injuries of . . . . . 96
Traumatic Nephritis . . . . 98
Perinephritis and Perinephritic Abscess . . . . . 98
Renal Fistula . . . . . 100
Pyelitis . . . . . 101
Suppurative Pyelonephritis . . . 103
Cysts . . . . . 103
Stone in the Kidney . . . . 104
Renal Tuberculosis . . . . 109
Hydatid Cysts . . . . . 115
Hydronephrosis . . . . . 116
Pyonephrosis . . . . . 117
Tumours of . . . . . 119
Actinomycosis . . . . . 120
The Ureter . . . . . 121
*Injury* . . . . . 121
*Inflammation and Dilatation* . . 121
*Stone* . . . . . 121
Operative Procedures . . . . 123


### Movable and Floating Kidney

Movable kidney differs from floating kidney in its relation to the peritoneum; the latter possesses a complete peritoneal covering and pedicle, while the former is retroperitoneal. The distinction is purely anatomical, and has no importance except in so far as it may increase the difficulties of the operator. The range of movement may be quite as great in

a movable as in a floating kidney, and the symptoms do not differ.

Küster believes that 2·5 per cent of the population, irrespective of sex or age, have movable kidneys; and Albarran states that in 10 to 12 per cent of women there is a slight degree of renal mobility.

(1) *The Surroundings.* — The kidney may move inside its fatty capsule, or the capsule may be loose and wander with the kidney (Morris). The amount of fat surrounding a movable kidney is not necessarily diminished even when the rest of the body is emaciated (Rosenstein).

(2) *The Range of Movement.*—This may be very slight or extensive, and is clinically described in three degrees (Glénard).

First Degree.—The kidney descends partly below the ribs on deep respiration, but its upper pole remains hidden. These slighter cases form the majority of movable kidneys (Morris).

Second Degree.—The kidney descends entirely below the ribs, and the fingers may be pushed above it.

Third Degree.—The kidney wanders over an extensive area of the abdomen. It is anchored by its pedicle of vessels, but may swing as low as the iliac fossa or across the median line of the body.

Sometimes a tilting movement takes place, by which the convex border turns forwards. The movement may be in the "plane of the loins," and has been termed "cinder-sifting," but this movement cannot be detected by palpation (Morris).

The mal-placed kidney may form adhesions in some new position and there become fixed.

(3) *The State of the Kidney.*—Often it is quite normal. A slight degree of pelvis dilatation is frequent, even hydronephrotic changes may take place from kinking of the ureter. Infection of the dilated or congested kidney may produce pyonephrosis or pyelonephritis. Calculus may complicate a movable kidney, but it is uncommon.

(4) *The Condition of other Abdominal Organs.*—Frequently there is no abnormality, sometimes a general enteroptosis is present (Glénard). Movable liver occurs pretty frequently with movable kidney, more rarely the spleen is mobile.

(5) *Some Accidents which may occur.*—Torsion of the renal pedicle, causing acute symptoms of renal pain, collapse, vomiting, and even anuria, may occur and be repeated. This usually happens after some exertion, and only occurs when the degree of mobility is very pronounced. Intermittent hydronephrosis is very frequent. The symptoms resemble those of strangulation, but in addition there is the presence of a renal tumour which disappears with the relief of the symptoms, and is followed by a marked temporary polyuria. Sometimes the hydronephrosis is permanent. Intermittent jaundice is said to occasionally complicate movable kidney on the right side, probably from pressure or dragging on the bile duct. This often passes off suddenly and after a few days reappears (Litten).

*Etiology.* — The kidney is normally more movable in women than in men, and the preponderance of the former over the latter in movable kidney is striking—six or eight times more frequent in females (Rollet).

The right side is much more often affected than the left (9 to 10 per cent, Fürbringer); though both sides are sometimes affected (7·2 per cent). Most cases occur between the ages of twenty and forty, but it also occurs in children. An hereditary tendency has been traced in some cases.

The most frequent exciting causes are injury, and those changes which are induced by weak, pendulous abdominal walls, the result of repeated pregnancies, or distension by ascitic fluid or abdominal tumours. Rapid emaciation, enlargement of the kidney, and tight-lacing are said to be factors in the etiology.

Symptomatology.—*Pain.*—The most constant and usually the most striking indication of wandering kidney is pain (96 per cent). It will therefore be considered somewhat in detail. The suffering caused by a movable kidney shows great variation in its intensity and character. Every kidney which can be detected by palpation and classed as movable does not cause pain; many, indeed, are symptomless, and this especially applies to the left side; but in the majority of instances pain is present in some degree. Often it is the only prominent symptom the patient complains of (43·3 per cent), at other times it is accompanied by general conditions of neurasthenia, or hysteria, or by disturbance of digestion; in other cases the genito-urinary system bears the brunt of the disorders (31 per cent).

In some cases pain amounts only to a dull, heavy aching in the loin, or a sense of dragging, which is continually present; in others there is occasional pain, often severe and situated in the lumbar region. Sometimes it is described as sharp, and may radiate to the abdomen and even to the thigh.

Exercise has a marked effect in starting and increasing the pain, while rest often relieves it.

In women, and they form the majority of patients, the menstrual periods are attended by more severe exacerbations of the pain, necessitating rest in bed.

In another class the pain is paroxysmal in character and agonising in severity, and shows the same radiation as renal colic. Some strain or extra fatigue often starts these attacks, and after a variable duration they suddenly cease.

Temporary disappearance of the symptoms of

a movable kidney sometimes occurs, very rarely the improvement is permanent.

The collection of symptoms which represent a movable kidney are of the most varied description. Simple enumeration only leads to further confusion, for none of them are characteristic. They fall, however, into three well-marked groups, and are thus most conveniently described although combinations of the different types occur:—(1) Genito-urinary group, (2) Gastro-intestinal group, (3) Nervous group.

(1) *Genito-urinary Group.*—These cases resemble renal calculus in many of its phases.

The pain is in the loin or side; it is often insidious in its onset, and of a dull, heavy, aching character. It may be occasional and increased by exercise (horse-riding, etc.), or it may be constant and become more severe as time goes on.

In a large number of cases (41·8 per cent) there are attacks at intervals of severe pain exactly simulating renal colic in their character and distribution. The same intense agony and prostration are seen, the same nausea and vomiting, sweating, feeble rapid pulse, diminution of urine, and even anuria and uræmia (Pribram) may be present, and the same sudden relief from the symptoms is obtained. The patient during an attack sits doubled up with his knees to his chin and his body bent (Newman). The temperature often rises during an attack (Furbringer). Hæmaturia may follow these attacks. Blood was present in the urine in 18 per cent of Morris's cases of movable kidney.

During an attack the kidney is increased in size, and relief is followed by a copious polyuria.

Frequency of micturition may occur (18 per cent), and pus in small amount may be observed.

Albumin is present in the urine in 14 per cent of cases (Schilling).

So closely do these cases resemble renal calculus that in many the exclusion is only complete when the renal substance has been incised and the kidney and pelvis explored by the finger.

(2) *Gastro-intestinal Group.*—The symptoms point to gastric trouble: the patient is dyspeptic, complains of pain in the back, a sensation of sinking and weight after food. Constipation is often present, nausea and anorexia are frequently complained of. Jaundice may occur as already noted.

On examination of the abdomen the stomach is sometimes found dilated (Litten says in 55 per cent), and sometimes a general condition of enteroptosis may be discovered.

(3) *Nervous Group.*—The symptoms vary greatly. The patient is often nervous, irritable, excitable, and suffers from palpitation. Severe neuralgia may be present, or the pains may be vague and variable. Spots of hyperæsthesia and anæsthetic areas often occur. Sometimes weakness of the lower extremities has been seen (Senator).

The symptoms may be typical of hysteria, or the patient may be neurasthenic. In those cases there is often a neurotic family history, and a slight injury frequently determines the onset of the symptoms.

*Diagnosis.*—In all cases the diagnosis rests upon the discovery of a movable tumour which is recognised as the kidney.

Sometimes a movable abdominal swelling is the first sign noted. It may be discovered by the patient either before or after the onset of symptoms. In the slighter degrees of movable kidney, if the mobility can be detected the position of the swelling leaves no doubt as to its nature; but in the more pronounced cases, where the range of mobility extends towards or into the false pelvis or towards the middle line, there are other conditions which may lead to confusion.

The movable kidney has certain characters which should always be looked for. The shape can sometimes be made out and is characteristic; there are no sharp borders; by manipulation the organ can be replaced in the loin; on percussion a dull tympanitic note is obtained anteriorly.

No conclusions can be drawn from the percussion of the loin, or the presence or absence of hollowing in that region. Albarran points out that sometimes, when the right lobe of the liver is displaced downwards and the kidney movable, the size of the kidney may appear very great on palpation, and a hydronephrotic tumour be diagnosed when none is present.

The abdominal swellings most likely to be confused with a wandering kidney are:—

(1) *An Ovarian Cyst with a long Pedicle.*—The swelling can be reduced into the pelvis, but cannot be placed in the renal region. It is dull on percussion, and sometimes the abdominal wall is sufficiently thin to recognise that the form is not that of the kidney.

(2) *A distended gall-bladder* has a smaller range of mobility, and its dulness is continuous with that of the liver, while it is not reducible into the lumbar fossa. Jaundice, if present, inclines to the diagnosis of distention of the gall-bladder; but it should not be forgotten that an attack of jaundice may complicate a movable kidney, and further, that biliary and renal colic may be very similar, and, lastly, that the two conditions may occur together.

(3) *An hydatid cyst* attached to the lower surface of the liver may cause difficulty. It is, however, painless, and swings round an axis corresponding more to that of the gall-bladder than the kidney. The tumour is not reducible into the loin.

(4) *A wandering spleen* has a sharp margin, sometimes notched, and a dull percussion note. A movable spleen may sometimes descend as low as the iliac fossa.

(5) *Mesenteric tumours* are median in position, and show a greater transverse than vertical mobility, and are dull on percussion.

*Treatment.*—(1) In some cases a floating kidney is discovered by the patient or medical attendant, but no symptoms which might arise from it are present. Here it is better to advise the patient to wear an abdominal belt, for symptoms may arise at a later date. Sometimes the knowledge of possessing a movable kidney seems to excite uneasiness and discomfort, and eventually hypochondriasis.

(2) In cases where symptoms are present a snugly fitting abdominal belt with an elastic cushion, with an air pad on its inner surface, and placed low down on the affected side, should always be tried, and in thin patients a fattening diet will sometimes be found of advantage.

Where the symptoms are controlled by the apparatus the patient will have the option of retaining it for life or having an operation performed. Where the symptoms are unaffected by wearing an abdominal belt, operation should be recommended. It will usually be welcomed by the patient as an almost certain means of escape from her sufferings.

The operation of nephrorraphy or nephropexy is, in the hands of an experienced surgeon, practically without a mortality (1 to 2 per cent). In the majority of instances the cure is complete and permanent. (For technique, see Operations.)

The success of the operation may be considered in relation to the various symptoms.

*Pain* disappears in 88 per cent of cases after nephrorraphy (Albarran).

In the *gastro-intestinal group* the troubles are less often completely relieved.

It is in the *nervous type* that treatment either by bandage or by operation has least effect. In Albarran's statistics only a small number were cured by nephrorraphy (14 per cent were improved, 36 per cent showed no improvement in their nervous symptoms). This author considers that if the cause be allowed to remain the condition will be aggravated, and he does not hesitate to recommend operation even in those nervous cases after orthopædic measures have failed.

(3) In cases where enteroptosis is present an abdominal belt should be worn, and no operation is called for unless some complication such as hydronephrosis arises.

## Kidney, Injuries of

Injuries to so essential and so vascular an organ as the kidney cannot fail to be of grave moment. Even a slight contusion in one so predisposed may start a tuberculous affection, or induce chronic interstitial nephritis; whilst the severer lacerations, involving, as they frequently do, the superjacent peritoneum or surrounding abdominal viscera, result in heavy mortality.

The practical consideration of the subject falls into two divisions.

(*a*) Subcutaneous lesions.

(*b*) Open lesions.

(*a*) *Subcutaneous lesions* of the kidney are more commonly met with in civil practice. They occur chiefly in men. The nature of the violence may be direct or indirect; its effects may be limited to the organ (simple lesion), or extend to the encasing structures or the surrounding viscera (complicated). Thus the peritoneum covering the anterior surface may be split, or the adjoining ribs may be broken (5·5 per cent, Küster) and their fractured ends buried in the kidney; or the liver, spleen, gut, even the diaphragm and lung, may be coincidently and extensively torn.

*Pathological.*—There may be mere subcapsular ecchymoses or laceration, or the capsule may be ruptured and deep multiple stellate fissures traversing the kidney substance even to the hilus may exist, or the kidney may be rent into two or more isolated pieces or reduced to a pulp. The fatty capsule and muscles are frequently torn, and in children up to the age of ten the peritoneum is very liable to be split open, because the prenephric subperitoneal fat is absent before that age (Poireault).

The mortality rises from 30·4 to 80 per cent when the peritoneum is torn (Edel).

*Clinical Notes.*—(i.) Anatomical limits of the hæmorrhage.

If the renal capsule is ruptured the blood issues into the perirenal tissues until it is checked by the fatty capsule; a firmish rounded tumour is thus formed. But if the fatty capsule is also torn, the hæmorrhage, if severe, escapes along the cellular planes in every direction, and may reach the other side of the vertebral column, or extend to the thigh, groin, or scrotum. In exceptional adult cases, and in children under the age of ten, the blood may pour into the peritoneal cavity through a split in the peritoneum. If a deep calyx or the pelvis be opened, urine follows in the track of the blood.

In the severer cases every element favouring virulent septicity is thrown in combination. An organ, whose rôle is to eliminate micro-organisms and toxins, is damaged; adjacent to it is the colon harbouring myriads of pyogenic bacilli, and around it is a widespread undrained bog of fluid blood, clot, damaged tissues, and putrescible urine. Small wonder that the mortality is great (30 per cent simple, 70 per cent complicated), and that it is largely due to septicity.

(ii.) The indication of renal hæmaturia consequent upon slight *indirect* violence.

It is important to remember that pre-existing disease of the kidney may cause that organ to be easily lacerated. I always suspect the previous health of a kidney which bleeds on

slight indirect violence, such as a short fall upon the buttocks, or a muscular strain in lifting. Severe renal hæmorrhage upon the occurrence of slight violence after mid-adult age and in old people should raise a suspicion of malignant growth: in the decade between thirty and forty of calculus, and in the young adult of chronic interstitial nephritis or tubercle. I have met and verified several instances of each of these diseases, the first intimation of which was given by the appearance of hæmaturia consequent upon a *slight* fall, blow, or strain.

*Symptoms.*—Some amount of shock is present in nearly every case of renal laceration, the collapse being the more profound and lasting in proportion to the severity of the lesion. As the shock passes off, nausea and vomiting set in, severe pain is experienced in the area of the injured kidney and along its ureter, the kidney becomes exquisitely sensitive, and the muscles over it markedly rigid. Tympany ensues in a few hours although the peritoneum may be uninjured. Blood is passed in the urine; the patient is often tormented with dysuria, although the actual quantity of urine may be markedly diminished, and a swelling is soon detectable in the renal region, whilst ecchymosis, more or less extensive, discolours the skin, and marks the subcutaneous limits of the effused blood.

*Hæmaturia.*—This cardinal symptom varies according to the site and extent of the laceration, although it is no indication of the amount of the blood escaping, for much of it may percolate from the kidney into the perirenal area. If the laceration is purely cortical the hæmaturial admixture may be only microscopical and in the shape of a few blood cylinders. If, however, the kidney is extensively torn, the bleeding may be so profuse as to pour down the ureter, enter and fill the bladder, and either clot there, producing retention, or frequently issue thence by the act of urination, arterial in hue and fluid in consistence. It may even cause death. Thus Grawitz has collected seventeen cases in which death ensued within half an hour to fifteen hours after the injury. Usually, however, although the hæmorrhage is at first bright, it rapidly darkens and clears off by the third day. If it remains profuse after the fourth day there is cause for alarm, especially if the swelling in the loin continues to increase. Sometimes it is intermittent, or varying in amount at different times, or it may even be absent (Newman). In forty-nine deaths from uncomplicated lesions of the kidney fifteen died rapidly without hæmaturia (Tuffier).

*Course of a Severe Case.*—When the peritoneum has been torn and the adjoining viscera have been lacerated the patient usually succumbs. Küster says that only one case—that of Kehr—in which the peritoneal covering was proved to be torn has recovered; but in the more favourable cases, when the injury is limited to the kidney, the patient, when he has escaped the immediate danger of hæmorrhage, is confronted with that of septicity (76 per cent, Tuffier). This usually takes the form of cystopyelitis, which quickly induces in its turn pyelonephritis, perinephritis, and finally septic peritonitis. It is needless to add, if the inflammatory products around the kidney are unrelieved by free drainage the patient dies.

Septicity is usually heralded by a rise of temperature, and increase of lumbar pain, and often these symptoms *follow hard upon the introduction of a catheter*; for there is no doubt that part at least of the heavy mortality is due to septic catheterism.

(*b*) *Open lesions of the kidney.*

The symptoms which attend open wounds of the kidney are the same as those which mark subcutaneous lesions, but with these differences: —Hæmaturia, which is the cardinal symptom, is nearly always present; the pain, which is in proportion to the laceration of the muscles, is usually more localised, and does not extend along the ureter; whilst, owing to the generally free escape of blood or urine, the lumbar swelling is not present, or does not become so marked a feature. The prolapse of the kidney into the wound is probably rare. The danger of hæmorrhage is as great as in subcutaneous wounds, but that of septicity is less, owing to the escape from the wound of the products of inflammation. Hence the death-rate is lower (15 per cent). Moreover, statistics show that many of the patients have been operated upon, and nearly always successfully.

*Treatment.*—(*a*) In *subcutaneous lesions.*

In a fair proportion of those cases in which the hæmaturia, the renal pain, and the loin swelling are slight, rest in bed will suffice. The application of an ice-bag for a few hours relieves mentally, if it does not influence the pain and hæmorrhage. Small doses of an opiate are always beneficial, but internal hæmostatics by injection or by the mouth are useless. Subsequently, strapping the affected side as if for fractured ribs (Morris) affords comfort and rest.

In graver cases, when the hæmaturia is severe and the loin swelling is marked, operative interference becomes a necessity, not only to arrest the hæmorrhage, but also to give a free outlet to the perirenal collection of blood and, may be, urine, and to anticipate and prevent those septic changes which prove so fatal.

In fact, early surgical intervention will become, I believe, the rule in all the severer cases. Whether tamponade of the surface of the kidney and subsequent free drainage, or stitching of the fissures, or partial or complete nephrectomy, should be undertaken are questions which must rest upon the judgment and experience of the operator. At the same time that the loin is

opened, the necessity or the advantage of perineal drainage of the bladder should be raised. If the bladder is feeble or atonic, and clots show a decided tendency to form in that viscus, it is good surgery to place a large perineal drain in the bladder so as to prevent the inevitable cystitis and those ascending changes which follow in its train.

If prolonged shock and symptoms of profuse hæmorrhage into the peritoneum point to the tearing of the peritoneal surface of the kidney, the abdomen must be opened by the transperitoneal incision and the blood removed.

(*b*) *Open lesions.*

In all cases of open lesion it is essential, if the hæmorrhage or hæmaturia be severe, to enlarge the wound, and to deal with the kidney as the injury demands.

### Traumatic Nephritis

After a blow or injury to the loin the onset of nephritis is marked by a rigor and the temperature rises. Blood is usually present in the urine, and when this disappears after a few days the microscope will show tube-casts and epithelium, and for a day or two red granular material is often observed (Küster). Albumin is present in varying amount.

A peculiarity of traumatic nephritis is the combination of albuminuria with polyuria, which contrasts with the scanty urine of acute nephritis. A further distinctive feature is the rapid development of œdema of the feet, face, or sometimes the whole body. The œdema is not uncommonly (three in five cases, Potain) confined to one side, the injured one, of the body (Küster). Some days later pyuria may appear, and if the temperature remains high suppuration has probably occurred.

It should not be forgotten in the diagnosis of traumatic nephritis that old-standing renal disease may have preceded the injury.

*Prognosis.*—In traumatic nephritis only one kidney is affected, and recovery as a rule takes place, the attack passing off in ten or fourteen days (Morris). Some cases go on to chronic nephritis (Albarran). Suppuration sometimes occurs and will require surgical interference.

*Treatment.*—The rest and diet already observed on account of the injury should be continued. Leeches may be applied to the loin.

### Perinephritis and Perinephritic Abscess

Inflammation of the areolar tissue around the kidney is comparatively rare, the published cases not exceeding more than a "few hundred" (Fürbringer).

*Etiology and Pathology.*—The inflammation may stop short of actual pus formation and form only a dense fibrous thickening, or suppuration may take place.

Before describing the symptoms of the disease it is convenient to review briefly those conditions which may lead up to it, and which one may expect to find in the history of a case, and also to note some facts of interest in their bearing on the prognosis.

The disease is more frequent in males than in females, and occurs usually between the ages of twenty and forty.

The right side is more often affected than the left; it is seldom bilateral (3 in 230 cases, Küster).

In many cases careful examination of the other organs and attention to the history reveal no condition likely to induce suppuration, but in others the history of a chill, a muscular strain or a blow in the lumbar region (probably 26 per cent, Duffin) is obtainable, or some small suppurating point, such as a whitlow, a boil, or carbuncle, is present; in 17 per cent (Fenwick) perinephritic abscesses occurred during the course of some severe toxæmic condition, such as pyæmia, smallpox, scarlatina, etc.; a few can be traced to disease in the appendix, liver, pelvic organs (23·5 per cent) or the vertebræ (3·7 per cent).

Lastly, a separate group of cases takes origin in disease of the kidney (32 per cent), and these are named "secondary" while the others are "primary."

*Symptoms.*—In some conditions the causative disease is severe, and masks the symptoms of the perirenal suppuration, which may only be discovered on the post-mortem table. Thus pelvic cellulitis may be complicated by perinephritic abscess without the latter condition sufficiently modifying the symptoms already present to attract attention, or during the malignant course of pyæmia or smallpox the kidney may become surrounded with pus without the latter giving any indication of its presence. In the primary class of cases the disease attains its most rapid and pronounced form; even here, however, the signs may sometimes be obscure and lead the practitioner astray, and this occurs more especially in the older and more weakly individuals than in the more robust.

In the other type of case where some renal disease is present and especially of calculous nature, the symptoms are more moderate, the course more prolonged, and the patient less likely to survive the disease.

*Onset.*—In most cases the disease commences insidiously with pain (64 per cent, Fenwick) of a dull, heavy character in one loin, or an indefinite aching on both sides, which later becomes more localised. This mode of onset is especially frequent when the abscess is secondary to renal disease, less often when other causes are present.

When the suppuration arises apart from disease of the kidney, a sudden rigor and rise of temperature is more likely to be the preface to further symptoms.

In a few cases (9 per cent) symptoms of urinary disease are already present when other signs appear.

*Pain.*—The initial pain soon becomes more severe. From a dull, heavy aching it becomes sharper and more stabbing, and often assumes a paroxysmal character of great severity. Sometimes it is intermittent, and it has at times a remarkable "tertian" character (Elias). Confined at first to the affected loin it later radiates along the distribution of the lumbar nerves to the hip, thigh, knee, groin, and sometimes to the testes and penis.

The pain is exaggerated by every movement, and especially by coughing or straining. Tenderness in the renal region is an early and invariable symptom. Some temporary anæsthesia or paresis of the thigh on the affected side has been observed (4 per cent, Nieden).

*Fever* may be severe, commencing at the outset of the disease with rigors. It is usually continuous, with morning remissions. Like the pain it may be absent at intervals, and in this way resemble ague. Fever is a more striking feature in those cases which arise apart from renal disease than in those where the inflammation spreads from the kidney. It has been noted that in some cases the pain and fever become less marked a day or two before the appearance of the swelling. In subacute or chronic cases the fever may be slight.

*Tumour.* — In the early stage nothing but marked tenderness is discovered on palpating the loin. So severe is this pain on pressure that the abdominal wall resents the palpating hand by firm contraction, and an anæsthetic is advisable in making examination. With the patient recumbent, if one hand be placed so as to support each loin, an increased sensation of weight may be detected by the surgeon before any swelling can be defined (Morris). After some days (eight to fifteen days, Trousseau) or weeks or even months, an indefinite fulness can be felt deeply placed, and later a lumbar tumour appears (87 per cent, Fenwick).

At first this is firm and ill defined, later an actual lumbar swelling can be seen. The natural curve of the waist becomes obliterated, the skin œdematous, and the surface temperature is raised. Sometimes when the abscess tends to point the skin is reddened and congested.

On palpation the swelling is very tender, and if the abdominal wall permits, the outline will be found indefinite and fluctuation can be elicited. The respiratory movements do not affect the tumour. The flank is dull, but at the interior and inner side of the swelling a tympanitic note betrays the position of the colon. Much depends for ease in examination on the thickness of the abdominal wall. As much as six pints of pus have been concealed beneath a fat belly wall without fluctuation being discovered (Morris). There is a distinguishing tendency to bulge backwards into the flank, rather than forwards into the abdomen (Rosenstein).

In some subacute cases a tumour is found lying under the unaltered skin, which is very elastic, often resistent and globular or egg-shaped, rarely of hour-glass form (Güterbock).

*Effect on the Attitude and Movements of the Patient.*—Lameness is often observed as an early symptom (Duffin), and the patient in sitting tends to rest on one tuber ischii (Morris). The body is often held bent forwards and inclined towards the affected side; this habit will draw attention to the hip, and in the presence of referred pain in this region without marked lumbar symptoms, may lead the observer astray.

On examination the patient lies on his back in bed, with the thigh of the diseased side flexed, and often abducted and rotated outwards, so that the heel is in relation to the dorsum of the other foot, the thigh cannot be extended without pain, and adduction is performed with difficulty (Gibney). The condition closely resembles that of the second stage of hip-joint disease, and is due to the unconscious attempt at relaxation of the abdominal and psoas muscles. There is, however, entire absence of tenderness, swelling, or muscular wasting about the hip-joint, and by flexion of the thigh the psoas muscle is thrown out of action, and rotation is now perfect and painless.

*Condition of the Urine.*—Changes in the urine occur in 33·3 per cent of cases (Güterbock) but these give very little aid to diagnosis.

In cases of old-standing urinary disease there may be blood, pus, etc. in the urine, but the condition is unaffected by the presence of perinephritic suppuration.

When renal disease is absent, albumin, casts, and even blood may appear in the urine, probably from pressure on the renal vein (Morris). Pus is present at intervals in 10 per cent of primary cases (Fenwick). In some cases a large number of bacilli have been discovered in an almost normal urine preceding rupture into the renal pelvis (Furbringer). Often no urinary trouble is present at all, or only the abundant lithatic deposit of febrile urine.

*General Symptoms.*—Constipation is invariable, and is a marked feature of the case, while flatulence is very troublesome.

Doubtless the constipation may be partly explained by the great increase of pain when the bowels are moved.

The appetite fails, there is often nausea, sometimes vomiting, and in acute cases rapid loss of flesh occurs.

Interference with movements of the diaphragm causes a marked frequency of respiration.

Unilateral œdema of the foot or leg is sometimes observed, and has preceded the other symptoms by some weeks.

*Diagnosis.*—Apart from the latent cases there are two broad types, the acute and the chronic.

The practitioner is most likely to be led astray only during the early stage before the swelling appears, and only pain and fever are present. In cases where *fever and rigors are the prominent feature* of the case, *some acute infectious disease*, such as influenza, scarlatina, or typhoid is simulated.

In the history there may be little to guide one, but a strain or lumbar injury should always lead to examination of the kidney region. The pain in the back in these fevers is more general, and there is no tendency to unilateral localisation as in perirenal inflammation. In a few days the appearance of a rash or other signs will clear up any doubt.

*In cases of less febrile type* the continuous aching or shooting pain may resemble lumbago, neuralgia, and even renal colic. In *lumbago* the pain shows some relation to movement, it is bilateral, the muscles are tender on pinching them up, and fever is wanting.

In *neuralgia* there is absence of the marked tenderness of perirenal abscess, no temperature or rigors, and the pain is more completely intermittent.

*Renal colic* is accompanied by marked urinary changes, blood, pus, and frequent micturition, and these are reliable guides, because the onset and course of the symptoms in the form of perirenal abscess which complicates renal calculous disease, and which is accompanied by changes in the urine, are usually insidious and moderate (Fenwick). At a later stage, *when a swelling has become evident*, the condition most likely to be mistaken for perinephritis is pyonephrosis.

*Pyonephrosis.*—Here, however, the course is more chronic and the symptoms less severe, pain is not a marked feature of the disease, tenderness is less marked, the tumour is well defined and regular, and the skin of the post-renal area lacks that waxy, œdematous condition so frequently observed in perinephritis.

*Appendicitis* may closely simulate perirenal abscess, and may be a cause of it. The tenderness is, however, usually at a lower level and the swelling is in the iliac fossa.

In less acute cases a *cold abscess*, originating in spinal caries, may resemble perinephritic abscess. The angular curve, the rigidity of the whole spine, the local tenderness on jarring the column or on pressing the spinous processes, and the slower course and smaller size of the abscess, will lead to a diagnosis.

*Treatment.*—Before diagnosis is certain, the treatment (rest, diet, medicine) is that of any acute fever. In early cases where perirenal inflammation is suspected, cupping, fomentations, and opium may aid in bringing about a resolution, for it is undoubted that a few cases do not go on to pus formation; but much time should not be wasted on these measures, and as soon as perinephritis is diagnosed, incision and evacuation of the abscess is the safest and most speedy means of treatment. It is well to bear in mind the following points in recommending operation:—

1. In acute cases septicæmia and pyæmia may occur.

2. In less acute, the abscess may burrow and rupture in various directions. Rupture occurs on an average in from three to five months (Lancereaux), and has a death-rate of 53 per cent (Rosenberger).

3. The majority of unoperated cases do not rupture, but die of exhaustion from hectic and waxy disease (Newman).

4. "Primary" cases are more favourable, mortality 16 per cent (Küster), than "secondary" (to kidney disease), mortality 49 per cent (Kuster).

5. And lastly, when free incision is employed 94·4 per cent recover, irrespective of primary or secondary disease, while without operation only 13·6 per cent survive (Poland).

### Renal Fistula

Etiology and Anatomy.—After wounds of the kidney urine may be discharged for some time, but the fistula is of short duration. If the calices or pelvis of the kidney be opened, and especially if suppuration be superadded, the discharge is more likely to be prolonged. Most renal fistulæ either follow operations upon the kidney or occur spontaneously.

1. *Fistula following Operation.*—The fistula is usually tortuous, and lined with granulation tissue which projects at its orifice. Phosphatic deposit may take place on the walls, and urea and urinary salts are present in the discharge. After a time the urine may disappear from the discharge from destruction of the remaining kidney tissue.

*Obstinate Symptoms.*—The general health is often quite unaffected so long as the discharge is free, and after lasting for many months or years (sometimes seven years, Morris) the fistula sometimes closes, and the wound becomes sound and permanently healed. In other cases the discharge diminishes, but at the same time the improvement in the general health apparent after the original operation is not maintained, there is continuous pain in the renal region, tenderness on pressure, and a swelling is still felt there. The temperature is raised, and now and again a sudden rise and fall occurs and the strength fails. In other cases the fistula closes completely, the temperature suddenly rises, and there is pain and tenderness in the renal region. Sometimes the attack is accompanied by a rigor, and vomiting may occur. In a few days the skin becomes reddened and tender, the scar breaks down and the discharge recommences, but from time to time these attacks of reten-

tion recur. Septicæmia or pyæmia supervene in some cases where the drainage is incomplete.

2. *Spontaneous fistulæ* are not often met with, since early operation has been recognised as the best treatment for kidney calculus.

The rupture of a calculous, less frequently of a tuberculous pyonephrosis or a perirenal abscess, most often occurs in the loin, and usually at the triangle of Petit (Albarran). The opening may, however, be in the loin or buttock, or some part of the abdominal wall distant from the seat of the disease. The opening is commonly single, and although secondary openings do occur, they are not so often observed as in urinary fistulæ originating in the lower urinary tract (Guterbock). Sometimes the orifice is situated in an intercostal space. In these cases, fortunately, the pleural sac is pushed up and is not involved (Morris). Rupture into abdominal viscera (stomach, intestines, etc.), and even a fistula communicating with the lung may be formed.

*Diagnosis.*—Spontaneous fistula opening at some distant point may cause difficulty, but the discharge of urine and the history of urinary disease and abnormality in the urine will betray its origin.

*Treatment.* — Fistulæ following operation should be kept clean, and the surrounding skin protected by an ointment. If phosphatic encrustation of the track has taken place, it should be syringed with an acid solution (weak hydrochloric acid).

When the discharge is slight a sufficient pad should be worn to absorb it, even a copious discharge of urine in the loin may be compatible with comfort if a receptacle be worn (Morris). Exuberant granulations should be touched with silver nitrate, and a small superficial sinus may sometimes close after a few applications of the hot wire; but there are some conditions which demand operative interference. These consist in:—

(1) Recurring attacks of retention of the discharge, complete or incomplete.

(2) The inconvenience of the discharge becoming intolerable.

(3) Nephrotomy for tubercular or calculous pyonephrosis is a temporary measure to tide the patient over a crisis; when the health has improved sufficiently nephrectomy will probably be required to avoid a permanent fistula and the effects of prolonged suppuration.

## Pyelitis

The relation between the kidney and its pelvis is so intimate that inflammation of the latter seldom occurs without the kidney participating to some extent in the process. It is practical, however, to draw a distinction between pyelitis and pyelonephritis, for in the latter the disease has invaded the kidney structure and the prognosis is grave.

*Etiology.*—Any period of life may be affected, but pyelitis is most frequent during middle life and in males, for the causes which produce it come into operation at that time and in men.

There are three classes:—

(1) Those arising from local conditions, of which stone in the pelvis is the most important (pyelitis calculosa).

(2) Those following disease of the lower urinary organs. These, which form the largest class of pyelitis, may be briefly enumerated:—

Cystitis with decomposition of the urine and ascending inflammation of the ureter and pelvis, in which the process travels from the bladder to the renal pelvis or kidney by direct continuity along the mucous membrane, or by way of the lymphatic vessels (Lindsay Steven).

Gonorrhœal cystitis accounts for 18 per cent of cases of pyelitis (Finger), but vesical calculus, bladder growths, etc., are predisposing causes, and septic catheterisation a frequent exciting cause.

Atony of the bladder from obstruction or paralysis and decomposition of retained urine is a frequent cause, and in fact 74 per cent of those who die with hypertrophy of the prostate suffer from pyelitis, and in fatal cases of urethral stricture, inflammation of the renal pelvis is found in 41 per cent (Fürbringer).

Atony of the bladder with cystitis, in spinal or cerebral disease, form a small class.

Lastly, operations in the neighbourhood of the bladder by spread of inflammation along the ureter induce pyelitis.

(3) Infection by way of the blood stream. Pyelitis sometimes occurs during the course of one of the acute infective diseases, such as scarlatina, diphtheria, dysentery, cholera, typhus, smallpox, or puerperal fever.

Again, poisons introduced into the body—such as cantharides, turpentine, balsams, etc.—may give rise to pyelitis during their excretion.

Tubercular pyelitis requires separate consideration (*vide* Renal Tubercle).

*Pathology.*—Pyelitis is more frequently unilateral than bilateral. This applies especially to calculous pyelitis: in the ascending form it is often bilateral, but one side is usually affected before the other (Fenwick) and to a much greater extent than the other (Senator). The acuteness of the process varies with the cause. Slight forms are catarrhal, with hyperæmia and thickening of the mucous membrane and desquamation of the epithelium, and are found especially in early calculus. Severe infective forms may be attended with formation of a membrane (croupous and diphtheritic), while the chronic forms due to long presence of calculi, etc., show a thickened, discoloured, grayish mucous membrane and an infiltrated wall. Small cysts may form with colloid contents (pyelitis cystica, Litten, v. Kalden), or small grayish lymph follicles (pyelitis

granulosa, Chiari), and ulceration is often present.

Backward pressure is often coincident with pyelitis, and dilatation of the renal pelvis and atrophy of the kidney substance results (hydronephrosis and pyonephrosis). Suppurative nephritis or chronic interstitial nephritis may occur from spread of the inflammation.

*Symptomatology.*—The amount of suffering depends more on the cause than on the pyelitis itself. The severe microbic types, *e.g.* pyæmia, etc., may be unattended by pain, or at most have a dull aching, while in slight cases due to calculus the agony may be intense and prolonged. Sometimes, however, pain in a pure case of pyelitis (for instance, puerperal fever) may resemble renal colic in its intensity and distribution (Ebstein). Ascending pain along the ureters, followed by dull aching in the kidney, may be due to pyelitis apart from the symptoms of its cause.

The pus in the urine intermits, being usually noticed to be more abundant in the first urination on rising in the morning.

*General Symptoms.*—Continued fever does not always accompany chronic suppurative pyelitis unless the ureter becomes blocked, but a slight rise of temperature may occur at night (Newman).

In uncomplicated pyelitis the most important signs are found in the urine.

Changes in the Urine.—*Pus.*—The urine, when passed, is milky and opalescent, but on standing the pus soon separates into a creamy layer at the bottom of the urine glass, clearly marked off from the supernatant urine. The amount of mucus present is slight and not sufficient to cloud the urine.

*Reaction* is acid; even when the urine of pyelitis is fœtid it may retain its acid reaction from admixture of the secretion of the normal kidney. In the later stages it sometimes becomes markedly alkaline.

*Odour.*—In the earlier stages there is no smell. When decomposition is pronounced Dickinson remarked a peculiar smell like sulphuretted hydrogen, which he distinguishes form the ammoniacal odour of the urine of bladder inflammation.

*Albumin* is present, but corresponds to the amount of pus. When the deposit on boiling is excessive, it raises the suspicion that the renal tissue has been invaded, and other signs of this complication should be looked for.

*Cells.*—Besides pus corpuscles numerous epithelial cells are often present, elongated, pointed, and often overlapping each other, which a skilled microscopist can detect as pelvic.

*Bacteria.*—Numerous micrococci and bacilli can be seen (quite apart from decomposition) in the recently passed urine.

Diagnosis.—Pyelitis must be distinguished from—(1) *Cystitis.*—In cases of pyelitis, when the urine is decomposed and alkaline, and painful and frequent micturition is present, cystitis will be stimulated, and yet the bladder be free from disease. The history is important, for it may show long-continued renal symptoms previous to the onset of bladder trouble; the amount of mucus and the ammoniacal decomposition are greater in bladder affections. The cystoscope is the best guide in doubtful cases—the diagnosis resting on the pyuric efflux, the shape of the ureteric orifice, and the urine obtained from each pelvis by the ureter catheter.

In the class due to ascending inflammation from disease of the lower genito-urinary tract the diagnosis of pyelitis apart from renal changes is very difficult, often impossible. Ascending ureteric pain followed by dull, heavy aching and tenderness of the kidney should raise the suspicion of this complication. It may be possible, by massage of the kidney, to obtain an appreciable increase of the pus in the urine.

(2) *Pyelonephritis and Chronic Interstitial Nephritis.*—In acute pyelonephritis there are rigors, high fever with a feeble, rapid pulse, a coated, dry tongue, thirst, and vomiting. The urine is diminished in amount, and contains a large quantity of pus and albumin. The patient rapidly sinks into a typhoid condition.

If chronic indurative changes have occurred in the kidney there is progressive loss of weight, failing appetite, headache, thirst, and sometimes vomiting; the tongue is coated and the mouth dry. There is polyuria, the urine is of low specific gravity, the albuminuria is in excess of the pus present, and tube casts may be found. The condition of the urine is often masked by the cystitis already present.

Treatment.—*Prophylactic.*—Many cases of ascending infection may be avoided by careful antiseptic catheterisation, and the prior administration before interference of hexamethylentetramine, gr. v.

*The indications* to be followed are: (1) to remove the cause; (2) to control the amount of pus and relieve pain.

(1) This includes the treatment of renal calculus, enlarged prostate, urethral stricture, and other conditions. A word of warning is necessary in cases where backward pressure is an element. If there is any reason to suspect that the renal tissue has been invaded, operative interference should be limited to the methods which throw least strain on the weakened kidneys.

(2) In all cases medical treatment should be adopted and urinary antiseptics exhibited.

Hexamethylentetramine (gr. v.), sandal-wood oil, ammonium benzoate, boric acid, natural salicylic acid, and salicylates give the best results. Sandal-wood oil is efficacious in the chronic types, or antiseptics may be combined with the liquid extract of white sandal-wood.

Tonics should be prescribed, and of these quinine and nitro-hydrochloric acid are valuable.

In acute cases the patient should be strictly confined to bed, and dry cupping or leeches applied locally, and opium fomentations to relieve pain.

### Suppurative Pyelonephritis

Suppuration in the renal pelvis and kidney is the result of secondary infection from the lower urinary tract. It is the closing stage of many cases of old-standing cystitis, and occurs especially when some form of obstruction is present.

*Etiology.*—Infection introduced into the bladder—often by a dirty catheter—spreads to the kidneys already damaged by obstruction. This occurs in cases of old-standing urethral obstruction (44 per cent), stricture, enlarged prostate, etc., in long-continued cystitis (28 per cent), from calculus, growths, etc., or in cases of bladder atony (24 per cent), from brain or spinal disease or injury (Dickinson).

*Pathology.*—The septic process spreads along the ureter or its lymphatics to the renal pelvis and kidney. Yellow streaks are found passing from the pyramids along the tubules to the cortex, where yellowish splashes or actual abscesses are dotted here and there.

*Symptoms.*—There is a combination of septic absorption with uræmia. At the outset there is a rigor, which is sometimes severe, and it may be repeated, but often this only occurs a day or two before death.

The temperature rises to 101°-103° F., or even higher, and remains up with slight morning remissions. Sometimes in old people, or those advanced in cachexia, there is no fever, although other grave symptoms are present, while in other cases the temperature may return to normal for four or five days, and then another rise occurs. The patient complains of thirst and headache; he refuses food, the bowels are constipated, and there is troublesome flatulence. The mouth is dry, the tongue coated, brown, and cracked ("parrot tongue"), and only protruded with difficulty. Sweating often occurs and is profuse, but shows no relation to the rise and fall of the temperature. There is rapid emaciation; the face has an anxious, sallow look, but is never jaundiced. The fever is unaccompanied by excitement or delirium. The patient becomes indifferent to his surroundings and dull; he replies to questions but slowly, and from time to time dozes off into a restless sleep from which he awakes with a start. Quiet muttering delirium often occurs, the torpor increases, and an hour or two before death coma supervenes.

*Urine.*—If the urine is clear before the onset, it becomes turbid and deposits pus on standing; often, however, it is already thick, muddy, and alkaline from long-continued cystitis. Albumin is constantly present in moderate quantity, and tube-casts and epithelial cells are found, but these signs are usually masked by the purulent urine of cystitis.

Diminution in the quantity of urine is constant and the specific gravity is low. There is usually little or no pain, but indefinite aching with tenderness on pressure is sometimes present.

The kidney cannot, as a rule, be left, although it is enlarged.

There are three conditions which resemble the clinical picture of a suppurative nephritis:—

(*a*) *Acute nephritis* (Bright's disease).

Here the urine is greatly reduced in quantity (four or five ounces in twenty-four hours); it is smoky or porter-coloured from blood, and the specific gravity is high (1025). The puffiness of the eyelids and dropsy, the dry skin, and the frequent occurrence of uræmic convulsions distinguish this disease.

(*b*) *Pyæmia* is distinguished by its high swinging temperature, repeated rigors with sweating, the secondary abscesses, and the hay-like odour of the breath.

(*c*) *Pyonephrosis* has already been discussed.

*Prognosis.*—The condition is one of extreme gravity, and is usually fatal within a few weeks (average 2-3 weeks, Morris). Occasionally, however, under treatment the drowsiness disappears, the appetite improves, and the temperature returns to normal.

In some cases of enlarged prostate with abundant residual urine, the infection of the bladder rapidly spreads to the damaged kidneys, and within a few days the patient succumbs; but in most cases the condition is of a more chronic type.

The age of the patient, the previous condition of the kidneys, and the cause of the obstruction (malignant tumours) are important factors in deciding a fatal result.

A dry coated tongue with increasing feebleness and drowsiness are very unfavourable symptoms.

*Treatment.*—Attention to antisepsis in the treatment of stricture, prostatic enlargement, etc., and the early removal of urethral obstruction by operation, have reduced the frequency of this disease.

When infection has occurred no operation is availing or advisable.

The diet should consist mainly of milk, the kidneys should be flushed with Contrexville water, distilled or barley water, and urinary antiseptics should be administered. Dry cupping of the loins may be useful. In more chronic cases the bladder should be washed out with boric acid, quinine, or other antiseptic solution.

### Cysts of the Kidney

*Conglomerate Cysts or Cystic Metamorphosis.*—The condition is a very rare one, only a few

cases have been diagnosed during life (Lindegger). Occurring in the adult it is probably of congenital or inflammatory (Newman) origin, and is almost invariably bilateral. There are usually some symptoms of renal disease.

In one class of case the symptoms have resembled those of chronic interstitial nephritis with polyuria, albuminuria, œdema, and circulatory changes, and sometimes symptoms of uræmia occur.

In another class pain, slight or severe, sometimes resembling renal colic, has drawn attention to the condition.

The discovery of a renal tumour is the only sign which may lead to a diagnosis, and this appears in only 29 per cent of the cases (Lejars). In fifteen out of twenty-two cases collected by Newman a wrong diagnosis was made before operation.

The swelling is almost invariably bilateral (unilateral, one in sixty, Lejars), and this differentiates it from hydatid and simple cysts which it may resemble. Primary malignant tumour of the kidney may be suspected, but in this the tumour is unilateral, and there are no signs of chronic nephritis, while the pain and hæmaturia of a malignant tumour of corresponding size are much more marked. Pyo- and hydro-nephrosis may be excluded by the history, the absence of fluctuation, and repeated examination of the urine.

The condition may last from fifteen (Lejars) to twenty (Senator) years. From its almost constant bilateral distribution treatment by operation is possible only in exceptional cases. The kidney will probably have been explored for severe pain under the impression that a movable kidney or a hydronephrosis is present, and it lies with the surgeon to decide the question of removal.

*Large serous cysts* are usually single, sometimes several are found. A fluctuating tumour is formed, having the character of a renal swelling, sometimes of large size. I have seen and operated on large cysts from both head and tail of the kidney, the evident result of obstruction to some calyx by inflammatory changes induced by stone in the pelvis or ureter. One cyst contained a large amount of crystals of cholesterine. It is diagnosed from an ovarian cyst by its renal characters. From hydronephrosis it is sometimes very difficult to distinguish.

Incision and drainage is the best routine treatment, but the expert will probably prefer to resect and stitch over the pared walls.

### Stone in the Kidney

Renal concretions usually form in the pelvis or the calyces of the kidney from deposition of certain solid constituents of the urine. Occasionally, however, a stone may be found in an isolated cavity in the parenchyma, the result of obstruction to the straight tubes, and of accumulations behind them.

There are three groups of renal stone: the acid (uratic and oxalate of lime stone), the alkaline (lime phosphate), and the bacterial (ammonio-magnesic phosphate). The uric acid is said to be the most usual (80 per cent, Fürbringer).

*Clinical notes on—*

(*a*) *The Size.*—Stones vary in size from that of a fig-seed to a dendritic mass moulded to the pelvis and weighing about two ounces. The heaviest recorded is one by Potel, of five pounds in weight. There is no fixed relation between the size of the stone and the duration of the symptoms, if only the urine remains *acid and sterile*. Pure oxalate of lime stones grow slower than the uratic group. It has taken five, even ten years to produce an oxalate stone the size of the crown of a molar. If the urine contain pus and micro-organisms the size of the stone is roughly commensurate with the duration of that alteration in the urine; for phosphatic material is quickly deposited under these conditions on any material acting as a nucleus.

(*b*) *The Surface.*—Much depends on the surface. Uratic stones are smooth, lime oxalate stones are often covered with minute or large clear crystals of a brownish hue. The smooth, polished uratic variety are most usually multiple, and once in the grasp of the ureter, they pass more readily, and give comparatively less suffering. The latter are more irregular in shape and take longer in transit, induce greater agony, and if unvoidable are often single.

The chief clinical feature of the crystalline surfaced stones is the tendency they exhibit to become *buried*; this is, of course, due to their acicular surface being forced by reflex spasm into the swollen mucous membrane. The favourite burial-ground is near the outlet of the renal pelvis; but adhesion (partial burial) is common in any part of the pelvis from the same mechanical reasons. When fairly in the ureter they may pouch the tube at its commencement or termination, and quietly increase in size without much obstruction to the passage of urine.

Phosphatic stones shift the least of any; they evince a dangerous tendency to grow into and block the pelvic orifice of the ureter.

(*c*) *The Site.*—The early pathological changes induced by the stone mainly depend on whether it leaves the pelvic orifice free or not.

1. *Free Outlet.*—An oxalate may remain buried for years near the pelvic orifice, and yet leave the outlet free, inducing merely a thickening of the pelvic wall and an increase in and a condensation of the fat around it; or a stone of the acid group may be fixed in a deep calyx for years, the outlet remaining free, and the only change induced being an induration of the kidney tissue due to chronic interstitial

nephritis. This is at first localised to the neighbourhood of the irritating body. In course of time most stones evoke inflammatory resentment in the mucous membrane, and pyelitis, pyelonephritis, perinephritis ensue. Eighty per cent of the patients who die from renal calculus do so in consequence of suppuration (Dickinson).

2. *Obstructed Outlet.*—Should, however, the stone be so buried near the orifice, or so situated as to abut upon or periodically to obstruct the outlet of the pelvis and the due egress of the secretion, serious back pressure changes will inevitably result, *e.g.* dilatation of pelvis (hydronephrosis) and atrophy of gland; and if septicity be grafted on these conditions, the destruction of inflammation is severe and serious.

(*d*) *The Remissions, Intermissions, and latent Periods of Stone-Pain.*—To the discredit of the original diagnosis, but to the relief of the patient, the pain of renal stone—like all other diseases of the urinary mucous membrane—is subject to extraordinary remissions. The pain may be absent or hardly noticeable for weeks, months, even years, and this without apparent cause; nay more, the suffering may end entirely.

I have seen patients free for eight, twelve, or fifteen years from any symptom of a stone which had originally caused intolerable suffering; and at the end of these periods pain has recurred and become so violent as to necessitate operation.

The theory is that the stone becomes fixed by adhesions in a hollowed-out calyx, or fixed by branches, or that the kidney may become inactive and even shrivel. It is to be remembered that an inert kidney which is void of all secreting power may still preserve its size and outline, may still be painful or become the seat of pain, may still be liable to inflammatory attacks, and may still pour pus down the ureter into the bladder.

(*e*) *The Pathological Changes in the opposite Kidney.*—It is highly probable that these depend greatly on the character of the calculus. When it is an oxalate of lime calculus the opposite kidney, as a general rule, does not form stone for many years—often not at all. In fact, the excess of depositable oxalate appears to find the easiest exit by the affected kidney, and a readier nidus in the original stone. In the uratic group the same rule holds, but only in a lesser degree. It is not uncommon to find first one kidney and then the other produce and expel a small uric acid stone. When, however, one has formed and become imprisoned, the surplus of uratic material is attracted for some time to that as a nucleus, and the opposite kidney remains free for years. In course of years the opposite gland commences and continues to form uratic stone, so that the operator must be prepared to deal with stone and its consequences in both kidneys when there has been a prolonged history of uric acid urine with symptoms of unilateral renal stone. Fifty per cent of renal stones are bilateral.

In my opinion the real danger appears after the onset of those suppurative changes which the original calculus excites. The healthy opposite kidney is habitually irritated and gradually deteriorated by the stress thrown upon it of eliminating special renal toxins derived by absorption from the suppurating foci of its diseased fellow-gland. In addition to this the healthy kidney is liable to ascending inflammatory changes from a bladder distressed by a constant flow of pyelitic urine. A decrease of functional activity and increased sensitiveness to shock or reflex inhibition is in either case gradually acquired, and this constitutes the greatest element of danger to the patient when colics are suffered from (*cp.* calculous anuria, or when nephrolithotomy is performed). The theory of "reflex nephritis" (Simon), which is accepted to account for these changes, is, in my opinion, untenable as well as unnecessary.

*Etiology.*—Injudicious diet (*e.g.* rhubarb and "hard" water in limestone districts, tending to form lime oxalates), sedentary habits, mental exhaustion, exposure to cold, inherited tendency to gravel, are all powerful factors in the deposition of calculous material.

*Symptomatology.*—There is one symptom—that of pain—which should be considered in detail before the classical symptoms of the complaint are alluded to, for it often affords the medical attendant a valuable clue to prognosis and even treatment.

*Pain.*—There are two main positions and forms of pain:—

The unilateral renal pain, and ureteric spasm (renal colic). They may be observed separately or coexist.

A. *The Unilateral Renal Pain Group.*—The maximum of pain is in the kidney region; the area can be covered by the patient's hand, the pain being both behind and in front—more behind. It may vary from an occasional dull ache, the outcome of mere congestion or irritation of a crystal-charged urine, to that intermittent agony which is induced by a rough surfaced calculus or to that constant suffering produced by a phosphatic-covered stone moulded to an abraded and inflamed pelvis. It is usually moderate, though exacerbated by exercise, jolting, jarring, local pressure, or injudicious diet. During the acute attacks the pain may radiate along the ureter to the groin, testes, or to the thigh, calf of leg, and foot. When moderate the pain can often be covered with the thumb pressed into the angle which the last rib builds with the erector spinæ muscle; any percussion of the spine or succussion of the body will cause in some cases a cutting pain to be felt in this position (Jordan-Lloyd).

It sometimes happens in the acid group that the pain of the calculus may subside altogether

in the kidney, and be felt only in the epididymis or ovary, or calf, or sole of foot. Thus I removed an oxalate of lime stone from the right kidney of a man who complained of incurable neuralgia of the right testis. I operated on learning that severe right renal pain preceded the pain in the testis by two years.

*Influence of Sleep Posture on Renal Pain due to Stone.*—In a certain number of cases, large enough to warrant the symptom being asked for and noted, the pain of renal stone is influenced by the posture of the body in sleep. In a certain percentage of the cases the patient must lie upon the affected side to obtain sleep. To lie on the opposite side induces or increases (by "dragging"?) the pain of the stone. If the renal pain is intermittent this posture is only assumed during the exacerbation. When the kidney has become inflamed the patient often lies on the *other* side, leaving the inflamed kidney free from pressure.

B. *Renal Colic Group.*—It is allowed that the agony of renal colic is due to spasmodic contractions of the renal pelvis and ureter. This is generally induced by the passage of a calculus along the ureter, or by some other foreign body such as a clot, a clump of mucopus, a mass of debris or growth, hydatid or worm. Any substance, in fact, which excites the muscular contractions of the tube, by direct irritation of its inner surface, and by obstruction to the free flow of urine along its channel, will cause more or less severe renal colic. But typical renal colic is not always caused by the transit of voidable stones.

An unvoidable calculus in the pelvis may so abut upon the orifice as to close it and produce a colic; or a small calculus may be so encysted near the pelvic orifice of the ureter as to cause transient swelling sufficient to close the opening and induce renal colic. In both cases the renal colic will be fruitless—no stone will pass—though the symptoms may be as severe as if a stone were in transit.

Renal colic, then, is significant of back pressure and distension of the pelvic cavity—one of the potent factors in the production and accentuation of suppurative changes about a renal calculus.

If, then, attacks of fruitless renal colic are suffered from, the practitioner may be certain that the pelvis is dilating, and on this account the prognosis is graver than one with unilateral renal pain without colic, and I believe operative interference should be undertaken sooner in those with colic than in those without. It must be remembered that a pelvis may dilate quietly without any colic, as occurs in slight bends of the ureteric tube, narrowed vesical orifice to the ureter, and vesico-urethral obstructions.

OTHER SYMPTOMS OF RENAL STONE.—*Hæmaturia.*—Slight hæmaturia after exercise or jolting is the cardinal symptom of renal calculus. It may be absent, or appear only rarely. It may occur without pain—be an early symptom and never recur; it may be the only symptom present. It differs from the hæmaturia of growth in that bleeding from a growth is apt to occur during sleep, at which time the bleeding of calculus, depending as it does on movement, is diminished or quiescent (Dickinson).

*Nausea and vomiting* is usually present during an attack of renal colic, but it occurs also in renal pain; in some instances it is undoubtedly due to reflex irritation, in others it indicates interference with urinary excretion.

*Frequency and imperious desire to urinate* are uncertain symptoms. I believe they often indicate descending waves of pelvic or ureteric congestion.

*Pus*—acid pyuria—more marked on rising; intermittent in quantity—is an evidence of pyelitis, and therefore of great importance in prognosis.

*Motile organisms* in acid pyuria with renal pain, *if no previous instrumentation has been carried out*, are evidences of pelvic infection, but neither pus nor bacteria are characteristic symptoms of stone, though valuable indications of the urgency and dangers of stone.

DIAGNOSIS.—*The following Group of Symptoms arouse the Suspicion of Calculus:*—Fixed renal pain, felt posteriorly, increased by abrupt bodily movement, exercise, or jolting, radiating when severe along the ureter to the groin or testicle, or down the thigh or to the knee or calf. Occasionally slight renal tenderness or deep bimanual palpation, and sharp stabbing pain elicited by percussion or succussion. Attacks of severe renal colic followed now and again by the passage of small calculi. Attacks of hæmaturia observed after jolting; the blood being intimately mixed but nearly always slight and dark in character; inability to sleep except on painful side. Clear urine constantly showing a marked deposit of oxalates or uric acid. These symptoms, extending over a period of three or four years without bladder irritation at night or day, point to calculus in the kidney.

*Radiography of Renal Calculi.*—Every case of doubtful renal calculus should be radiographed if possible. Kidney skiagraphy is, however, still disappointing, for the organs lie in a region of the body having great relative opacity to the rays; and rays of sufficient power to penetrate these parts penetrate the calculi also and leave no shadow. I have removed a calculus which was not detected by the X-rays, but I have seen a sufficient number of accurate diagnoses by its means to warrant the advice given above. It is especially the oxalate and the phosphatic-covered calculi which give the deepest shadow.

*Mimetic Conditions.*—(1) Tubercle of the Kidney.—Primary tuberculosis of the kidney induces renal pain and tenderness on manipula-

tion, and even renal colic in 8 per cent of the cases, but in a less marked degree. The hæmorrhage is, however, much brighter, more dependent on cold than jolting; the urine is murky, lightish in colour, lower in specific gravity, and contains tubercle bacillus and pus corpuscles.

The patient is not forced to sleep on the painful kidney; in fact, the opposite side to it may be selected for the sake of relief. Early pyrexia is not absolutely reliable in the early stages, but it is a valuable indication of destruction and absorption of tuberculous products, and characteristic, if it follows bimanual examination or prolonged exertion. Usually in nine months to a year vesical irritation and penile pain appear consequent upon descending infection.

Renal tubercle consecutive to a vesical source is invariably preceded by irritability of the bladder and meatal pain after micturition.

Renal tubercle consecutive to a primary epididymal tubercule is easily recognised by the knot in the globus minor of the same side, and in most cases by a knot in the corresponding prostatic lobe or seminal vesicle.

(2) Ascending mild septic pyelitis—induced by irrigation of a tuberculous or otherwise inflamed bladder—is a fruitful source of renal pain without colic. It simulates pelvic stone very closely.

(3) Bends of the ureter from movable kidney, narrowing of ureteric tube near the pelvic orifice, have also to be considered. These may give rise not only to renal pain, but also to colic. Frequency of micturition in the day, but not at night, is often marked in these cases.

(4) Interstitial shrinking nephritis produces occasionally unilateral renal pain and hæmorrhage, the pain being apparently capsular and not due to changes in the pelvis.

(5) Stone lodged in the lower ureter near the bladder should be always carefully excluded by rectal and vaginal and vesical examination. A stone lodged in any part of the upper two-thirds of the ureter excites symptoms almost exactly similar to those which a stone in the renal pelvis would evoke, whilst a stone in the lower third simulates ovarian or uterine trouble (Sunderland).

*Treatment* (Medical).—Two main objects are to be aimed at—increasing the volume of urine and diminishing its acidity.

The former is best attained by the patient taking large quantities of rain water, boiled water, distilled water, but better still by the use of such waters as Contrexville, Vittel, Wildungen, Kronenquelle. All these latter are best taken warm, two hours before breakfast, and accompanied by gentle exercise.

If pus is present such drugs as boric acid, benzoate of ammonia, and sandal oil are of use.

In the uric acid type, piperazine, lysidine, urotropine are valuable.

In the oxalate of lime an attempt should be made to fill in and smooth over the sharp crystals which project from the surface (*cp.* clinical note on surface) by over-alkalinising the urine. Lime phosphate is thus deposited on the stone. To this end bicarbonate of soda, the benzoates, and lithia salts should be employed.

The good effects of glycerine are testified to by Hermann, Richter, and Ravaldini, but the reason for its action in expelling gravel is unknown; one or two ounces are given in an equal amount of water twice a day. The remedy is still on its trial.

The treatment of renal colic resolves itself into relieving pain, diminishing ureteric spasm, and increasing the flow of urine. It is asserted that a spasm can be aborted or relieved by tilting the patient on his head and massaging the ureter upwards. The older remedies, however, suffice: hot baths, subcutaneous injection of morphia, inhalation of chloroform. Albarran records a case in which he cut a nephritic colic abruptly short by passing a ureteric catheter and by washing out the pelvis of the kidney.

If the usual treatment for the relief of stone fails to cure the patient within twelve months, the question of operative interference should be considered.

*Advice to Patients concerning Operation.*—On three separate counts may the medical adviser be forced to tender advice to his patient concerning the expediency or necessity of operative interference for supposed renal calculus.

(*a*) The patient may demand relief from pain or from recurrent attacks of colic.

(*b*) The practitioner notices that the urine is changing its sterile nature for puriform or septic characters.

(*c*) Sudden suppression of urine may supervene in a patient with renal calculus symptoms, and immediate relief be urgently needed.

(*a*) Should renal pain be so constant and so severe as to cripple the patient, or should renal colic recur so frequently as to hamper the patient's pursuits, and should therapeutics have failed to relieve within reasonable time, say twelve months, an exploratory operation should be advised. It is hardly necessary to hesitate on the score of uncertainty as to whether stone is, or is not present. In the absence of tubercle and carcinoma the mere exploration of the kidney surface through a loin incision and free separation of the fatty capsule, even if no stone is discovered, will effectually relieve if not cure the patient of the pain induced by kink or bend of ureter, by slight dilatation of the pelvis from narrowing of the pelvic orifice of the ureter and by interstitial shrinking nephritis. This is probably due to fixation of the kidney and straightening and splinting of the ureteric tube

by inflammatory products, and interference with the nerve-supply of the capsule. The mortality in skilled hands is nil. Should, however, the kidney be opened, and calculus be found and removed (lumbar-nephrolithotomy), a cure is effected with a mortality of perhaps 1 or 2 per cent, provided the urine be sterile and the surgeon judiciously gentle with the tissue of the kidney.

(*b*) But should the urine be noticed to be changing from sterile to septic, it is not now so much a question of a demand for the relief of pain on the initiative of the patient, as of the urgent advice of the practitioner for an operation to arrest inflammatory changes in the kidney structure. Here the evident duty and responsibility of the practitioner increases, I submit, in proportion to the frequency and severity of renal colic (*cp.* clinical note C). His arguments are based on the destruction of renal function of one side and the involvement of the opposite kidney (*cp.* clinical note D).

It has been pointed out by Newman, and accepted as axiomatic, that the death-rate of lumbar-nephrolithotomy rises with the presence of suppuration to 39·6 per cent. Moreover, it is to be remembered that suppurative disease from renal calculus has a higher mortality even than suppurative disease of the kidney from other causes. Cases must therefore be attacked in the sterile stage.

Henry Morris's latest statistics are still more convincing, for they represent the work of one operator and not that of the many collected by Newman.

In non-suppurative cases Morris lost 2·9 per cent (1 case in 34) by the lumbar incision, but when suppuration was so advanced as to need nephrotomy or nephrectomy the mortality rose to 25 per cent. I have lost, without regard to aseptic or septic cases, 1 patient out of 50. This low mortality is, however, due in all probability to the change in professional opinion, the outcome of the above teaching, for patients in the last ten years which cover the writer's statistics have been made to realise the paramount necessity for early operation, and have applied for relief before the opposite kidney has deteriorated.

(*c*) When the practitioner is confronted with a case of calculous suppression his duty is obvious and imperative, for calculous anuria is the gravest and most fatal of the many serious complications of renal lithiasis, and it is only in rare instances that the suppression, once established, is overcome. It must be remembered that pain is the best indication both to the side to be relieved by operation and to *the appropriate time for interference.* The tender kidney, which has been the site of the pain at the onset of the suppression, is to be operated on. As long as pain is experienced in the flank or along the ureter the stone may be shifting; directly the pain ceases and suppression continues, the renal vitality is endangered and operation should be considered. Let the advice for interference be urgent, be early, and, in the stage of tolerance, between the third and fifth day (*vide* Calculous Anuria). The percentage of recoveries in cases operated on is 51 per cent as compared with 20·8 per cent which were not operated on, but recovered spontaneously (Morris).

*Suppression of Urine due to Calculus. Calculous Anuria.*—This occurs from the ureter becoming blocked, generally at the opening of the renal pelvis or in its upper third, by a calculus—the other kidney being absent (13·8 per cent, Donnadieu), functionless, or so affected by disease as to be sensitive to reflex inhibition. It is a disease affecting mid-adult life, often occurring in the fat and gouty. There is usually a previous history of repeated renal colic affecting both sides with the subsequent discharge of gravel or stone.

*Onset.*—After a prolonged colic, started perhaps by a sudden jerk, exercise, a fit of temper, or apparently without cause, the patient is seized by a constant desire to urinate, only succeeding, however, in expelling a few drops, and that blood-stained. Then the secretion is completely arrested.

*Stage of Tolerance* (Merklen).—For a variable period, at least five or six days, the patient is in no distress, suffers no pain. He continues his avocation, walks about strongly; but he does not pass water, though he may still have constant desire to do so. Some pass a few ounces daily of pale urine of low specific gravity, if the anuria is not absolute. In favourable cases in this stage the calculus may be passed and the patient recover, or the calculus may become dislodged, drop back into pelvis, and a rush of many pints of urine heralds the probable return of health, though even after this a relapse may take place. Spontaneous cure takes place in 28·5 per cent of cases (Legueu). Usually, however, general debility, sleeplessness, and nausea supervene and usher in the stage of *uræmic intoxication.*

*Stage of Uræmic Intoxication.*—This is usually marked by the appearance of hiccough, vomiting, and intense thirst. As the intoxication deepens, muscular twitchings, pin-pointed pupils, and torpidity appear. Then comes the drop in temperature, irregular pulse and respiration (Cheyne-Stokes), and death between the ninth and twelfth day (or twenty-fifth day).

*Diagnosis.*—The history of former attacks of renal colic; the passage of calculi; the sudden onset of pain in one kidney, or a prolonged colic followed by suppression and accompanied by constant desire to urinate; the presence of a swelling in the renal region, of tenderness there on pressure, or along the ureteric tract; the appearance of a little blood in the small quantity of urine evacuated,—are points of especial

diagnostic importance. Rectal or vaginal examination to determine the condition of the lower ureter is important.

*Treatment.*—The only medical treatment, in the light of the pathology of the disease, is drastic purgation—calomel is especially indicated. Opium should be avoided, and the renal areas may be dry cupped. Operative interference (ureterotomy, pelvotomy, or nephrostomy) must not be delayed; it should be carried out between the third and fifth day.

### Renal Tuberculosis

There is, perhaps, no urinary disease of surgical importance so little understood by the profession at large or so injudiciously treated as urinary tuberculosis. Its initial obscurity, its insidious progress, its power of mimicry, and its extensive, often silent, invasion of adjoining sections of the urinary tract, tend to deceive, to disconcert, and finally to dishearten the practitioner. As likely as not, in well-meant effort to relieve the patient the medical attendant washes out the bladder, and thus unwittingly introduces those septic organisms which exert so baneful an influence upon the progress and amenability of the disease. It cannot be too strongly inculcated that renal tubercle, if it is to afford even a hopeful prognosis, should be, *as it can be*, detected early and treated judiciously.

*Pathology.*—It is admitted that tubercle bacilli detached from any extra-urinary focus may be swept into and collect around the glomeruli of the kidney (Durand Fardel), and even pass out thence and be discovered in the urine without having caused any damage, either to the glands or to their conducting channels. A suitable nidus is therefore necessary for the development of tubercle in the kidney, and this is probably prepared by the deterioration of tissue induced by traumatism, pre-existing inflammation, or transient congestions of the organ.

*Lines of Invasion.*—The kidney may be invaded:—

A. *Primarily* by way of the blood stream (hæmotogenous), or

B. *Consecutively* by way of the ureter from some lower genito-urinary source (urinogenous).

Each class has a characteristic initial macroscopy and a definite initial symptomatology, and the treatment for each should, at least at first, be in accordance with the line of invasion.

A. *Primary Tuberculous Invasion of the Kidney* (*Hæmatogenous Invasion*).—The primary invasion of the kidney assumes one of two different forms—the acute miliary and the chronic caseating.

Acute miliary tuberculosis of the kidney is devoid of interest, for it is the outcome of a general systemic infection arising usually from a deposit in the lungs. It occurs principally in children. It attacks both kidneys, is not usually diagnosable, is quite inoperative and uniformly fatal. Hence the practitioner concerns himself with the second—an important and often amenable class—the chronic caseating form.

A. *Primary Chronic Caseating Renal Tuberculosis.*—The initial stage of this form is nearly always *unilateral*. Its comparative frequency is still debatable; but there is no doubt that disease in this organ is more commonly met with than is generally believed (Israel).

Its microscopy is as follows:—A few miliary tubercles, produced by the irritative action of tubercle bacilli deposited from the blood stream, form in the connective tissue at the junction of the cortex and medulla, or immediately under the mucous membrane of the pelvis. These pass through the usual well-known changes until the final caseous necrosis stage is reached. By the coalescence of these isolated necrotic groups larger areas of disintegration are formed, whence fresh infection spreads outwards to the cortex or inwards towards the pelvic mucous membrane. Instead of an initial shower of tubercle bacilli producing discrete nodules, a single thrombus of tuberculous material may become lodged in and block a small renal vessel. The plug softens and invades the surrounding area to induce like changes. No matter what or where the anatomical starting-point of the deposit, the final shape, size, and destruction of the kidney depends upon the attitude of the tuberculous mischief towards the mucous membrane of the *orifice* of the pelvis. Early narrowing or occlusion of this opening leads to pyonephrosis and rapid destruction; whilst patency, by permitting the discharge of tuberculous debris and urine, allows the gland to become gradually impaired, the capsule to be invaded, even the capsule and fatty envelope to be enormously thickened and cartilaginous, without septic suppurative changes taking place. As the former condition is marked by colics, and the latter by fixed renal pain, an important clue to the prognosis is obtained by noting the extent and the character of the initial suffering.

B. *Ascending Renal Tuberculosis, consecutive to lower Genito-urinary Tuberculosis. Tuberculous Pyelonephritis.*—It is an open question whether there is not in all cases of invasion from a lower urinary source an ascending wave of simple ureteritis prior to those anatomical conditions which are recognised as tuberculous ureteritis In a *genital* invasion the kidney may be affected without the bladder being involved. Be this as it may, the earliest appearance of infection of the kidney from a lower source is a tuberculous change in the lower calyces of the kidney. If the invasion has ascended via the ureter, it attacks the papillæ of the lower third of the kidney; if it has short circuited from the epididymis via the lymphatics

of the vas to the lymphatics of the ureter, the submucous layer of the lower calyces seems most often affected. The parenchyma of the kidney is then consecutively invaded by progressive extension along the lymphatics and vessels running towards the cortex from the lower papilla. Once started, the changes which ensue, the gradual erosion of the pelvic mucous membrane, and the hollowing out of the parenchyma of the gland, resemble those which are noticeable in primary renal tuberculosis.

It is, of course, to be understood that tuberculous disease in the neighbourhood of the kidney may involve that organ by direct continuity. Thus in psoas abscess, in rare instances, the ureter may be perforated and the disease extend upwards to the kidney and downwards to the bladder. I have met with two such cases on the post-mortem table. In still rarer instances an empyema may perforate the diaphragm, surround and involve the kidney. Such cases of infection by continuity are, however, too uncommon to merit more than this—a passing reference.

Clinical Notes based on Pathological Conditions.—1. *Extent of Renal Tissue involved.*—Primary renal tuberculosis is at first unilateral. This is a rule upon which the practitioner may usually depend. Hence in the earliest stage the disease is often sharply localised and therefore removable by operation.

There is no symptom or group of symptoms by means of which one can accurately gauge the extent of destruction of the kidney tissue. One can only assume that when renal colics are a marked feature—that is, when a narrowed outlet exists — the destruction is greater and more rapid than when the ureter is sufficiently patent to admit of the escape of urine and debris. Should the ureter become suddenly and permanently blocked in the earlier stages before septic infection, the kidney may first swell and then gradually shrink; under such conditions the kidney power is destroyed, and the opposite kidney takes on the renal function. Should, however, the kidney become blocked after septic infection, the kidney must become pyonephrotic, and the collection of pus, urine, and debris, if unrelieved, will perforate the capsule and form a perinephritic abscess of an especially destructive character. Hence attention is especially directed to a cross-examination for the symptom of renal colic. Additional evidence is obtained by a bimanual examination of the size of the kidney and in the aspect of the urine whether it is clear (blocked ureter) or murky (open ureter).

2. *The Rules of the Spread of Renal Tubercle.* — Primary renal tuberculosis, as has been stated, is usually unilateral (80 per cent, Albarran). One organ is affected to begin with. As the disease spreads and cripples the secretory power, the fellow-gland becomes hypertrophied compensatorily. In the ascending invasion the same rule holds; one ureter, its pelvis, and its kidney is attacked before the other, the ureter and pelvis being generally inflamed before the tubercle develops in it.

If the invasion be from a primary bladder source there is no clinical rule as to which kidney will become affected, though it is likely that any known pre-existing inflammation of that gland will serve to locate the disease.

There is, however, a rule as regards the side first affected in invasion from a *genital* source. That side on which the disease starts is first affected in 80 per cent of the cases. Thus, right epididymal tubercle, or right-sided vesico-prostatic tubercle, is followed by right-sided renal tubercle.

3. *The Stress Resistance of the Fellow-Kidney.* —The excretion of the toxins of primary renal tuberculosis is carried on by the opposite healthy gland. A very gradual but distinct deterioration in the renal function is noticeable, and it is supposed that an interstitial nephritis gradually results (Albarran). As the tissue degenerates, it becomes very vulnerable to ascending waves of inflammation from the bladder, which must become involved in tuberculous processes descending from the original focus. If the action of the fellow-kidney is not cut short by suppression, as so often happens, it becomes, in its turn, the seat of destructive tubercle.

4. *The Chronology of the Disease.* — Like tubercle in other regions, renal tubercle obeys no law of progress. Much depends upon the active or torpid character of the initial deposit, upon the suitability of the nidus, upon the part of the kidney first invaded (the parenchymatous deposit probably developing slower than one near the pelvic mucous membrane); upon the condition of the pelvic orifice of the ureter, upon the incursion of septic microbes from the bladder or gut, and, finally, upon the inherited resistance, nourishment, and hygienic surroundings of the patient.

Dissemination is specially retarded by *early* and permanent occlusion of the ureter, and by the formation of a thick fibroid envelope around the kidney, induced by leakage of irritating material through the cortex. This condition merits a passing allusion. When the tuberculous process assumes a chronic type the perirenal fat becomes invaded, and infiltrated, and sclerosed, and the diseased kidney is finally imprisoned in a dense cartilaginous material. At the same time the intimate connection of this armour with the vessels and surrounding viscera renders any attempt at dissection hazardous in the extreme. In such a case subcapsular nephrectomy is indicated. (*Cp.* Operations on the Kidney.)

In the larger number of cases the disease in the kidney has extended to other parts before

the third year, though from a variety of causes it may remain torpid or only progress very slowly—possessing a life history extending over ten or fifteen years.

I submit that a slender basis in the estimate of the duration or rate of progress of the disease is afforded by the clinical aspect of the hæmaturias. Bursts of sharp hæmaturias are indications of torpidity.

5. *On the Macroscopy of Renal Tubercle.*—The final shape and size of the diseased gland is irrespective of the character of the invasion. But there is much clinical evidence to be obtained in determining the character of the tumour.

(*a*) *It may be unfeelable.* This happens when the disease is in its earliest stage, or in its latest obsolescent stage. In either case a kidney may be so small and fixed so high up under the ribs as not only to be unfeelable, but to be also insensient to pressure. This occurs both in primary and consecutive renal tuberculosis if the disease is very chronic, if the upper part of the cortex is inflamed, and if the ureter is open. Unfeelable kidneys are rarely, accompanied by pyrexia.

(*b*) It may form a definite, smooth, but irregular, *movable tumour.* This type is usually the result of primary tuberculosis with more or less intermittent occlusion of the ureter, forming slight pyonephrosis. The temperature is in this case usually slightly raised (99° F.) at night. If septic pyelitis coexists the temperature rises much higher (101° F.). The tumour is tender in proportion to the inflammation. Very rarely is a huge tumour tuberculous throughout; when such exists it is called "tuberculose massive" (Monti).

(*c*) It may present itself as a *large fixed mass* in the loin. Such a condition occurs usually as a leakage through the cortex, leading to enormous thickening and matting of the peri-adipotic capsule, or even perinephritis (*cp.* end of clinical note 4).

It is of importance for the surgeon to remember that the kidney may appear to the eye and be to the touch *absolutely healthy*, and yet be extensively ulcerated by tuberculosis at either extremity of the pelvis.

6. *Clinical Note on Morbid Additions to the Urine. Formation and Passage of "Dirt" Stones.*—True renal calculi of the acid type (*vide* Renal Calculi) are *extremely* rare, but it is not uncommon to find phosphatic grit, or phosphatic scale-like concretions in a tuberculous pelvis or even lining the entire ureter. These may be the result of ulcer crusts, or even be phosphatic material which has become deposited upon scraps of debris. To find a single phosphatic cast of the pelvis is very uncommon. These "dirt" scale-stones add to the pain, the hæmorrhage, and the ureteric obstruction. They may cause colic, but their form, if they are evacuated, is distinctive and should not mislead. Radiography cannot differentiate such dirt scale-stones from phosphatic-covered culculi.

7. *Clinical Note on the Detection of the Bacillus Tuberculosis.*—The presence of the tubercle bacillus in clear, sterile urine is not pathognomonic of urinary tuberculosis, for the bacilli have been found in the urine of those who were suffering from phthisis, or from tuberculous bone or joint affection. If, however, they are found in the pyuric urine of those who complain of urinary symptoms, there is but little doubt of their having "effected a destructive lodgment in some part of the urinary tract."

Much stress is laid upon the similarity of tubercle bacillus to the smegma bacillus, and many urge that the specimens examined should be obtained by aseptic catheterism. It is held by some that there is a special grouping of the bacilli in renal tuberculosis—that in this disease they become massed into groups resembling the letter S.

It is, I believe, an impression, neither proved as yet nor accepted, that tubercle bacilli are more easily and more abundantly found in renal than in vesical tuberculosis even before putrefactive bacteria have contaminated the urine. When the urine is decomposing in the bladder from septic cystitis, tubercle bacilli are not found. It is generally noticed that once the bladder has been subjected to a course of irrigation tubercle bacilli are not found, or only discovered with difficulty.

The same statement may be made for urine examined soon after a course of injections of Koch's new tuberculin.

8. *Clinical Note on the Addition of Septic Microbes.*—The especial danger to the patient lies in the introduction of septic microbes. This takes place in some cases from contagion with the colon, but in many it is, I am certain, the outcome of injudicious interference with the bladder, this interference taking the form of careless irrigation, rough sounding, unskilful cystoscopy, and the like.

It cannot be too strongly insisted on, that the practitioner can do infinitely more harm than good in the majority of cases of urinary tuberculosis by washing out the bladder, for the septicity which is thus introduced ascends by way of a weakly resisting ureteric mucous membrane to the pelvis, and destroys the renal-secreting tissue of that organ very rapidly. I state most emphatically that a kidney affected by tuberculosis is ruined more quickly by bladder washing than by the destructive action of the tubercle. Every careful practitioner will obtain, if possible, a bacteriological report of the urine of a young adult patient, who has a causeless mild cystitis, before he irrigates the bladder as a curative measure for the inflammation.

Symptomatology.—A. *Symptoms of Primary Renal Tuberculosis.*—In a small proportion of cases the disease commences and progresses to the entire destruction of the gland, without evoking any marked symptoms; but this is rare, and there is usually certain pronounced symptoms which may lead one to suspect primary renal tuberculosis. These symptoms are renal pain coexisting with pale, feebly acid or neutral murky urine of low specific gravity, and occasional hæmaturial attacks. There may not be, and frequently is not, at first that anæmia, that rapid emaciation, and that elevated evening temperature, upon which physicians place so much reliance. These symptoms appear later in the disease, it is true, but not at first, unless the renal tubercle has become affected by septicity from the bladder; or as some writers assert, unless the parenchyma of the gland is much destroyed without any implication of the pelvic mucous membrane. The cases range themselves in two distinct classes: (*a*) those with fixed renal pain; (*b*) those with renal colic.

(*a*) *Tuberculous Kidney evoking fixed Renal Pain and early Pyuria.*—This class is much more commonly met with (80 per cent). The symptoms seem to depend on the destruction of the pelvic mucous membrane and renal structure, the urine and debris escaping freely along the open channel of the ureter. Although there may be some thickening of the walls of this tube from ureteritis, yet the channel is wide enough to carry off the secretion without exciting renal colic as a general rule, though occasionally a clump of mucus or debris may be caught, and may give rise to a sharp ureteric twinge or even a colic. The pain is at first slight, intermittent, disappearing for weeks, but reappearing in a more severe form until it becomes constant. It is chiefly felt behind, over the lower ribs; it is coverable with the palm of the hand (not the thumb, as so often appears to be the case in oxalate of lime stone).

After a few months the patient becomes liable to transient attacks of frequency of micturition of greater or less severity, and meatal pain after the act. These attacks will vary in duration, last from a few hours to a few days. They may be due to the caustic action of the urine or to transient waves of descending pyelitis: probably the former, for it is conceivable that now and again ptomaines from the ulceration or other chemical substances produced by the disintegration of the tuberculous processes are added to the secretion which, passing over the sensitive neck of the bladder, evoke temporary dysuria.

As months pass the renal pain ceases, but coincident with its subsidence appear those symptoms which are characteristic of the disease having effected a permanent lodgment in the bladder: habitual frequency of micturition, diurnal or nocturnal; glans or meatal pain after the act, and occasional slight hæmorrhages.

When the bladder has become definitely ulcerated there is a "posture" symptom of some value which may be present in women. When the ureteric orifice has become ulcerated the patient cannot sleep on that side at night, for this position aggravates the irritability of the bladder. The patient, therefore, sleeps on the side opposite to that of the diseased ureter.

This brief sketch delineates the usual course in primary renal tubercle, but it must be remembered that exceptionally the disease in the kidney is more or less "latent," and it is only when the bladder becomes affected by descending changes that any symptoms appear. Moreover, these symptoms are referable to the *bladder*, and the disease is supposed to be primary there because the symptoms first complained of can be located there. The cystoscope alone detects these latent cases, for by its means the ureteric orifice of the diseased kidney is shown to be ulcerated, or patulous, or displaced by tuberculous changes.

(*b*) *Tuberculous Kidney evoking Renal Colic.*—Primary tubercle of the pelvis of the kidney may, in the minority of cases (20 per cent), produce a renal colic almost exactly like that of renal stone, and this almost from the onset of the disease. The first symptom may be a renal colic, and this may continue on and off until the kidney has given up secreting urine. The colic is due to the narrowing of the pelvic orifice of the ureter and to thickening of the ureteric wall. It is surprising how thick the ureter can become; some are the thickness and solidity of thumbs or forefingers, and on section a tiny circle represents all that is left of the ureteric channel. I venture to suggest that in such cases there is an inherited tendency to *fibroid* phthisis. I believe that when such kidneys are shut off *early* by occlusion of the ureter, the lymphatic trunks of the channel become plugged and the disease is walled in. Anyway the patients who have *early* occlusion seem to have a longer lease of life and to be the most favourable for cure by nephrectomy.

Other Symptoms of Primary Renal Tuberculosis.—(*a*) *Formation of Tumour.*—It is generally accepted that primary renal tuberculosis rapidly transforms the kidney, and so enlarges it as to cause a definite tumour to be formed. This is inaccurate. It does enlarge it, but as often as not no renal tumour can be felt in men; and in women, who are of laxer habit, the renal swelling can only be discovered with difficulty. When a renal tumour is found it indicates great destruction of the kidney, either by progressive infiltration, or by pelvic dilatation from a narrowed orifice. The practitioner, however, has to decide as to whether the kidney which he finds enlarged is a tuberculous kidney or one compensatorily

hypertrophied. This is generally decided by the history of pain. An hypertrophied kidney has an uneasy ache, but a tuberculous organ generally causes decided suffering. Moreover, the cystoscopic appearances of the vesical orifices of the ureters differ, as I have just mentioned. The ureteric catheter settles the point, for by it secretion from the kidney is obtained direct.

(*b*) *The Hæmaturia of Primary Renal Tuberculosis.*—Attacks of profuse hæmaturia from the kidney may antedate the characteristic symptoms of primary renal tubercle for months —even years. The practitioner is sometimes unable to locate it without the use of the cystoscope, for it may occur without any guiding symptoms as to its source; but this is unusual, for some renal pain and tenderness is generally present. The attacks are apparently causeless. As often as not tubercle bacilli cannot be found, for the amount of blood in the sample renders the detection of the bacillus difficult if not impossible; whilst in many instances the disease has not really broken into the pelvis, and the debris, containing the bacilli is not yet free. A small collection of crude tubercle under the mucous membrane of the pelvis may suddenly evoke *localised* pelvitis and extravasation of blood, and this patch, small as its area may be, is sufficient to provoke a very arterial hæmaturia for a few hours. But these attacks are nearly always as transitory as they are alarming.

It is only later, when the disease has eroded the mucous membrane and opened the venous plexuses near the papillæ, that the bleeding becomes intractable. In the intervals of these later hæmorrhages the bacillus is usually found in the urine without difficulty, and the secretion has the ordinary characteristics of tuberculous urine.

(*c*) *Polyuria.*—Some stress is laid upon the fact that the patient may pass large amounts of urine prior to the development of renal symptoms of tuberculosis, and this symptom is ascribed to the irritation of parenchymatous deposits of tubercle. It needs much circumspection before allowing this symptom to influence the diagnosis, for polyuria is often a transient feature in the course of many renal diseases.

(*d*) *Morbid Changes in the Urine.*—The urine of pronounced renal tubercle is characteristic. It is light in colour, murky with mucus, depositing a fine layer of pus and a few caseous clumps. It is always albuminous. It is faintly acid or neutral, and of medium specific gravity. Bacilli are discoverable, and if the urine is injected into guinea-pigs subcutaneously, a typical tuberculosis is produced in a fortnight or three weeks.

Later, as the disease advances, putrefactive bacteria cause an offensive odour, and muco-pus in large quantities is passed; pus increases in proportion to the grade of pyelitis.

B. Symptomatology of Consecutive Renal Tuberculosis.— *The Ascending Form.*—The kidney in many cases is invaded by tuberculous changes which originate in the lower genito-urinary organs, and under these circumstances there are always pronounced symptoms attending the site of origin. If the bladder be the primary site there is the history of frequent micturition, especially at night; pain after the act, at the meatus urinarius or glans penis, suprapubic pain on over-distension and slight hæmaturia; or if the epididymis has harboured the primary focus, a history of causeless abscess or thickening of that body is always ascertainable.

The practitioner cannot be too exacting in cross-examination for these symptoms, for the line of treatment to be adopted depends upon whether the form be primary or consecutive implication of the kidney.

*Diagnosis.*—The renal conditions which resemble renal tubercle fall into two groups, the *early* appearance of pus in the urine serving, although roughly, to mark the division.

The First Group.—Calculus of the kidney, movable kidney, and renal tumours rarely produce puriform urine in their earlier stages, whilst pus appears very early in renal tubercle.

From the second group, which includes septic interstitial nephritis, septic pyelitis, and pyonephrosis, the tuberculous disease of the kidney is separated by the fact that it produces marked night frequency and other vesical symptoms. Moreover, from both groups it can in course of time be distinguished in the male by the inevitable progress and invasion of the genital organs; for a tuberculous deposit can always be discovered as the disease advances, in the epididymis of the same side, or the vesiculæ or prostate. The great element in accurate diagnosis is the discovery of the bacillus in the urine. The cystoscope, if skilfully handled, is often of prime importance in detecting which kidney is affected (*q.v.*).

*Prognosis.*—The prognosis in unilateral primary renal tuberculosis is very grave; in the ascending form it is almost hopeless. Concerning the former it may be said that occasionally it obsolesces, as in fact tubercle can and does in other parts of the urinary tract. Evidences of this natural cure are occasionally found on the post-mortem table, but it is a consummation that is probably rare. It is possible for a tuberculous deposit in the kidney to slough out and come away by the ureter with the urine, but it is not common.

The favourable cases are those in which the ureter becomes choked early in the course of the disease, and before septic material has had access to the pelvis, in which case the secretion of urine ceases, the kidney contracts and re-

mains quiescent, the fellow-gland doing the work of the body.

The useless kidney is, however, liable to recrudescence on the intercurrence of some debilitating fever such as epidemic influenza. Even a cold, a wrench of the body, or a blow on the loin, will start the disease afresh.

*Treatment.*—It will be remembered that the disease in the kidney, when primary, remains localised for months; and it is in this stage that the chance for operative interference curing the disease is greatest. It is in this period that the acumen and judgment of the practitioner is of such vital importance to the future well-being of his patient. I cordially agree with the justice of Newman's remark that the practitioner who makes an early diagnosis in a case of primary renal tuberculosis renders a service to the patient as valuable as that of the surgeon, who at a later date performs a successful nephrotomy or nephrectomy.

SERUM THERAPY.—(*a*) In the "fixed renal pain" group.

*If tubercle has been found* and there are no symptoms of bladder irritation, a course of Koch's new tuberculin should be tried, on the chance that the disease is limited, and that it may become so affected by the injection as to break down and pass by the natural channel, for we are quite unable to say how much of the kidney is affected. A course of six injections is cautiously given, one every third day into the thigh, the skin being first carefully cleansed with soap, carbolic lotion, and finally ether—a Luer syringe is the best form of instrument to employ. The initial dose is $\frac{1}{250}$ mg., and the strength is gradually increased thus: $\frac{1}{250}$, $\frac{1}{200}$, $\frac{1}{150}$, $\frac{1}{100}$, $\frac{1}{50}$, 1 mg. The injections are suspended if fever or renal pain develops.

If tubercle cannot be found in the urine it is of paramount importance, in cases where the family history of phthisis is marked, to cystoscope and examine the ureteric orifice and its immediate neighbourhood.

(*b*) In the "renal colic" group.

In treating primary renal tuberculosis marked by renal colic, I object to the use of Koch's tuberculin, for I hold that the renal colic is absolute evidence of the tuberculous invasion and thickening of the ureteric channel, and the swelling in the deposit in the wall of the tube caused by Koch's tuberculin is quite sufficient to block the tiny channel which remains. The debris and broken-down tissue which is released in the kidney cannot pass, and is therefore retained. Swelling of the organ ensues and extension of the disease follows, for the thick inflammatory wall which the disease has already constructed around the dangerous foci is overstretched and perforated. It is probably better in the renal colic class to nephrectomise immediately after cystoscopy of the ureteric orifice.

*Treatment of Ascending Renal Tuberculosis.*—The treatment of ascending renal tuberculosis depends largely upon the cystoscope. An examination of the ureteric orifices under the electric light determines if the kidney is affected, and which, and how far the ureter is involved, also whether the bladder is so far ulcerated as to render remediable measures of value. If the ulceration in the bladder has not advanced to the muscle area, if it is limited to patches in the postero-superior wall, if one kidney is decidedly affected, I nephrectomise the diseased gland, taking away as much of the ureter as possible, and after the patient is healed I treat the bladder by means of Koch's new tuberculin. But I hold it a doubtful expedient to use serum therapy in the ascending form of renal tuberculosis until the kidney has been removed.

*Medicinal.*—Neither the fixed pain nor the renal colics are severe as a general rule; both are amenable to opiates. The administration of small doses of sandal oil in capsule relieves the pyelitis; hexamethylentetramine (gr. v.) or boric acid, ammoniac benzoate, or salol, keep the urine as sterile as is possible. Creasote occasionally seems beneficial. Methylene blue in pill is worthy of a trial.

When the disease has reached the bladder its distressing effects must be combated on the principles laid down for cystitis, the practitioner bearing in mind the intolerance of the urethra to instrumentation and the resentment of the tuberculous bladder to irrigation. Strict hygiene, a generous diet, with fat and sugar, if these articles are well borne by the digestion; a high and dry climate, if obtainable—are all that can be done to alleviate and arrest the spread of the disease. Care should always be taken to impress upon the patient the necessity for adding carbolic acid to the chamber utensil in order to prevent the spread of the disease. The following experiment endorses the wisdom of such a proceeding. Five rabbits were confined in a box and made to breathe vapour from an atomiser charged with the urine of two *phthisical* patients. After a couple of months all the animals were found markedly tuberculous.

*Operative.*—For primary renal tuberculosis.

Operative.—Two courses are open. If, on exploration, the disease is limited to a single cavity or to a small area, nephrotomy may be performed and the entire disease curetted out. Against this is the great uncertainty as to how far the disease has involved the gland, and the danger of allowing tuberculous material to foul the operation wound and the exposed perirenal tissue.

Nephrectomy or nephro-ureterectomy is the operation which must be performed in most cases, even when taken early; by it the disease is removed absolutely, and with it the greater part of the ureter; the remainder of this tube being curetted or energetically dealt with.

The same operation is advocated for suitable cases of ascending renal tuberculosis.

*Advice to Patients concerning Operation.*—Advice to the patient anent operation in renal tuberculosis should rest finally and entirely with the operating surgeon. Upon his judgment must the necessity, the advisability, and the danger of nephrectomy depend. His must be the consideration of the exact stage which the disease has reached; his the determination of the stress resistance of the opposite kidney. This being made clear to the patient, the following statistics will be of value in discussing the consideration of operation.

Bolton Bangs of New York has collected cases from various sources. They are selected since 1888, for the means of diagnosis have been so improved since then by the perfection of the cystoscope and the catheterisation of the ureters, that statistics taken prior to that date are misleading.

135 cases are quoted. The immediate operative mortality was 20 per cent.

*Immediate Mortality.*

| | | |
|---|---|---|
| Uræmia | 11 | cases (42 per cent). |
| Various accidents | 11 | ,, |
| Exhaustion | 2 | ,, |
| Extension Tuberculosis | 3 | ,, |
| | 27 | out of 135 = $\frac{1}{5}$ or 20 per cent. |

*Later Mortality.*

| | |
|---|---|
| Died within nine months | 13 cases. |
| Survived and fairly well up to nine months | 31 ,, |
| Survived one to eight years | 45 ,, |

Half the cases, then, were very favourable, some living as long as eight years. Dr Bangs remarks:—

"The first and undoubted conclusion which these statistics warrant is that the immediate result of the operation for renal tuberculosis in the cases in which it is indicated is brilliant. Many cases which seemed *in extremis* and liable to speedy death from hectic hyperpyrexia, pain, etc., were immediately relieved, their existence made tolerably comfortable, and their lives prolonged. A clear and positive conclusion of the *remote* results is exceedingly difficult to reach; still I think the opinion based upon such statistics as I have been able to get is warranted that operation affords better remote results than hygiene."

### Hydatid Cysts

Hydatid cysts are comparatively rarely met with in the kidney, forming only 5 per cent to 8 per cent of all hydatid disease (Davaine, Neisser).

The cyst is usually limited to one kidney (98 per cent, Béraud), often the left, and starts in the cortex. As it increases in size it projects from the surface of the organ and encroaches upon the renal pelvis. It frequently bursts into that cavity (82 per cent of the cases, Roberts). In size it may vary from that of an egg to that of a man's head. When feelable it forms a rounded, painless, elastic tumour in the hypogastrium—often irregular because of the multilocular nature of the cyst. If the contents have not suppurated spontaneously the swelling is movable; if the cyst has inflamed the tumour becomes fixed. Occasionally the cyst wall is transformed into a semiosseous envelope, and in this condition it is densely hard, and affords a striking but misleading skiagraph on radiography. Adhesions are nearly always contracted with neighbouring viscera, but they vary greatly in density. In one case in which I had to operate for the relief of intestinal obstruction, I found a kinked colon so adherent to the fixed bony case of a small unsuspected renal hydatid that I could not dissect it off without opening the gut.

When the cyst calcifies and dies, the contents are transformed into putty-like material which is composed of fat, cholesterine, and laminated membrane.

*Symptoms* may be entirely absent, and a renal swelling discovered by accident, the patient's attention being drawn to the loin by a dull aching in that region. The first characteristic symptom is produced by the bursting of the cyst into the pelvis, and the passage of the hydatids or membranes in the shape of grapes or grape skins. Urticaria has been observed to follow rupture into the ureter (Mosler).

Usually a rigor, accompanied by all the symptoms of renal colic, precedes the first attack, but the ureter soon enlarges and isolated cysts are passed without much suffering, unless collection enters the pelvis and blocks ureter. The cysts vary from the size pigeon's egg to that of a pea, but the are ruptured and are passed as mere coll sacs.

It is astonishing for how many years attacks continue. One of my patients st that he had thus suffered for thirty-one ye another that he had noticed the renal tum for thirty years, but had only passed the cy for five years.

Suppuration sometimes takes place in the tumour, I suspect, as a consequence of its intimate adherence to the colon, and instead of sterile urine, a thin, gruel-like urine, laden with pus, broken cysts, and large fragments of laminated membrane, is passed. I have treated a case of renal hydatid which discharged along the ureter for thirty years before suppurating.

*Diagnosis.*—The presence of an elastic renal tumour in conjunction with attacks of renal colic of that side and the passage of an hydatid cyst, either whole or collapsed, is, of course,

absolute indication of a renal hydatid. It has been pointed out that hydatid cysts may be passed per urethram from a collection situated behind the bladder, and bursting into that viscus, but this chance may be disregarded. Only three unequivocal cases are on record in the literature of the last two hundred years. Moreover, if such a collection exists it is discoverable by rectal examination.

The diagnosis of renal hydatids based upon an elastic, uneven tumour in the hypogastric region may be established by aspiration of fluid with hooklets in it from the loin; but this method of investigation is not without risk, for it is liable to induce suppuration.

*Operative Treatment.*—It used to be urged that these cysts, enjoying as they seem to do long periods of quiescence (ten, twenty, thirty years), need not be interfered with as long as the suffering of the patient is not great, and no rapid increase in size is noticed. It is, however, to be remembered that they gradually contract adhesions with the gut, and these may give rise to obstruction by kinking the bowel; or the cyst may suppurate spontaneously. In every case also the pressure of the enlarging cysts acts injuriously on the renal structure, and it is wiser to operate early in order to anticipate such untoward events. It is seldom that nephrectomy will be needed. Laying bare the surface of the cyst, incising it, stitching the fibrous capsule to the wound, evacuating the contents, and peeling off the mother cyst, is the operation of choice. It is, I think, better to drain the cavity, but this may not be always necessary. If the cyst is already suppurating free drainage is absolutely requisite.

Fifteen cases treated in this way have resulted in fifteen successes, while nephrectomy as been done five times with four deaths Barran).

HYDRONEPHROSIS

tial or intermittent obstruction in the ry passages causes dilatation of the renal s and kidney, and if the sac which is formed ains urine the condition is called hydrohrosis. There are two forms, congenital and uired.

CONGENITAL HYDRONEPHROSIS needs but a rief reference.

In 38 per cent of all cases of hydronephrosis some congenital defect is present (Roberts), but the term congenital hydronephrosis is applied to the form present at birth or soon after birth.

The condition is due to deformity of the ureter (impervious, contracted, twisted, or kinked ureter), or to the pressure of abnormal renal vessels, or to some abnormality in the urethra. It is sometimes present at birth and gives rise to difficult labour. If the child survives birth it is usually fatal in a few months or years (Newman).

Congenital hydronephrosis may be unilateral or bilateral, and other congenital defects, such as harelip, are often present.

ACQUIRED HYDRONEPHROSIS.—*Etiology.*—(1) Ureteric Causes.—Calculus impacted in the ureter or stricture of the ureter following its passage (in 59 per cent, Roberts). Contraction of the ureter following injury, pressure on the ureter by pelvic tumours (27 per cent, Newman), by pelvic scar tissue from inflammation or sometimes displacements of the uterus, or pregnancy. (In thirty-six autopsies on females recently delivered, Olshausen found hydronephrosis twenty-five times.)

Kinking of the ureter in a movable kidney.

(2) and (3) Any vesical or urethral impediment to the flow of urine.

*Pathological Anatomy.*—There is dilatation of the renal pelvis, flattening of the papillæ, and gradual destruction and disappearance of the renal tissue until only a fibrous sac remains. Sometimes it attains enormous dimensions. The largest on record is one which held thirty gallons of fluid, while the abdomen measured 6 feet 4 inches in circumference (Glass). The sac is subdivided by septa into loculi which open into the greatly dilated pelvis. The walls may be thick and fibrous, sometimes they are very thin. In some cases a varying amount of renal tissue remains. The cyst contains water with a varying amount of sodium chloride and traces of urea. Sometimes mucus and epithelium are present. With the destruction of one kidney the other hypertrophies and performs the entire urinary function.

*Symptoms.*—Tumour.—Often the only sign of hydronephrosis is a large fluctuating tumour situated in the loin. In 59 per cent of cases there is a palpable tumour (Roberts).

The loin sometimes bulges, and renal "ballottement" may be obtained by placing one hand on the abdomen and projecting the tumour forward with the other in the loin. The swelling is rounded on all sides, and descends slightly with respiration. On percussion dulness extends to the spine posteriorly, while in front the tympanitic note of the colon is obtained, and in a tumour of moderate size a resonant note can be obtained separating it from the liver on the right and the spleen on the left.

Pain.—The tumour, as a rule, is painless and no tenderness is present; often there may be slight aching, but both these symptoms depend on the rapidity with which the hydronephrosis grows.

There is rarely *hæmaturia*, and the *urine* is normal in quantity and quality.

INTERMITTENT HYDRONEPHROSIS.—In a certain number of cases the tumour suddenly disappears, and coincident with this there is a temporary marked polyuria of urine of a low specific gravity. After a time the tumour gradually

reappears and again evacuates itself. Re-accumulation of the fluid is often preceded by an attack of renal colic.

Bilateral Hydronephrosis occurs when there is incomplete obstruction of the urethra; sometimes the cause is in the bladder, rarely in both ureters.

If during the course of an urethral stricture a constant polyuria is present with diminution in the specific gravity of the urine, commencing dilatation of the kidneys may be suspected. There is often some albumin in the urine and tube-casts may be present. There is not usually sufficient enlargement of the kidneys to be felt on palpation of the abdomen, and there is no pain or tenderness.

These cases often have transient attacks of suppression of urine in the later stages, and eventually die of uræmia.

Diagnosis.—(1) Of the renal tumour. (2) Of the nature of the renal tumour.

(1) *The Renal Tumour.*—The history of renal colic, hæmaturia, pyuria, or other urinary symptoms, if present, points to the renal origin of the swelling.

Mimetic Conditions.—(*a*) *Tumours of the Liver and Gall-Bladder.*—These grow from above downwards, while kidney tumours pass from the loin forwards.

They move more freely with respiration than do renal tumours. The dulness is continuous with that of the liver, and there is never a tympanitic note in front. Sometimes a history of jaundice or of biliary colic may be obtained.

(*b*) *Tumours of the spleen* occupy a higher position, are more freely movable with respiration, the whole tumour is dull on percussion, and there may be a well-defined edge and a typical notch. General symptoms of malaria, leukæmia, etc. are frequently present.

(*c*) *Ovarian cysts* grow from the pelvis upwards; if large enough to simulate a renal tumour they are median, dull on percussion, and both loins are resonant. The tumour is more movable than a renal one, and vaginal examination shows a displaced uterus and a fluctuating pelvic tumour. There is no urinary history, but often one of menstrual disturbances.

(*d*) *Ascites* may be simulated by a very large lax hydronephrosis. There is dulness in both flanks, which shifts with the varying position of the patient, and a percussion thrill may be obtained.

(2) *The Nature of the Renal Tumour.*—*Hydatid and serous cysts* also form painless, fluctuating tumours of the kidney. They are comparatively rare conditions.

In the case of hydatids there may be a history of renal colic from the passage of small cysts. The discovery of one of the causes of hydronephrosis, such as an ureteric stone felt per rectum, or the history of passage of gravel or stone, are of importance.

*Pyonephrosis.*—The swelling in these cases is often tender and frequently painful. There is usually a history of pus in the urine and rigors, and fever will indicate the purulent nature of the contents.

*Treatment.*—Medical treatment is unavailing. Operative measures, such as drainage or plastic pelvectomy, or removal of the kidney, should be advised in all but the smallest hydronephroses. The following points are the most important in considering the question of operation:—

(1) The patient may seek relief from the pressure effects of a very large hydronephrosis, especially shortness of breath, palpitation, constipation, or vomiting.

(2) In bilateral cases the destruction of kidney tissue is progressive, and eventually the issue is certainly fatal. Patients with hydronephrosis seldom live beyond the age of fifty (Dickinson). Death occurs from suppression and uræmia.

(3) In unilateral hydronephrosis the causal condition (most frequently stone) often becomes bilateral, and the remaining kidney is destroyed.

(4) Rupture of the hydronephrotic sac (spontaneous or from injury) may occur with fatal results.

(5) At any time suppuration may occur in the sac by the use of septic catheters or in other ways; and the condition is much graver than in simple hydronephrosis. This is well shown by the cases published by Henry Morris. In ten operations for hydronephrosis he had no deaths; in nine operations for pyonephrosis three patients died.

In cases due to the pressure of malignant tumours—cancer of the uterus, for instance—operation is, of course, contraindicated; but the question will seldom be raised, for in such cases a renal swelling is rarely discovered (Morris).

### Pyonephrosis

Pyonephrosis is a term used to denote dilatation of the kidney and its pelvis, with suppuration superadded. The dilatation may occur first and sepsis be added, either from septic catheterisation or some operation, or no such cause may be present, and infection come through the blood stream. In other cases the ureter becomes blocked in the course of a calculous pyelitis and pus accumulates.

*Pathological Anatomy.*—The structure of the sac is the same as in hydronephrosis, but the lining membrane is roughened, shreddy, and the contents purulent. Frequently there is phosphatic deposit on the walls, and calculi may be present if the condition has developed from calculous pyelitis. If one kidney alone is affected the cause will be found in the renal pelvis, and is almost invariably calculous. Both kidneys are often dilated, and these cases arise from obstruction in the bladder or in front

of it. In such chronic cystitis is present, and the ureters are dilated and thickened.

The development of a primary pyonephrosis is more rapid than that of a hydronephrosis (Morris), and also more complete.

*Condition of the other Kidney.*—It is seldom quite normal even in unilateral pyonephrosis, and may be congested and inflamed, or the seat of waxy disease (in 56·4 per cent of cases some disease is present, Tuffier). When the condition follows enlarged prostate, stricture, etc., one kidney is always more damaged than the other, and the least injured organ shows a varying degree of suppuration and dilatation. In these cases the more healthy kidney unfortunately does not hypertrophy, and suppression of urine is liable to occur.

*Symptomatology.*—Pyonephrosis is merely a stage in the progress of various urinary maladies. The swelling is in the lumbar region; if large it bulges backwards as well as forwards, the surface is usually lobulated, it moves slightly with respiration, ballottement can be obtained. In consistence it is firm, sometimes fluctuating. It is dull on percussion, except in front, where the colonic note can be obtained. It has, in fact, the characters of any renal tumour, and is distinguished from swellings of other organs by the same means as a hydronephrosis.

It has certain special characteristics. It varies in size at different times; when the pus in the urine diminishes the tumour increases, and *vice versâ*. It is usually accompanied by general symptoms of septic absorption.

Pain is seldom completely absent. It is usually a dull lumbar aching, and is liable to exacerbations when the pus is retained and the tumour enlarges. Sometimes it strays along the line of the ureter. Some patients suffer a good deal, and especially after exercise, and renal colic with rigors may occur. Pain is increased by pressure in front, but often relieved by pressure posteriorly (White and Martin). Tenderness may be felt along the line of the ureter (Albarran).

*The Urine.*—When pyonephrosis is due to "ascending" changes the urine is always cloudy, alkaline, has a thick deposit of muco-pus and an ammoniacal odour. When the pyonephrosis is "open" the urine contains a large quantity of pus. A deposit of pus, representing about one-fifth to one-sixth of the total liquid, is never produced by a bladder lesion alone. The relieving flow of pus after an attack of retention in the pyonephrosis is often enormous. In unilateral pyonephrosis pus may be constantly present in the urine ("open"); it may be intermittent or completely absent ("closed"). The urine in these latter cases is faintly acid, sometimes alkaline. During an attack of retention in a unilateral pyonephrosis the urine is merely the secretion of the other kidney, and valuable information may then be obtained of the working capacity of that organ.

*General Symptoms.*—There is general debility, loss of weight, the appetite is poor, and the patient suffers from indigestion. Sickness and diarrhœa are sometimes present, and the skin acquires a yellowish sallow tinge. Fever is present in most cases; sometimes it is slight, and if prolonged will become hectic; in other cases it is more acute.

When suppuration occurs in a hydronephrosis the first sign will probably be a rise of temperature, with shivering.

Two types of pyonephrosis will be met with.

(1) Where the bladder is free from disease and the pyonephrosis is unilateral. These are mostly cases of stone in the kidney or ureter.

(2) Where cystitis is present, usually with some obstruction. Here both kidneys are affected in varying degree, and the cause is in or anterior to the bladder.

*Clinical Types.*—(1) Pyonephrosis in a case of renal calculus.

In a case of calculous pyelitis the pus in the urine may diminish or disappear entirely, and the urine become clear. At the same time the patient complains of aching or increased pain in the diseased loin, some fever appears, and there is a loss of appetite and general feeling of debility. On examination of the loin a tumour is felt and there is marked tenderness. The block may remain, but often after a time the pus suddenly reappears in large quantity in the urine, the pain and fever disappear, and the tumour can no longer be felt. This may be repeated from time to time.

(2) In a case of long-standing cystitis with obstruction the urine is turbid, alkaline, and ammoniacal, with abundant deposit of slimy pus. Without any noticeable change in the urine fever appears and some aching in the loins. The patient loses flesh, his appetite is poor, and his skin sallow. There is tenderness along the ureter on one or both sides, and in the loin, and sometimes a tumour may be felt. Here there is probably retention of pus in the kidney pelvis, but the symptoms may be due to a suppurative pyelonephritis. In the latter there is seldom a tumour, the fever and general symptoms are more rapid and severe, and the amount of pus in the urine is usually less than in pyonephrosis.

Diagnosis.—(*a*) *Hydronephrosis.*—The tumour is not tender and seldom painful, there is no pus in the urine, and no symptoms of septic absorption.

(*b*) *Tuberculous disease of the kidney* is differentiated by the cheesy character of the urinary deposit, the presence of tubercle bacilli in the urine, the hæmaturia, and in the male the descending invasion of tuberculous disease along the urinary tract. Often there is the history and evidence of pre-existing tubercle elsewhere.

(*c*) *Purulent collections* opening into the

bladder may cause intermittent pyuria. The cystoscope is the best means of diagnosis.

TREATMENT.—*Medical.*—The indications are to support the strength by tonics and to administer urinary antiseptics, such as boric acid, ammonium benzoate, and salol.

The operative treatment of the two classes described is different. In the "ascending" form the cause (stricture, etc.) must be attacked, the residual urine got rid of, and the cystitis arrested; in the other class the interference is direct, and the kidney is opened and drained, or, if need be, removed. Operation is to be recommended as early as possible on the following grounds:—

(1) In unilateral pyonephrosis the other kidney may become involved by development of stone or waxy disease resulting from septic absorption.

(2) Septicæmia or pyæmia sometimes occurs, and hectic fever often results from long-continued disease.

(3) Rupture of the sac may occur with rapidly fatal results.

(4) Suppression of urine and uræmia are prone to occur.

### KIDNEY TUMOURS

Benign tumours hardly merit reference. Fibromata, lipomata, and adenomata have been encountered, but so rarely that no clinical picture can be formed for them as yet. Papillomata of the mucous membrane of the pelvis, unconnected with malignant growth, is a very unusual disease, the literature containing only eight examples. They do not increase the size of the kidney, but give rise to hæmaturia of the painless type.

Primary malignant disease of the kidney may be considered to comprise three groups of tumour-forming growth:—

1. Sarcomata.
2. Carcinomata.
3. Malignant transformation of accessory adrenals (suprarenal "rests").

PATHOLOGY.—*Sarcomata* are met with before the age of five and after thirty. The microscopy at these ages differs. The tumour of childhood is often largely composed of striped muscle fasciculi, that of the adult is comparatively free of this tissue. The sarcoma of childhood is apparently congenital, bilateral (50 per cent), and attains a greater relative and even absolute size than in the adult.

*Carcinomata* are probably rarer than sarcomata; they are met with after the age of forty-five, and originate either in the cortex or in the mucous membrane of the pelvis, the latter being a rare site.

*Malignant accessory Adrenals.*—Grawitz has demonstrated that tumours composed of suprarenal elements (epithelium and zona pigmentosa) develop in the cortex or immediately under the capsule of the kidney: ordinarily they are no larger than a cherry-stone. These suprarenal "rests" many become malignant in adult life, sometimes forming very large tumours. They are extremely vascular, and this, together with the fatty material always present, produces a characteristic red-yellow appearance. They form secondary deposits (20 in 28 cases, Lubarsch).

SYMPTOMATOLOGY.—*In the Child.*—The growth is generally rapid and sometimes colossal; indeed, Osler remarks that very large (solid) abdominal tumours in children are nearly always renal or retroperitoneal sarcomata. They are usually symptomless except towards the last, when pressure symptoms occur. (Hæmaturia in 25 per cent, Newman.)

*In the Adult.*—There are two salient features: (*a*) the appearance of a solid irregular renal tumour—palpable and visible; (*b*) the occurrence of a causeless, usually painless, intermittent, *profuse* hæmaturia. The tumour may be first discovered (in 63 per cent of the cases), or the hæmorrhage may be the onset symptom (in 37 per cent of the cases). If these two features are present together they indicate neoplasm of the kidney.

*Life-History.*—1. Latent Period.—The first state is symptomless unless it be an occasional ache or drag in one loin. The growth in the parenchyma is either spreading towards and stretching the capsule to form a tumour (63 per cent), or it is pressing on and invading the mucous membrane of the renal pelvis to cause hæmaturia (37 per cent).

2. Period of Onset Symptoms.—If the growth spreads outwards to form a tumour, and is so rapid as to distend the capsule quickly, the pain may be a marked symptom. The pain area may cover the entire loin and hypochondriac region, and referred pain may shoot along the distribution of the nerves to the hip, thigh, leg, and even testicle. If the growth only increases slowly it may cause no pain. The increase in either case is generally forwards to the peritoneal cavity; then, as its weight increases, it tends downwards; on the right it tends to pass completely to the outside of the ascending colon; on the left it is habitually crossed by the upper part of the descending colon (Stimson). The kidney may be sensitive in carcinoma, but not in sarcoma (Thornton). If the growth tends to invade the pelvis rather than the capsule, the hæmaturial onset is as follows:—

The patient, apparently in good health, suddenly experiences a difficulty in starting the act of micturition; with an effort a clot is shot out, and then a quantity of blood and urine follows. There may be a suggestion of slight indirect violence as a cause for the blood, such as lifting a heavy weight, or sitting heavily down on a chair lower than was anticipated. Such slight violence lacerates a soft knot of growth fungating into the renal pelvis. If

clots are not a marked feature at the onset the hæmorrhage at first ceases quietly. If clots are abundant at the onset or in the course of the case, the bleeding ceases *abruptly*, and a pain varying from a little uneasiness to distinct localised *transient* lumbar pain is experienced in the affected kidney, or even clot colic (renal) may be noticed. *Cystoscopy* will show a long black or decolorised gray clot distending and hanging from the corresponding ureteric orifice. With the expulsion of this corking clot, which is like a worm, the bleeding recommences. Occasionally clot retention is suffered from, and the catheter is necessary. Cancer clumps are rarely found in the clot or urine.

Pressure symptoms now arise from mere bulk of tumour, such as sudden varicocele, œdema of one extremity, or of the abdominal wall. Even ascites may ensue.

Period of Dissemination.—Sooner or later the capsule of the kidney gives way; and, coincident with the loss of this barrier, diffused and increased pain is often noticeable, loss of flesh is a marked feature; anorexia and cachexia supervene.

*Diagnosis* is often impossible without direct inspection through a loin or abdominal incision, but the presence of a renal tumour with severe hæmaturia is most suspicious of growth. If the diagnosis has to be made upon the clinical grounds of an irregular tumour in the renal region, renal calculus with chronic perinephritis and tuberculosis have to be excluded. Other tumours simulate renal growth. On the left side there is the enlarged spleen; but this is readily distinguished from renal growth by its distinct edge, its notch or notches often, by a murmur, or a pulsation, by its creaking fremitus, and the microscopic character of the patient's blood (*vide* "Blood," vol. i.). Moreover, the gut lies behind it. Hepatic growths are occasionally confusing, but they lack that resonant zone which exists between the upper margin of a renal tumour and the ribs. It must be remembered, however, that in the later stages when a renal growth fuses with the liver tactile differentiation is impossible. Malignant degeneration of a movable kidney sometimes resembles cancer of the ovary, but it possesses a free *upward* mobility at first, and pelvic examination is negative.

*Data for Advice to the Patient and Friends.*—In the Child.—Statistics of recovery after nephrectomy for the sarcoma of childhood hardly justify an operation. The only chance of success in the adult is early detection and early nephrectomy.

Due stress should be laid upon the following unfavourable symptoms: extensive adhesions, immobility of tumour; extreme thirst; pressure symptoms, other than varicocele; wasting; cachexia unaccounted for by hæmorrhage; loss of sulphocyanide in the saliva.

The operative mortality is between 50 per cent (Guilleman) and 42 per cent (Barth), but individual surgeons have varying success—that of 16·6 per cent being the lowest (Schede, Israel).

In my opinion and work the cases in which nephrectomy is most hopeful are those in which hæmaturia is the onset symptom—those who are operated upon directly the profuse hæmaturia appears. These are cystoscopy cases. Thus I have removed kidneys in which the growth was the size of a small monkey-nut, a walnut, a small fig, having detected the side from which the profuse hæmorrhage was issuing by means of cystoscopy.

Treatment of the Hæmaturia.—*Instrumental.*—It is unwise to sound any profuse symptomless hæmaturia. Nor is it good practice to wash out the bladder unless clot retention necessitates this procedure. Judicious investigation of those cases without renal tumour consists in administering Contrexeville water until the urine is blood free, and then examining with the cystoscope with the bladder full of clear urine. This determines the absence of any *vesical cause* of the hæmorrhage. The next step consists in recystoscoping during an attack of hæmorrhage; the origin of the blood is thus detected by watching the ureteric efflux. The surgeon should *at once* proceed to explore and remove the kidney which is bleeding. Nephrotomy may become necessary in a few cases merely to relieve the agonising pain due to tension of a rapidly growing hæmorrhagic neoplasm. The relief of the incision into the mass is great, but rapid death (seven days) ensues from septicity.

*Drugs.*—It is better to avoid drugs which tend to arrest hæmorrhage by increasing the clotting power of the blood, for this generally leads to aggravation of the distress by inducing clot colic or clot retention. Hot Contrexeville water, taken fasting, or a large dose of potash, is worthy of a trial. It is always to be borne in mind that if the case is inoperative, gentle hæmorrhage relieves pain and lessens the term of life.

When pain is a marked feature reliance should be placed upon opiates, and no anxiety need be felt about their action on the opposite kidney.

### Actinomycosis of the Kidney

Actinomycosis may invade the kidney under two conditions.

(1) *The Metastatic Form.*—This resembles pyæmia in its acute fever, rigors, and secondary abscesses. When a primary lesion is present it is usually about the face or mouth.

(2) *The Consecutive Form.*—This consists in an extension from the intestinal canal, usually the cæcum or appendix (Hinglass); 18 in 40 cases of abdominal actinomycosis affect the cæcum or appendix (Grill).

In the first variety the deposit is in the kidney substance, and will probably remain unrecognised; in the second a perinephritic abscess of very chronic type is formed. It is also said to occur as a primary condition (Fischer). The condition is very rare indeed, and the kidney is less liable to invasion than the lower urinary tract (Ruhrah).

*Diagnosis.*—The special characteristics of actinomycosis are its very chronic course, great infiltration, marked tendency to invade the skin and form sinuses, with a peculiar red-violet colour of the integument, and the discharge of pus containing "sulphur granules."

The condition most resembling actinomycosis is tubercular inflammation, but the above characteristics may sufficiently differentiate it.

In 75 per cent of cases some occupation in which constant contact with straw or grain (coachman, farmer, field-labourer, miller) is followed by the patient (Leith).

"Sulphur granules" may be found in the urine when the urinary tract is invaded (Billröth).

*Treatment.*—Medical.—Potassium iodide is a specific for the disease, the results of continued administration being very satisfactory.

*Local.*—Abscesses are opened and scraped and sinuses dressed.

### The Ureter

The ureter extends from the renal pelvis to the bladder, running in a kind of lymph-space between the laminæ of the subperitoneal tissue. Its average length is twelve inches, and diameter when distended a sixth of an inch. It is conveniently divided into abdominal pelvic and vesical portions. The abdominal portion lies on the psoas and genito-crural nerve, and under the peritoneum. On the right side it has the inferior vena cava almost in contact with it internally, and on the left the aorta internally.

The pelvic portion crosses the sacro-iliac synchondrosis, the obturator internus, and then turns below the psoas to enter the bladder.

In this latter position in the male it is crossed superiorly and internally by the vas deferens, and lies under cover of the free extremity of the vesicula seminalis, separated from its fellow by a distance of an inch and a half. In the female it runs parallel with, and four to six lines from, the servix uteri—behind the urine artery; finally crossing the upper third of the vagina to reach the vesico-vaginal interspace, and pierce the bladder opposite the middle of the vagina (Anderson).

1. *Injury.*—Rupture of the ureter from violence without an external wound is exceedingly rare, and the symptoms it gives rise to are not characteristic.

After blows on the abdomen or loin, or crushes, the symptoms of damage to other organs will probably claim attention, and the ureteric injury may pass unnoticed. Pain and tenderness on pressure in the line of the ureter are the only symptoms referable to the ureter. At first hæmaturia may occur, or it may be entirely wanting. It may be marked or slight. Neither does the fact of it being absent after a severe injury to the loin show that the ureter has been torn across. The first reliable sign is the appearance of a swelling in the loin, of a rounded or oval contour, some time after the accident; it is formed by retroperitoneal accumulation of urine, for the serous membrane is unlikely to be ruptured coincidently.

If a rigor now occurs, suppuration may be presumed to have taken place in the sac, and the case may run an acute course with fatal issue, or may gradually approach the surface as an abscess, and finally discharge, leaving a urinary sinus. Suppuration does not always result, however, and a cicatrix may form in the ureter, which narrows or entirely obliterates the lumen of the tube. Under these circumstances a hydro- or a pyo-nephrotic swelling is likely to result, but in some cases complete atrophy of the kidney has been demonstrated.

Months or even years may elapse before the dilated kidney is discovered, and at this distance the original injury is apt to be overlooked. If the peritoneum be ruptured at the time of accident, peritonitis rapidly leads to a fatal result.

*Treatment.*—Operative interference should be advised, when from the appearance of a swelling the diagnosis of ruptured ureter becomes probable. It will usually be limited to incision and drainage. Afterwards an attempt may be made to restore the calibre of the ureter.

2. *Inflammation and Dilatation.*—When the back pressure of prostatic or other urethral obstruction begins to affect the ureter, the only symptom which may evidence the fact is pain before micturition and relieved by the act. The pain corresponds to a point internal to the mid-Poupart line, and on a line with the anterior superior iliac spine; sometimes the external abdominal ring is the seat of pain. By the use of the cystoscope further evidence may be obtained, for the mouth of the ureter usually shows signs of dilatation.

Inflammation of the ureter (ureteritis) either ascends from the bladder or descends from the renal pelvis. The patient complains of intermittent pain along the course of the ureter, and that tube may sometimes be felt as a tender cord on abdominal, vaginal, or rectal palpation.

No treatment is specially directed towards the ureteric affection, for it is the same as that of pyelitis.

### Stone in the Ureter

The majority of renal calculi either remain in the renal pelvis or pass quickly through the

ureter and drop into the bladder; in some cases, however, the stone, although small enough to enter the ureter, is too large safely to traverse the normal tube, or is arrested by some abnormal stricture or valve.

Clinical Notes.—1. *On the Ureter in Relation to Stone.*—There are three narrow points in the normal ureter at which a stone is likely to be arrested.

(1) At its upper end or just below this (4-7 cm.).

(2) At the point where it crosses the iliac artery and rounds the brim of the pelvis. (Narrowed in three out of five cases, Kelly.)

(3) Where the tube passes into the thick muscular wall of the bladder.

At any one of these points a "migratory" calculus may be stopped; most often, however, the tube is blocked at the upper end (66 per cent, Morris), less frequently at the lower end (17·8 per cent), or at the pelvic brim (12·5 per cent). It is supposed that in many cases calculi have already passed along the ureter, and the damage caused by their rough crystalline surface has been followed by scarring and constriction of the tube, and a stone small enough to pass the natural danger points is arrested at the stricture. The calculus may completely block the ureter, but it may only partially obstruct the passage, and at the level of the stone the wall becomes pouched to form a bed for it. The calculus may be found lying loose within the ureteric pouch, and may by further deposit increase in size. I have generally found *acid* stones in these pouches. The phosphatic calculi tend to fill and block the ureteric channel.

At the lower end of the ureter a calculus, when impacted, may slightly bulge the bladder wall; but it sometimes projects through the ureteric opening into the vesical cavity, and can be seen with the cystoscope.

2. *On the Stone.*—Usually a single stone, rounded, ovoid, or oblong in shape and of small size, is present; sometimes several are found. A considerable size may be reached by fresh deposits occurring, and a long sinuous calculus sometimes results. Stones projecting from the lower ureteric orifice often attain a remarkable shape, with a vesical cap, narrow neck, and thicker ureteric stem (Bishop's, Zuckerkandl's cases). At the upper end of the ureter they often resemble a nail with the head lying in the pelvis (Albarran).

3. *On the Examination of the Ureter.*—The means which are at the disposal of the practitioner for investigating the condition of the ureter are few but simple, and should never be neglected.

Palpation of the abdominal wall in the line of the ureter—a vertical line from the junction of the inner and middle thirds of Poupart's ligament—should be systematically carried out. The spot where it crosses the pelvic brim, at the intersection of a horizontal line between the anterior iliac spines with a vertical one from the pubic spine (Tourneur), sometimes shows a point of special tenderness. If the abdominal walls are thin and the ureter enlarged the tube itself is stated by Fenger to be recognisable. I can only state that it is extremely difficult to find the ureter, unless it is tuberculous, even when the finger is introduced through a parietal wound.

Pain and intense desire to micturate are evoked by pressure on a diseased ureter. Kelly says: "It is not sufficiently appreciated that a very important section of the ureter may be explored from the rectum by the finger when the patient is in the knee-elbow position, in either sex as high as the iliac artery, and I have on several occasions detected stone in the ureter by this method. A stone may be overlooked by not carrying the finger as high as the perineum will permit. In the female the ureter can be palpated by vaginal examination, from its vesical termination as far as the broad ligament, and can be rolled beneath the finger."

*Symptomatology.*—A stone may lie in the ureter without completely blocking it, and give rise to no symptoms (Albarran); but in the majority of patients in whom a calculus becomes arrested in the ureter, there is a history of previous attacks of "renal" colic and other symptoms of renal stone, so that little doubt exists of the calculous nature of the case.

Instead of a sudden relief from an attack of renal colic indicative of the "migrating" stone dropping into the bladder or falling back into the renal pelvis, the suffering of the patient whose calculus remains in the ureter only slowly declines, and the clinical course of the case will now be either acute or chronic, depending on the completeness of the obstruction and the state of the other kidney.

The more acute course is that of calculous anuria, already discussed, and it only remains to consider the chronic cases where the block in the ureter is incomplete and the fellow-gland sufficiently healthy to maintain the secretion.

These cases differ according to the situation of the calculus. If at the middle or upper part of the ureter, there is nothing which will serve to distinguish them before operation from calculus in the renal pelvis or calyces.

The initial colic subsides gradually, and often recurs from time to time with less intensity than on the first occasion. The pain is sometimes referred to one particular spot in the line of the ureter, and may be fixed and constant between the attacks of "renal" colic (Le Dentu), and in some cases a tender ureter may be discovered on palpation, which, if constant, has some localising value (White and Martin). In exceptional cases dilatation of the ureter above the impacted stone has been felt. In one case

a ureteric calculus was recognised through the abdominal wall (Fenger).

These signs will, in the majority of cases, be absent, and it is only after months or years, when the kidney is explored for stone or a swelling appears with the characters of a hydronephrosis or a pyonephrosis, and is exposed by the surgeon, that the situation of the calculus is ascertained.

A stone situated at the lower end of the ureter may give rise to symptoms in no way differing from one in the higher parts of the tube, but many of the cases are sufficiently distinct to lead to a certain diagnosis.

In a case where signs of renal calculus were present, constant and marked pain in the lower part of the abdomen has drawn attention to the situation of a stone in the lower ureter; but more striking are the cases where symptoms have pointed to stone in the bladder.

There is marked and frequent desire to micturate, with straining and the discharge of small quantities of urine, pain at the end of the act often referred along the urethra or to the glans penis. Some hæmaturia may be observed, often recurrent, and the urine may contain besides red cells and leucocytes, oxalate or uric acid crystals.

The effect of movement and vibration is not, however, so marked as is usually the case in vesical stone, for walking and driving may have little effect in increasing the symptoms.

The effect of posture is sometimes striking, for the suffering may be more intense on standing or sitting, especially with an empty bladder (Zuckerkandl's case), or the patient may be unable to lie on the affected side (Bishop and Fenwick's cases). On passing a sound in these cases a sensation of a "soft membrane" covering some hard substance may be detected (Morris's case), or the metal may ring clearly on a projecting part of the stone, and the fixity and constant position will be noticed.

On rectal examination a hard mass is usually felt lying in close relation to the bladder wall and tender to the touch, and on vaginal examination a similar body may be felt in the region of the broad ligament. In some cases the finger in the rectum has detected a calculus where none was felt from the vagina. The cystoscope in one case showed the lower end of the ureter to be proptosed and covered with a small villus tuft (Fenwick).

*Diagnosis.*—Mimetic conditions.

*Renal Lithiasis.*—A calculus lying in the middle or upper portion of the ureter cannot be differentiated from one in the renal pelvis or calyces.

*Vesical Calculus.*—A stone in the lower end of the ureter may closely resemble vesical calculus. The means of diagnosis are by rectal or vaginal examination of the ureter and the information gained by the sound or cystoscope.

*Primary Tubercle in the Vesicula Seminalis.*—This must be guarded against by a careful analysis of the urine for bacillus and by rectal examination.

*Ovarian and Tubal Disease.*—Long-continued pelvic pain with radiations and increase during the menstrual congestion, together with a tender swelling in one fornix on vaginal examination, due to calculus impacted in the lower ureter, has led to removal of the ovaries and appendages from erroneous diagnosis.

A careful examination of the ureter by vaginal examination and of the ureteric orifice by cystoscopy, and attention to the history, should prevent such mistakes.

*Additional Aids to Diagnosis.*—In some cases a radiograph has been taken of the pelvis, and evidence of the extravesical site of the calculus has been obtained (Leonard, Zuckerkandl).

The cystoscope and the catheterising cystoscope may give useful information in a calculus situated low down.

The passage of a wax-tipped bougie along the ureter in women may, on withdrawal, give the evidence of scratches produced by the calculus (Kelly).

*Advice to Patients about Operative Treatment.*—The only treatment of any avail is removal, and this should be urged as soon as possible before the kidney has become deteriorated by blockage and ascending inflammatory changes. One danger which is not sufficiently emphasised is perforation of the ureter above the calculous block, and consequent extravasation. I operated on such a case lately, the calculus being four inches below the kidney and the perforation was one inch from the pelvic orifice of the ureter.

It should, I submit, be a general operative rule that if the stone be found in the upper third of the ureter, the ordinary lumbar incision should be employed; if at the pelvic brim, the common iliac artery incision suffices; if below the pelvic brim, the incision should be perineal or vaginal. Several cases have lately been recorded in which the abdominal incision for tying the common iliac artery has been followed, and, the peritoneum having been raised, the lower end of the ureter has been reached extraperitoneally. There is no doubt that this incision is of value when one has to remove the ureter in its entire length, or to extract stones which have become impacted in that canal at the pelvic brim or *about that level*; but I question if it is not unnecessarily severe when the stone has become lodged *below the pelvic brim.*

### Operative Procedures

*Methods of Exposing the Kidney.*—The patient lies upon the sound side with a hard pillow beneath the loin. Many incisions have at various times been suggested and practised in laying bare the kidney. There are two chief

methods, the lumbar or extraperitoneal, and the abdominal or transperitoneal. Without entering into the discussion on the merits of these methods, it may be said that the latter is now almost universally confined to those cases where a tumour of such size is present that removal by the loin becomes impracticable from want of space, and that such include only large renal growths. The technique is that of other abdominal operations, the incision being made through the linea semilunaris, or in such manner as to give freest access.

The lumbar incision most usually adopted is an oblique one from the angle between the last rib and the erector spinæ muscle, or a little below this and passing downwards and forwards towards the anterior superior iliac spine. The length of this incision varies with the extent of the operation. In an exploration of the kidney, or a nephrolithotomy, a small incision will often suffice; for the operation of nephrectomy more room will be required, and for the thorough examination of the ureter or its removal the incision should be prolonged down and forward, passing about an inch in front of the anterior superior iliac spine, and continued if necessary as far as the internal abdominal ring (Morris). In dividing the muscles the quadratus lumborum need not be incised. The further steps are described under the different operations.

With the object of avoiding the danger of a lumbar hernia Mayo Robson has applied M'Burney's method of treating the abdominal wall by incising the muscles in the direction of their fibres, and freely retracting them. The space for manipulation is, of course, curtailed, but often suffices for exploration or nephrolithotomy, and its advantages are great.

*Nephropexy.*—Since Hahn in 1881 introduced the operation of fixing a wandering kidney, many methods have been tried; some have been found trustworthy. The operation on a floating kidney differs from that on a movable one, in the fact that the peritoneal cavity must be opened, but in all other points the procedure is similar.

The kidney is approached by the usual lumbar incision, and its fatty capsule exposed. The hand of an assistant guides the organ into its natural position, and the perinephritic fat, which is often atrophied, sometimes even wanting, is incised and torn through until the capsule proper of the kidney is laid bare.

Albarran considers it of great importance to remove as much fat as possible, so as to allow the kidney to lie upon the muscular wall, and in this manœuvre the fingers or forceps are used. The kidney is now very carefully examined and palpated to ascertain whether other conditions, such as stone, tubercle, etc., are present.

Sutures, usually three in number, are then passed, at intervals of about half an inch, through the kidney substance, and on each side include the fatty capsule, the transversalis fascia, and the muscles. These are tied firmly, and the superficial wound closed with or without drainage.

The patient is kept recumbent for three or four weeks, and thereafter wears a belt for some months.

Some authors consider it necessary to lay bare an area of kidney substance by splitting the capsule and turning back flaps, or by removing a portion of it (Tuffier, Jacobson, Kocher). Thick catgut may be used as suture material; but this is rapidly absorbed, and the part of the suture within the kidney substance is said to be more quickly destroyed than that without (Newman). Kangaroo tendon has also been used, but sterilised silk sutures are perhaps the best of any. Vulliet uses a strand of the erector spinæ tendon torn from its upper attachment, but remaining attached below; this he passes through the muscles into the lumbar wound, beneath the kidney capsule, and back through the muscles again. The method is ingenious, although somewhat complicated, and is at present on probation. The living suture is said to have sloughed out, but this is an unusual occurrence.

To ensure fixation of the kidney, Guyon, Albarran, and others pass the upper suture round the lowest rib, while others promote granulation and subsequent increased cicatrisation of the wound by inserting a large drainage-tube or by packing with gauze.

*Nephrolithotomy.*—In this operation the kidney is exposed by the oblique lumbar incision, and the finger introduced into the wound is passed at once to the pelvis, which is carefully palpated for stone. No concretion being discovered, the anterior and then the posterior surface of the kidney is examined.

Should no stone be revealed the lumbar incision is now extended downwards and forwards, and the kidney freed from its surroundings and drawn out into the wound, *or merely into the wound* if the pedicle is short and inelastic. A further careful palpation between the finger and thumb may now discover a spot of increased resistance, but failing this the organ is incised along its convex border, the left finger and thumb of the surgeon meanwhile controlling the bleeding by pressure on the renal pedicle. The finger is now introduced through the kidney substance into the pelvis, and a further search, if necessary, is made by introducing a small metal sound, the upper and lower calyces receiving especial attention.

No stone having been revealed by these measures, a bougie is passed down the ureter to ascertain its permeability; nor should this precaution be neglected after the discovery and removal of a renal or pelvic stone.

The stone is removed, if possible, entire by means of the forefinger, aided, if need be, by

fine forceps or scoop; but sometimes it is necessary to break up a large mass before it can be delivered from the renal wound.

A stone after being removed from the pelvis should always be examined very carefully for chipped surfaces, for there is a very real danger of leaving a portion of a branched calculus behind; and for the same reason it is well to flush out the pelvis with a copious stream of aseptic solution.

After removal of the concretion the kidney wound is closed by catgut sutures, and the lumbar wound closed except for a couple of drains retained in place for a few days.

Some authorities prefer to open the pelvis (pyelotomy) rather than cut through the kidney substance. The advantages claimed are the rapidity and ease of the operation, and that the kidney is spared the after-effects of an incision through its parenchyma. For small stones in the pelvis discovered immediately by the finger the method is undoubtedly of service, but there is much less space for manipulation and greater difficulty in removing a large calculus, and especially if it is branched, and there is a greater probability of a sinus resulting (Albarran). Morris and Israel recommend the closure of the pelvic wound by Lembert's sutures, by which means the danger of a persisting sinus is lessened; the application of sutures, however, is often very difficult (Güterbock).

The patient is usually able to be up three weeks after the operation.

*Nephrotomy* consists in incising a kidney which has been more or less destroyed by suppuration or back pressure, or both, or which is the seat of a cyst. The incision in no way differs from that described in other kidney operations.

In cutting on the kidney the tissues are likely to be œdematous and excessively vascular, and the perinephritic fat infiltrated and often increased in amount, while sometimes pus is discovered surrounding the kidney.

On reaching the distended kidney an incision is made into it and the finger introduced. A calculus should be searched for in cases of hydro- and pyo-nephrosis, cheesy material removed in tubercular kidney, and the mortar-like phosphatic material sometimes found in dilated kidneys, scraped and washed away. A large double drain should be placed in the kidney sac.

Sometimes it is possible to stitch up the kidney wound, even after operation upon a suppurating kidney, but in most cases this is undesirable.

*Nephrectomy.*—A kidney may be removed by an abdominal or a lumbar incision, the relative values of which have already been noticed.

The kidney having been exposed, adhesions to the surrounding parts should be separated. This is often exceedingly difficult, and requires great care and gentleness.

The pedicle next claims attention. It should be isolated as far as possible; sometimes, however, dense adhesions surround the vessels and may unite the kidney by a cicatricial mass to the vena cava or aorta, and make this part of the operation difficult and dangerous. The kidney is now raised and steadied by an assistant while the ureter is separated from the rest of the pedicle. Through the vascular part of the pedicle a stout silk ligature is now passed by means of an aneurysm needle, tension on the pedicle is then relaxed, and the ligature firmly tied.

The ureter is clamped with forceps, and the whole pedicle cut through by short cuts of the scissors, and at a sufficient distance from the ligature to avoid the danger of its slipping. The ureter is now examined; if healthy, it is dropped back into the abdomen after being closed by ligature. Thornton and Albarran have recommended fixing it in the lower part of the wound, but this is an unnecessary precaution. If inflamed, or the seat of tubercular infection, some attempt may be made with antiseptics or the cautery to remove at least a part of the disease, and often it gives no further trouble. The most radical method of treatment is to follow it down, and to isolate and remove it either at the time of the nephrectomy or at a later date.

The wound is then closed, and the cavity left by removal of the kidney is drained for varying periods according to the character of the case.

Subcapsular nephrectomy was introduced by Ollier. It is most useful when an enlarged kidney is firmly bound to surrounding structures by adhesions, but should never be used in dealing with malignant tumours.

The capsule proper of the kidney is incised and peeled off, and the pedicle clamped. The kidney is now removed, and a silk ligature is applied to the pedicle.

If the pedicle is short and thick, clamps may be used. They should be allowed to remain *in situ* for at least forty-eight hours.

The objection to the operation is the rigidity of the walls of the cavity, which delays healing.

Partial nephrectomy or resection of the kidney has been performed by Czerny, Tuffier, Morris, and others for localised tumours, abscesses, and cysts, and for injury. The operation has not as yet been widely adopted, but is a sign of the present tendency to conservatism in renal surgery.

### Operations on the Ureter

Operations on the ureter usually follow an exploration of the kidney, and the abdominal portion of the tube is exposed by extending the lumbar incision downwards and forwards and raising the peritoneum.

A stone situated in the ureter may sometimes be pushed up by the fingers and extracted through the renal or pelvic wound; sometimes,

however, the stone is so firmly impacted that this is impossible, and an incision in the long axis of the ureteral wall (ureterotomy) directly on to the culculus is necessary for its removal. The ureteric wound should, if possible, be sutured; but when the stone is directly cut upon, the damage already produced by pressure may render this inadvisable (Morris).

In all cases, whether sutures be applied or not, drainage of the wound should be provided for in case of retroperitoneal leakage. In the case of a stone impacted in the pelvic portion of the ureter the same method may be employed; but in the female an incision through the vaginal wall is best in stones impacted low down, and in the male the stone can be removed through the perineum (Fenwick).

Strictures of the ureter are now treated in the same way as pyloric stenosis by a longitudinal incision united transversely (Heineke-Mikulicz method), or the contracted portion of the tube may be resected, and the lumen re-established by one of the various methods of "uretero-ureteric anastomosis." Ureterectomy consists in partial or even total removal of a diseased ureter; it has been employed in tubercular and also in suppurative ureteritis, being performed either at the same time as nephrectomy or at a later date.

**Kiestein or Kyestein.**—A whitish scum or pellicle (consisting of phosphates), a product of decomposition, which forms on the surface of urine which has been allowed to remain in a vessel for some hours after being voided; it is not now regarded (although it was so formerly) as a sign of pregnancy. *See* PREGNANCY, PHYSIOLOGY (*Changes in the Urine*).

**Kiln-burning.**—The burning of bricks in an open furnace is, on account of the sulphurous gases evolved, an offensive trade; but if closed kilns be used there is little or no nuisance.

**Kinæsthesis.**—The muscular sense. *See* PHYSIOLOGY, SENSES (*Muscle and Joint Sense*).

**Kinæsthesiometer or Kinesiæsthesiometer.**—A means of testing and measuring the muscular sensibility.

**Kincough.** *See* WHOOPING COUGH.

**Kindergarten Teaching.** *See* MIND, EDUCATION OF ("*Learn by Doing*").

**Kinesioneurosis.**—A malady of the nervous system in which muscular action is abnormal.

**King's Evil.**—Scrofula. *See* EVIL; TUBERCULOSIS.

**Kinks.** *See* INTESTINES, SURGICAL AFFECTIONS OF (*Obstruction, Kinks*).

**Kino.**—A reddish-coloured juice obtained from the *Pterocarpus marsupium*, a tall tree of Malabar, is evaporated to dryness to give small, reddish-coloured angular fragments. It contains *kinotannic acid*, to which it owes its astringent properties, and a glutinous substance which renders it somewhat slow of solution. It is freely soluble in alcohol and in boiling water. *Dose*—5-20 grs. *Preparations*—1. Tinctura Kino. *Dose*—½-1 ʒ. 2. Pulvis Kino Compositus. Contains opium in the strength of 1 in 20. *Dose*—5-20 grs. 3. Pulvis Catechu Compositus. *Dose*—10-40 grs.

Kino is used in the treatment of diarrhœa, especially when the cause is situated in the lower part of the intestine. It has been tried in dysentery, but with only slight success. Powdered kino may be insufflated to check epistaxis. It is occasionally prescribed as an astringent gargle in relaxed conditions of the throat, and trochisci have been prepared for use in similar conditions.

**Kino, Bengal.**—Buteæ Gummi, which is the inspissated juice of the stem of *Butea frondosa*, is known as Bengal Kino, and is used for the same purposes as ordinary or Madras Kino (*q.v.*).

**Kinship.** *See* CONSANGUINITY.

**Kirkilissa.** *See* BALNEOLOGY (*Turkey, Bulgaria*).

**Kirrhonosis.**—A disease of the fœtus in which the tissues, including the serous membranes, are stained yellow; cirrhonosis (Zurmeyer, *De Morbis Fœtus*, Bonn, 1832).

**Kissingen.** *See* BALNEOLOGY (*Germany, Muriated Waters*); MINERAL WATERS (*Muriated Saline*).

**Kjeldahl's Method.**—A method of estimating the organic matter in water; the water is acidulated, is concentrated by evaporation, and is then submitted to a process of combustion with strong sulphuric acid; from the amount of nitrogen in the ammonia produced (estimated by Nesslerisation) the quantity of organic matter is arrived at; the nitrogen constitutes $\frac{14}{17}$ of the ammonia. Hunter Stewart's modification of this method makes it possible to estimate both the organic carbon and the organic nitrogen.

**Klebs-Löffler Bacillus.** *See* DIPHTHERIA (*Morbid Anatomy*); MICRO-ORGANISMS; NOSE, EXAMINATION OF (*Secretion*).

**Klein's Bacillus.**—A bacillus resembling the *b. coli communis*, found in pneumonia epidemica; also the *streptococcus scarlatinæ*.

**Kleptomania.** *See* INSANITY, NATURE AND SYMPTOMS (*Defects of Inhibition*).

**Klikuschi.**—An epidemic or endemic hysterical malady found amongst women in the districts of Orel and Kursk; it would seem to

be a form of hystero-epilepsy, often associated with religious emotion, closely resembling *Ikota* (*q.v.*).

**Klopemania.**—A synonym of *Kleptomania* (*q.v.*).

**Klopsophobia.**—A morbid dread of spies and thieves.

**Klumpke's Paralysis.** — Paralysis with atrophy due to a lesion of the seventh and eighth cervical and first dorsal nerve roots, affecting the muscles of the hand and forearm, with incapacity of dilatation of the pupil. *See* Brachial Plexus (*Tears and Contusions*).

**Knacker.** — An individual who kills animals (horses, mules, cattle) whose flesh is not to be used as butcher's meat; the *Knacker's yard* is the place set aside for such slaughtering (which is one of the offensive trades), and it must be licensed under certain restrictions.

**Knee-Jerk.** *See* Tendon-Jerks; Tabes Dorsalis; etc.

**Knee-Joint.**—This subject is considered in two articles:—

1. **Diseases of.**
2. **Injuries of.**

*See also* Brain, Physiology of (*Motor Areas, Movements of Knee*); Brain, Tumours of (*Localising Symptoms, Reflexes*); Bursæ, Injuries and Diseases of (*Housemaid's Knee*); Deformities; Diabetes Mellitus (*Nervous System Symptoms, Loss of Knee-Jerk*); Diphtheria (*Complications, Abolition of Knee-Jerks*); Joints; Gout; Hysteria, Surgical Aspects of (*Knee*); Meningitis, Tuberculous (*Symptoms, Absence of Knee-Jerks*); Osteo-Arthropathies (*Arthropathies, Knee*); Rheumatism; Rheumatoid Arthritis; Rickets; Tabes Dorsalis; Tendon-Jerks; etc.

## Knee-Joint, Diseases in the Region of.


Anatomical Considerations . . . 127
Deformed Attitudes in Disease of Knee 128
Classification of Diseases—
1. *Tuberculosis* . . . . . 129
2. *Syphilis.* See Joints.
3. *Pyogenic Diseases (including Gonorrhœa and Osteomyelitis in Region of Knee)* . . . . . 138
4. *Rheumatism.* See Joints.
5. *Arthritis deformans* . . . 139
6. *Hæmophylia (bleeder's knee)* . . 140
7. *Charcot's Disease* . . . . 140
8. *Hysterical Knee* . . . . 140
9. *Loose Bodies* . . . . . 140
10. *Contracture and Ankylosis—under different diseases.*
11. *Pathological Dislocation* . . 140
12. *Congenital Dislocation—*
*of knee* . . . . . 140
*of patella* . . . . . 140
13. *Diseases of Superior Tibio-fibular Joint* . . . . . 141
14. *Diseases of the Bursæ around the Knee* . . . . . 141
15. *Ganglia in the Region of the Knee* . 141
16. *Tumours in the Region of the Knee.* 141
*Hydatid Cysts* . . . 142
17. *Paralysis of Muscles acting on Knee—*
*Paralytic Contracture* . . 142
*Paralytic genu recurvatum* . 142
18. *Other Deformities in Region of Knee.* See Deformities, vol. ii.
19. *Operations on the Knee—*
*Arthrotomy, Arthrectomy, Excision.* Under "Tuberculosis," p. 135.


The knee is probably more often the seat of disease than any other joint in the body, probably because of its size and the great extent of its synovial membrane, and because it is more exposed to injury and to cold, either of which is capable of favouring the action of disease-producing agencies. Some of the diseases met with in the knee are rarely seen in any other articulation, while if we pass in review the different diseases that involve joints and note their seats of election, it is the knee in almost every instance that is most prone to be attacked. Although the joint is to a large extent subcutaneous, and therefore lends itself to direct examination, the number and variety of morbid conditions to which it is liable may render their clinical recognition a matter of difficulty and sometimes of uncertainty.

### Anatomical Considerations

*The synovial membrane* extends upwards above the patella and beneath the quadriceps extensor in the shape of a pouch or *cul de sac*; it is rendered very distinct when the joint is distended with fluid, or when the synovial membrane is the seat of the diffuse thickening which so frequently attends tuberculous disease. The precise limit of this upper pouch varies with the development of the subcrural bursa and with the size of the communication between it and the bursa. Sometimes the communication is so wide that the cavities are practically continuous with one another; sometimes it is very narrow, and will scarcely admit the tip of the little finger; in young children the communication is either very narrow or is not developed. The reflection of the synovial membrane is a little higher in the extended than in the flexed position of the limb; it is generally stated as being an inch or more above the upper margin of the patella, but it may be as high as three inches or more. The capacity for distension exhibited by the upper pouch of the joint in the extended position of the limb is partly due to the capsular ligament above the patella being replaced by the quadriceps extensor, which is,

of course, relaxed and flaccid in the attitude of complete extension. When there is only a small amount of fluid in the joint, it is most easily recognised if the patient stands with his feet together and the trunk bent forwards at the hip-joints; the complete relaxation of the quadriceps allows the fluid to bulge above and on either side of the patella, where its presence is readily detected; if the healthy joint of the other limb is examined in the same attitude there should be no likelihood of making a mistake.

The great *extent of the synovial membrane of the knee* is concerned in the rapidity with which effusion may take place, and it is also concerned with the severity of the poisoning from the absorption of toxines when the joint has been infected with pyogenic organisms.

The great development of the *villous processes and fringes of the synovial membrane* is responsible for the frequency with which, under the influence of disease, they may take on an exaggerated growth, and give rise to the pedunculated and other forms of loose body which constitute a prominent feature in many of the chronic diseases to which the knee is liable.

The *communications between the synovial cavity and the surrounding bursæ* are of importance in relation to hydrops and to the spread of infective conditions. That with the subcrural bursa has already been referred to. In about 50 per cent of bodies there is a communication with the bursa between the semimembranosus and the inner head of the gastrocnemius, which latter may form a considerable swelling in the ham in cases of hydrops. There is sometimes a communication between the knee and the superior tibio-fibular articulation, usually through the mediation of the popliteal bursa.

The *epiphysial junctions* in the region of the knee are chiefly responsible for the growth in length of the lower extremity; they are later (twenty-one to twenty-five years) in uniting with their respective shafts than those at the hip or ankle. If their functions are interfered with, whether by injury, or disease, or operation, serious shortening of the limb may result. In relation to disease, it is of great importance to bear in mind that infective lesions at the epiphysial junctions are less likely to spread to the joint than is the case with similar lesions at the hip, shoulder, or elbow; in the knee the epiphysial cartilages reach the surface beyond the limits of the synovial cavity.

### Deformed Attitudes in Knee-Joint Disease

The attitude assumed in many forms of knee-joint disease, and especially in tuberculosis, is that of *flexion* with or without *external rotation of the leg and foot*. The occurrence of flexion is explained by its being the natural attitude of the joint at rest, and by affording most ease and comfort to the patient. Whether or not the preponderating influence of the flexor muscles can inaugurate flexion is doubtful; it is certain, however, that when the joint has become flexed, however slightly, the involuntary effort of the patient to fix the joint is chiefly exercised by the flexor muscles. If the patient is able to walk on the limb, the weight of the body is a powerful factor in increasing an already existing flexion. The greater capacity of the joint sac in the flexed position may be an occasional factor in determining this attitude; it is commonly observed, in cases in which a large amount of fluid is thrown out rapidly, that the patient is unable to extend the limb, whereas in chronic effusions, such as the hydrops of arthritis deformans or of Charcot's disease, the joint may contain an enormous amount of fluid and yet be completely extended without discomfort, because the capsule has had time to yield and stretch. The external rotation of the leg is supposed to be associated with the contraction of the biceps muscle; this may or may not be the case; it is certain that the outward rotation is most marked in cases in which the patient has been confined to bed.

One of the most characteristic deformities of the knee is that associated with *backward displacement of the tibia*; it is especially met with in neglected cases of chronic and especially tuberculous disease, where the patient has been allowed to walk and bear weight on the limb when it is already flexed at the knee. This mechanical explanation of the occurrence of backward displacement is, in our opinion, a more reasonable one than others which have been suggested. By many it is ascribed to the traction of the hamstring muscles and the cicatricial contraction of the capsular ligament and other structures in the ham, the displacement being rendered possible by the softening and yielding of the crucial and other controlling ligaments; it has also been suggested, inasmuch as the backward displacement is invariably associated with flexion, and that it is only observed in patients before the skeleton has attained maturity, that the growth of the femur, in the flexed position of the limb, may result in its projection beyond the tibia.

There is still another deformed attitude met with in knee-joint disease, viz., a certain degree of *genu valgum* or *abduction of the leg*; it is commonly associated with slight flexion, and is chiefly met with in patients who have borne weight on the limb in walking; the valgum is also associated with slight outward displacement of the patella on to the external condyle, with prominence and apparent enlargement of the internal condyle, with depression of the pelvis on the diseased side, and apparent lengthening of the limb.

All of the above deformed attitudes are especially met with in tuberculous disease of the knee-joint, and an accurate knowledge of

them is of great importance in the treatment of the disease in question.

1. Tuberculous Affections of the Knee

General Facts . . . . . 129
Pathological Anatomy . . . . . 129
Clinical Types—
1. *Hydrops*. . . . . 130
2. *Papillary or Nodular Tubercle of the Synovial Membrane* . . . 130
3. *Cold Abscess or Empyema* . . 131
4. *Diffuse Disease of Synovial Membrane*—
*Synovial fungus. White swelling* 131
5. *Primary Disease in the Bones* . . 131
The Formation of Abscess . . . 132
Diagnosis . . . . . 132
Prognosis . . . . . 133
Treatment—
*Conservative* . . . . . 134
*Operative*. . . . . 135
*Indications for* . . . . 135
*Arthrectomy* . . . . 136
*Excision* . . . . . 136
*Amputation* . . . . 137
*Treatment of Deformities following Disease of Knee* . . . . 137

In patients of all ages affected with tuberculous disease of bones and joints the knee-joint is the second most frequent seat of disease. While especially common in childhood and youth, it may be met with at any period of life, and is not uncommon even in patients over fifty or sixty years of age. It is less serious to life than the same disease in the spine, pelvis, or hip, chiefly because it is much more amenable to treatment.

Pathological Anatomy.—The frequency with which the disease originates in the synovial membrane and in the bones would appear to be about equal (351 synovial to 281 osseous, König). When the synovial membrane is the seat of disease it exhibits a marked tendency to grow inwards over the articular surfaces; in the case of the femur this usually takes place from the lateral margins of the condyles at the level of the lower margin of the patella, often shutting off the suprapatellar pouch and resulting in fixation of the knee-cap. In the case of the tibia and of the patella, the ingrowth takes place from the margins towards the centre, diminishing the area of their articular surfaces. The ingrowth of the synovial membrane may come to fill up the entire cavity of the joint or may divide it up into compartments, *e.g.* one above the patella, and two below in relation to each femoral condyle, and may succeed in shutting off a focus in the bone which would otherwise have erupted into the joint. In addition to the tuberculous thickening of the synovial membrane, the thickening and gelatinous transformation of the parasynovial tissues, and especially of the fat around the joint, contribute in an important degree to that form of the disease which is known clinically as "white swelling." The destructive changes in the synovial membrane and in the articular cartilages, which result from the tuberculous infection, are similar to those met with in other joints.

It is important to bear in mind that, when the infection is arrested and the newly-formed granulation tissue is converted into fibrous tissue, this is often attended with alterations in the joint of a more or less permanent character. The suprapatellar pouch is often obliterated, the patella may be more or less fixed on the condyles of the femur; the tibio-femoral articular surfaces may be fused together by fibrous tissue or by bone, and the joint may be ankylosed in the position in which it was maintained during the disease. In relation to the question of relapse, it is important that the reparative process may be limited to one or other portion of the joint, *e.g.* one condylar area; while the disease may remain latent or may progress in other parts, and that by the formation of new fibrous tissue a focus in the bone may be shut off or encapsulated.

The rarer forms of synovial disease will be described under the clinical features. The nature and seat of *the bone lesions* are subject to the same wide variations as in other joints. König gives the following figures as to their relative frequency in the different bones: in 281 cases the patella was affected in 33, the femur in 97, the tibia in 107, and several bones in 48. Small, frequently multiple, granulation foci may be found beneath the articular cartilage of the tibia, or along the lateral margins of the femoral condyles, especially the internal. Caseating foci are less often met with; they may, however, attain a considerable size, so as to merit the description of a caseous abscess, especially in the interior of the head of the tibia. Foci of the sclerosed variety, resulting in sequestra, are comparatively common; the most characteristic examples are met with in the substance of one or other condyle of the femur, and when they abut on the articular surface they present the peculiar eburnation. Extensive caseation (tuberculous osteomyelitis) of the marrow of an entire epiphysis is exceptional, but when present may extend into the adjacent diaphysis. Primary foci in the patella may erupt externally; more often they give rise to disease in the knee-joint.

In young children the frequency of severe bone lesions, such as sequestra, is considerably above the average; they are more often located in the femur than in the tibia, but instead, as in the adult, of infecting the joint, they commonly erupt extra-articularly, *e.g.* into the popliteal space or on the lateral aspects of the condyles.

### Clinical Types of Tuberculous Disease of the Knee

1. *Hydrops tuberculosis* is the name given to that type in which the outstanding feature is the accumulation of fluid within the joint. It is analogous to the ascitic type of peritoneal tuberculosis, and it is the chief representative of the "chronic simple synovitis" of the older authors. It is most often met with in the knee of young adults, but may occur at any age. Inasmuch as it frequently terminates in recovery with a useful joint, it may be regarded as the least serious form of tuberculous joint disease; this is largely due to its origin from a purely synovial lesion; foci in the bones are exceptional, but they may be the cause of the hydrops in those cases where the joint becomes suddenly distended with fluid.

In the *serous form* of hydrops the fluid accumulates gradually and imperceptibly; the capsule is chiefly distended in its upper recess, the patella is pushed forwards and floats; there is no pain; the functions of the joint are fairly well preserved, as the movements are only hindered by the distension of the capsule; there may be some complaint of tiredness, but the patient is usually able to walk in moderation without discomfort; prolonged distension of the capsular and other ligaments may cause a sense of insecurity and instability. The variation in the amount of fluid from time to time is characteristic; it subsides under rest and increases after exercise.

In *the fibrinous form* of hydrops the joint also slowly fills with fluid, but unless the capsule is tightly distended we are usually able to recognise some thickening of the synovial membrane, especially along the line of its reflection on to the femur. By displacing the fluid from one part of the sac to another by means of the fingers, we may recognise a peculiar friction or crepitation from the contact of floating masses of fibrin and melon-seed bodies against one another; this is best appreciated if the knee is rapidly flexed and extended by the patient while it is grasped and compressed by the fingers. If the fluid is evacuated, it is found to have suspended in it fibrinous bodies, often of the melon-seed type, which are very apt to block the cannula. If the joint is opened a similar fibrinous material may be found lining the synovial membrane, and it may be also covering certain areas of the articular surfaces.

The diagnosis of tuberculous hydrops is to be made from that arising from other causes, *e.g.* from injury, and especially repeated sprains of the joint, from gonorrhœa, from arthritis deformans and Charcot's disease, from the hydrops which may attend staphylococcus disease (*e.g.* Brodie's abscess) in one of the adjacent bones, and lastly, from the hæmarthrosis met with in bleeders. Given a patient, and especially a young adult, with water in the knee, and having excluded injury, the probability is that it is due either to tuberculosis or to gonorrhœa. The presence of melon-seed or other fibrinous bodies in the fluid is confirmatory of tuberculosis. The demonstration of bacilli and the results of inoculation in animals are too uncertain to be of any value.

The treatment will be considered with that of tuberculous disease of the knee in general, but it may be pointed out in this place that the hydrops is especially amenable to conservative treatment by rest, and by injections into the joint of iodoform glycerine. In the fibrinous variety a large cannula must be used, otherwise it is liable to be blocked; even then it may be impossible to evacuate the contents of the joint; the choice will lie under these conditions, between diluting the joint contents with saline solution introduced by a syringe and washing out the joint before introducing the iodoform, and making an incision into the upper pouch, squeezing out the fluid and loose bodies, and injecting the iodoform by means of a rubber tube attached to the nozzle of the syringe, the tube being gradually withdrawn as the stitches closing the incision are tightened up.

As already indicated, the prognosis is favourable as regards cure of the disease and recovery of a useful joint; but relapse is not uncommon, and experience shows that neglect of the disease may be followed by a gradual transition of the innocent hydrops into the graver types of synovial tuberculosis.

2. *Papillary or nodular tubercle of the synovial membrane* may be employed as a clinical term to distinguish a group of conditions in which the dominant feature is a fringy, papillary, polypoidal, or tumour-like growth from the synovial membrane, sometimes localised and circumscribed, sometimes generalised throughout the entire membrane. The conditions referred to are comparatively rare, but are usually attended with characteristic clinical features; they are more often met with in male adults between the ages of twenty and forty. The onset and progress of joint symptoms are extremely gradual; the patient complains of stiffness and swelling, chiefly after exertion, sometimes subsiding for weeks or months and then relapsing. In a certain number of cases there are indefinite or atypical symptoms of loose body in the joint, such as occasional painful locking and inability to extend the limb, especially liable to occur in walking or in going up a stair; the locking is usually easily disengaged and the movements are again quite free. The patient may give a history of several years of partial and intermittent disability, with lameness and occasional locking, and still he may be able to go about or even continue his occupation.

On examination of the joint there is usually a moderate degree of hydrops which subsides

under rest; one is then able to feel ill-defined cords or tufts or nodular masses, and in the suprapatellar pouch one may be able to grasp these between the fingers. There is none of the diffuse thickening of the synovial membrane which is so characteristic of white swelling, there is little wasting of muscles, and it is quite exceptional to observe any symptoms of implication of the articular surfaces or the formation of a cold abscess.

On opening the joint there may escape similar fluid and fibrinous bodies to those described under hydrops. With the finger one may feel that the upper pouch is occupied by fringes, or polypoidal processes, or tumour-like masses derived from the synovial membrane; they are easily scraped away with the spoon or finger-nail; the articular cartilage is usually normal, but it may be fibrillated as in arthritis deformans.

The diagnosis is to be made from arthritis deformans, and in certain cases from loose body of other than tuberculous origin; it is not usually made with certainty until the joint is opened.

The treatment specially applicable to this type of tuberculous disease is to perform a partial arthrectomy, and remove the whole of the synovial membrane which is affected; in exceptional cases, and especially those in which there are also foci in the bones, it is better to perform the more radical operation of excision.

3. *Cold abscess* or *empyema* of the knee has been specially observed by Konig and others, and its outstanding feature is the accumulation of pus in the joint. It is analogous to the purulent type of peritoneal tuberculosis; its clinical features resemble those of tuberculous hydrops so closely that the differential diagnosis is rarely completed until the fluid in the joint is withdrawn by means of a trocar and cannula. It is usually the result of a primary tuberculosis of the synovial membrane. It may be met with in patients who are up in years as well as in childhood and youth, more often in those who are much reduced in health, and who are the subjects of tuberculous lesions elsewhere. Its development is insidious, often without the phenomena of inflammation, and its progress is chronic. Like hydrops, it is most often met with in the knee; it is less favourable from the point of view of prognosis, because the patients affected are usually those of feeble resisting powers and the subjects of tuberculous disease elsewhere.

Rovsing has described an acute form of tuberculous empyema which begins suddenly in very young and apparently healthy children; the joint rapidly swells, becomes sensitive, and the child is feverish. It is liable to be mistaken for the "acute arthritis" of infants, because evacuation of the pus by a simple incision usually brings about recovery with complete restoration of function. The synovial membrane, if examined, shows miliary tuberculosis.

4. *Diffuse Primary Disease of the Synovial Membrane. Synovial Fungus. White Swelling.*—This may follow upon the hydrops and other preceding types of tuberculous disease of the knee, or it may commence as such.

*When the disease is still limited to the synovial membrane* the chief complaint is of the swelling in the region of the joint. The appearance of the swelling is eminently characteristic; in contrast to hydrops, in which it is most marked in the upper pouch, it is here most marked in the vicinity of the tibio-femoral junction and on either side of the patella; it gradually tapers off above and below, so that the swelling is more or less spindle-shaped or fusiform. As a result of the thickening of the lateral pouch of the synovial membrane the condyles of the femur may appear to be enlarged, and on superficial examination one is very apt to think that the bone is affected. The patient at this stage may limp slightly and keep the joint a little flexed, and may complain of tiredness and stiffness, especially at night after exertion. The movements of the joint, although restricted, are remarkably natural and easy. The wasting of the muscles in front of the thigh makes the swelling of the joint appear greater than it really is.

*When the disease has involved the articular surfaces*, and led to partial destruction or "ulceration" of the cartilages, the disease assumes an aspect which is much more serious. The previous mobility diminishes and is soon entirely lost; this rigidity of the joint is seldom so absolute as that observed in hip disease; like it, however, it is chiefly due to the involuntary contraction of muscles, and it disappears under anæsthesia. There is much more complaint of pain, which is readily excited by any jar or attempt at movement, and there are usually "startings" at night. If no treatment is adopted the knee becomes more flexed, usually to a right angle, the leg and foot are rotated outwards, and in young patients the tibia is gradually displaced backwards. The wasting of the muscles becomes more pronounced. The synovial swelling is not only greater in amount, but it often becomes more boggy in consistence and is hot to the touch. In addition to the deformity just mentioned there may be a certain amount of genu valgum, especially if the patient has continued to put the affected limb to the ground; under the same conditions the pelvis may be depressed on the diseased side with apparent lengthening of the limb.

The formation of cold abscess will be referred to later.

5. *Primary Tuberculous Disease in the Bones forming the Knee-Joint.*—It is usually impossible to recognise osseous foci in the vicinity of the knee *so long as they are confined to the interior*

*of the bone* affected. They remain latent for long periods, and may even cure without their existence having been suspected. They may be responsible for certain vague symptoms in the region of the knee, which are often dismissed as being "rheumatic" or hysterical; the patient complains of tiredness and aching, aggravated by exertion, and therefore usually worst at night, and there may be some stiffness of the joint. It is quite exceptional to be able to demonstrate an enlargement of the bone or tenderness on tapping. Radiography gives the most valuable information if the osseous focus be of sufficient size.

*If the osseous focus makes its way to the surface outside the limits of the synovial membrane*, the complaint of the patient will be more definite and localised, there is pain in walking and marked lameness, the bone will become tender on pressure, and a swelling will develop which will gradually soften into a cold abscess. The joint, although perhaps a little sensitive and restricted in its movements, and in some cases showing a moderate amount of effusion or hydrops, may escape infection altogether unless the condition is neglected or improperly treated.

*If the osseous focus reaches the surface at the point of reflection of the synovial membrane*, the membrane becomes thickened, at first in the region of the osseous focus; the thickening then spreads throughout its whole extent, and the case then follows the course of ordinary synovial disease, with the exception that it is usually more stubborn, that it usually goes on more quickly, that there is more pain and rigidity at an early period, and that abscess is very apt to form soon in the synovial membrane at the point where the osseous focus has reached the surface. (Watson Cheyne.)

*If the osseous focus reaches an articular surface* it tends to destroy the cartilage over it and to infect the joint. The symptoms vary with the suddenness of the perforation and with the amount of infective material thrown into the joint. Sometimes the communication between the focus and the joint cavity is incomplete, and the resulting joint disease evolves very gradually and partakes of the character of a dry arthritis with marked articular symptoms, which is very obstinate, and tends to incomplete ankylosis, with fixation of the patella and an entire absence of any synovial fungus. This type is the nearest representative in the knee of what is called "caries sicca" in the hip or shoulder. Sometimes, on the other hand, the communication with the osseous focus is shut off by newly-formed fibrous tissue, and the joint lesion, although similar to the last, is confined to a limited area such as that of one of the condyles. In exceptional cases a quantity of tuberculous material may be discharged suddenly into the joint, and we may have the following clinical features as described by Watson Cheyne:—"The patient experiences sudden severe pain, probably at the time of perforation, followed by swelling of the joint and in some cases by fever; the whole joint becomes rapidly infected, the surface of the synovial membrane undergoes caseation, and the cartilages are quickly destroyed. In this condition some cheesy pus is generally formed at an early period in the joint, the patient suffers much pain, especially on the slightest movement, there is starting pain at night, the knee is flexed and rigid. When abscesses form and are opened they are found to communicate with the joint, the bones are carious, and if recovery takes place there is bony ankylosis."

*The Formation of Abscess in Tuberculous Disease of the Knee.*—Statistics show that suppuration and sinuses are met with in rather more than 50 per cent of cases, and that the influence of these on the prognosis is decidedly unfavourable. Although abscess formation may take place at any stage in the progress of the disease, it occurs more frequently after symptoms of involvement of the articular cartilages have manifested themselves. Abscesses may originate in the substance of the synovial membrane, and spread externally to the periarticular soft parts, or they may originate in the joint itself; within the joint the suppuration is often confined to one or more areas which have been shut off from the general cavity, *e.g.* in the suprapatellar pouch, or in the area of one or of both femoral condyles. A cold abscess may also form in relation to one of the bursæ in the popliteal space. If untreated the abscess tends to spread into the periarticular tissues, into the popliteal space, and down the back of the leg, or upwards into the thigh between the vastus internus and adductors on the inner side, and along the edge of the vastus externus on the outer side; such abscesses may extend for a considerable distance, and by infecting extensive tracts of tissue, increase the risk of general tuberculous infection. Abscesses on the anterior aspect of the upper part of the leg are less common, and result chiefly from foci in the patella or in the head of the tibia.

The sinuses which result from the external rupture of abscesses are often multiple and extensive, and by allowing of the entrance of septic infection, and by militating against the primary healing of operation wounds, are always to be regarded as a serious aggravation of the disease and an additional risk to the life of the patient.

*Diagnosis of Tuberculous Disease of the Knee.*—The diagnosis of tuberculous hydrops has been referred to under this head. There is usually no difficulty in recognising typical cases of white swelling. In some cases the thickening of the synovial membrane may resemble in certain respects the much rarer condition of *perisynovial gummata*; the latter are chiefly

met with in women; the gummatous swelling is more nodular, uneven, and less uniform than the tuberculous; there are frequently tertiary ulcers or depressed scars in the neighbourhood of the patella, and the joint symptoms are less prominent. When there is in addition *gummatous disease of the tibia or femur*, with sinuses leading down to carious bone, the diagnosis may be extremely difficult, especially as the overwhelming frequency of tuberculous disease predisposes one to assume that the disease is of this nature, and the syphilitic origin is apt to be missed because it is not suspected.

The very early stages of tuberculosis and of *arthritis deformans of the knee* may resemble each other very closely because the respective clinical peculiarities have not had time to develop. Difficulty is met with, especially in adults, who complain of pain, stiffness, and lameness, and who present a moderate amount of swelling of the joint with or without effusion. In arthritis deformans the progress is more intermittent and erratic, the symptoms are rather aggravated than improved by rest, and they are influenced by changes in the weather. The presence of crackings or of creaking on movement, the sensation of roughness or crepitation on grasping the joint while it is rapidly flexed and extended, and, finally, the presence of crackings and irregular pains in other joints, may be of help in recognising arthritis deformans.

A resemblance between tuberculous and *pyogenic affections of the knee* may arise when there is a partial infection of the joint, resulting from a focus in one of the neighbouring bones. There is less likelihood of mistake when the tuberculous lesion assumes, as it does occasionally, an acute character, because the general feature and the progress of the disease clear up the difficulty before much time has been lost. Staphylococcus lesions, on the other hand, *e.g.* Brodie's abscess, whether in the upper end of the tibia or lower end of the femur, may run a chronic course, and may be associated with changes in the knee-joint (swelling, effusion, adhesions) which are very similar to those resulting from tuberculosis. The history of the case and the local features must be gone into very carefully, and the bones should be examined by radioscopy.

Cases of tuberculous disease of the knee may occasionally present themselves with the symptoms of *loose body in the joint*; the recognition, however, of the other evidences of joint disease is usually sufficient to differentiate them from the more numerous group of cases in which the loose body (or bodies) is the only discoverable lesion in the joint.

Cases are on record in which treatment has been carried out for tuberculous disease of the knee, and in which the after progress has shown the patient to be suffering from *sarcoma of the lower end of the femur, or of the synovial membrane of the knee.*

It may be instructive to refer to the points in which disease of the knee-joint and sarcoma, especially of the lower end of the femur, resemble one another:—The initial symptoms are often those of vague pains and of a limp, which may be relieved for a time by rest or by the application of a blister; at a later stage the characters of the swelling may be deceptive, as the tumour tissue may project from the bone into the upper recess of the joint; the swelling may be hot and tender, the muscles may be wasted, and the joint may be flexed and stiff; there may be considerable evening pyrexia greater than that observed in tuberculous disease of the knee—in the latter affection the temperature is usually quite normal so long as the patient is confined to bed. The following are the chief points favouring the diagnosis of sarcoma:—There is often a history that the swelling was first noticed on one side of the joint; the swelling is more often uneven or nodular; it does not accurately correspond to the shape of the synovial membrane, but extends beyond the limits of the joint, and involves the bone to a greater extent than is usual in cases of joint disease. The swelling is also more unequal in consistence, being harder than the synovial fungus in some parts, and softer or fluctuating in others. If a trocar and cannula is pushed into the swelling it may be felt to grate on roughened bone, or may even perforate the thin shell of the tumour, and it only abstracts blood. It is useful in difficult cases to confine the patient to bed, and fix the limb in a splint for a week until the œdema of the soft parts and any fluid in the joint have been absorbed; the nature of the swelling, and the presence or absence of joint symptoms, can then be determined with greater accuracy. Radiography is most useful in cases where the bone is expanded by the tumour, or where much new bone is formed, as in the ossifying sarcoma. Finally, recourse should be had, without too long delay, to exploratory incision and immediate microscopic examination of the suspected tissue elements. The diagnosis of *hysterical affections of the knee* is the same as in other joints. The "*bleeder's knee*" met with in the subjects of hæmophylia may resemble tuberculous disease very closely indeed, especially when repeated hæmorrhages have taken place into the joint, and the latter has become swollen, stiff, and flexed. The differential diagnosis is considered in paragraph 6, p. 140.

The *prognosis in tuberculous disease of the knee* is chiefly concerned with the possible retention or loss of the functions of the joint, and, in the case of children, with the future growth of the limb. In hydrops and in mild forms of primary synovial disease recovery with a movable joint may be confidently anticipated. When

the articular surfaces are seriously implicated, recovery with mobility is most unlikely; on the other hand, the occurrence of rigid and preferably osseous ankylosis affords the best prospect of permanent cure. Inasmuch, however, as this result can only be attained under expectant conditions, with considerable loss of time, there is great inducement to securing this result more rapidly and with greater certainty by means of an operation which will at the same time remove the disease. Most of the deformed and shortened limbs from knee-joint disease to be seen on our streets are capable of prevention.

As regards the prognosis to life we may cite the statistics of König. Out of 615 cases observed at the Göttingen clinique over a period of eighteen years, no less than 205 succumbed (33⅓ per cent) chiefly from tuberculosis of the lungs and other internal organs.

*Treatment of Tuberculous Disease of the Knee.*—As in other joints, this may be discussed under the headings of conservative and operative. Conservative measures are specially applicable in children, and that for several reasons: in them spontaneous recovery is much more likely to take place than in adults; time is of secondary importance, because there is no question of their having to earn a living; excision, which in the case of the adult restores a usable limb with great certainty, is to be avoided in children, because it may lead to interference with growth; and, finally, the alternative operation, arthrectomy, is unreliable as to the functional results obtained. On the other hand, adults, and especially breadwinners, cannot be expected to wait two or three years for a problematical spontaneous recovery when one can promise an almost certain cure within a definite time by means of excision. It cannot be too strongly insisted upon that it is not only waste of time but a source of danger to the patient to persist with conservative measures in cases in which spontaneous recovery is impossible or unlikely. Konig's statistics, which cover a period of eighteen years, bring out the remarkable fact that a larger proportion of patients finally succumbed among those submitted throughout to expectant treatment, than among those in which the disease was removed by operation.

*Conservative Measures.*—These are to be employed in the first instance, unless, as already stated, the condition of the patient or of his joint is such as renders the prospect of spontaneous recovery with a useful limb unlikely or impossible, and with this reserve, that if the disease does not yield, we must not hesitate to have recourse to operation.

(*a*) *The Joint must be put at Rest in the Extended Position.*—The patient should be confined to bed during the initial period of treatment. If the joint is flexed and sensitive it should be supported on a pillow, and extension by the weight and pulley is applied to the leg until the limb is straight. Genu valgum deformity is more difficult to get rid of than flexion; if it does not yield to extension, the bones should be forcibly brought into line with one another under an anæsthetic. Once the limb is straight, it must be kept so by suitable apparatus, *e.g.* a trough of Gooch (known as Watson's splint), a gutter of wire or basket work with a foot-piece, lateral poroplastic splints secured with an elastic webbing bandage, or plaster-of-Paris. The external application of iodine, mercurial ointment, or of fly blisters does not appear to have any curative influence. If the disease readily yields to treatment by rest alone the patient may be allowed to leave his bed, but the fixation of the joint and the extended position of the limb must be maintained by a Thomas or other suitable splint for a period of at least twelve months. The splint is removed at intervals for hydrotherapy, massage, and electricity of the atrophied muscles and passive movements of the ankle. Before the splint is discarded altogether, it may be left off during the night; it is ultimately replaced by an elastic bandage.

(*b*) *Venous Congestion by Bier's Method.*—This method of treatment is variously appreciated by different observers, and is still *sub judice*. Some cases appear to improve under it more rapidly than with rest alone; it may therefore be combined with the latter, *i.e.* either while in bed or going about with a Thomas splint; if there is no decided improvement in a fortnight it should be abandoned.

(*c*) *Injection of Iodoform Glycerine.*—The details of this method have been described in the general article on Joints. So far as the knee is concerned it is most easily introduced and most efficient in cases of hydrops. In the more common synovial fungus or white swelling the injection is more difficult, more painful, and requires to be repeated more frequently and at shorter intervals (ten days to three weeks). The value of iodoform injection, as of venous congestion, is variously estimated by different observers; by some it is accepted as a method of treatment which has very largely done away with the necessity for operation, by others it is regarded as capable of bringing about an improvement which is only temporary. Our own experience is decidedly encouraging.

(*d*) *The treatment of abscess* is conveniently included with the conservative methods. One of the objects of keeping a patient with tuberculous joint disease under observation is that of recognising an abscess at the earliest possible moment. When discovered it should be treated by the iodoform-glycerine method, as already described in the article on Joints (*q.v.*). If the abscess does not yield to the iodoform treatment it should be cleared out by operation;

in doing so, if an osseous focus is discovered it should be cleared out at the same time.

*Treatment of Extra-articular Tuberculous Foci in the Bones.*—Intermediate between the conservative and operative treatment, the question may arise of clearing out osseous foci in the neighbourhood of the knee, either to prevent infection of the joint, or, where this has already taken place, to increase the chances of cure by conservative measures. The great difficulty is to diagnose the foci in question apart from abscess formation, for the symptoms are not at all definite. Local pain, tenderness, thickening, or enlargement of the bone may indicate a focus, especially in the head of the tibia or in the patella. The proper treatment is to cut down on the bone, remove any infected soft parts *en masse*, clear out the focus in the bone with the spoon, gouge, or chisel, and stuff the cavity with iodoform gauze. If the joint is opened into in this procedure, the technique will depend on the state of the synovial membrane: if healthy, the opening into the joint may be closed with sutures; if there is circumscribed disease of the synovial membrane it may be clipped away; if the thickening of the membrane is more extensive, the joint may be filled with idoform glycerine and closed; if the joint as a whole is diseased, then the case is one for arthrectomy in children or excision in adults.

The above described partial operations are especially successful in children; in the adult they are less certain to cure and more dangerous to life than the more radical operation, viz., excision.

*Operative Treatment of Tuberculous Disease of the Knee.*—The operations concerned are arthrectomy (erasion), excision, and amputation. When the disease has implicated all the structures of the joint, and spontaneous recovery is unlikely, and is in any case likely to be attended with a stiff joint, it is waste of time to persist with conservative measures when the same result may be obtained with rapidity and certainty by means of an operation which will also at the same time get rid of the disease. Among the *indications for operative treatment in disease of the knee* in contrast to other joints, we should therefore place in the front rank the hopelessness of obtaining a movable joint, as inferred from symptoms of destruction of the articular cartilages, rigidity, pain on the slightest attempt at movement, startings at night, and fixation of the patella. In the second rank may be included cases which are unsuited for conservative treatment, *e.g.* where there is deformity incapable of being rectified otherwise, or when the general health requires that the disease should be removed by the most rapid method. In the third group we should include cases in which the disease progresses in spite of a fair trial of conservative measures, in which the synovial thickening is increasing or is showing signs of softening, or where, from the mere persistence of the disease, there is reason to suspect the existence of serious disease in the bones, or finally, where the disease has relapsed after apparent care under expectant treatment. Other things being equal, the fact of the patient being an adult would determine the balance in favour of operation.

Having decided on the necessity of operation, the next and almost equally difficult question to decide is as to its nature. There is considerable difference of opinion regarding the wisdom of aiming at a movable joint, and of recommending arthrectomy with this object in view. Increasing experience of the results of this operation shows most conclusively that a movable joint, which will at the same time be useful, is exceedingly rare—so rare, in fact, that the question of mobility should scarcely be entertained. As has aptly been pointed out by König, it is hardly reasonable to expect mobility after removing the entire capsule and synovial membrane, upon which the mobility of the joint depends. There is another side to the question, viz., that the slight extent of mobility secured by an arthrectomy may not always be an advantage to the patient, inasmuch as it may permit of gradually increasing flexion and disabling deformity of the limb. It is also maintained that the disease is more liable to relapse after arthrectomy than after excision, both because the disease is less radically removed, and because the remanent mobility exposes the limb to strain far more than if there were a rigid ankylosis.

The real advantage claimed for arthrectomy is that it not only avoids any immediate shortening of the limb, but also that it does not interfere with its future growth. On these grounds alone it is to be preferred to excision, in patients under fifteen or sixteen years of age. In performing it, however, one must not be hampered with the obligation of aiming at a movable joint; if the articular surfaces are affected they must be pared with a strong knife. After the wound has healed, means must be employed to prevent flexion for a period of two years. If at the end of this period the joint is found to have retained a certain degree of mobility, well and good; but one must not sacrifice the greater certainty of curing the disease and of obtaining a useful limb, for the doubtful advantages of mobility. In adults, the operation of excision is preferred because there is no question of interfering with growth. The ends of the bones are removed by means of a saw, the sections being made in such a way as will secure the most accurate and most rigid adaptation to one another, and the certainty of bony ankylosis. Amputation is indicated, whether in children or in adults, in cases where arthrectomy or excision is incapable of removing

all the disease: it should not be reserved for hopeless cases.

*Arthrectomy of the Knee.—Erasion.*—Flexion of the joint should, if possible, be corrected before operation by means of extension with the weight and pulley, in order to stretch the structures in the ham. There are several methods of performing the operation: those in which a transpatellar or H-incision or an anterior U-shaped flap is made, have this in common, that the patella or its ligament is divided transversely. Other methods are to be preferred which maintain the integrity of the extensor apparatus, viz., that by two vertical incisions, one on either side of the patella, or the single large external J-shaped incision of Kocher. We shall describe the last-mentioned operation. The limb should be rendered bloodless in the usual way. The incision is made upon the outer aspect of the joint. It begins a hand's-breadth above the upper margin of the patella, and at first descends vertically at a distance of two fingers'-breadths from the outer border of that bone; it then inclines gently inwards, and terminates on the inner aspect of the tibia, a little below the tubercle. The fat and fascia lata are divided in the line of the incision, and at the upper part the fibres of the vastus externus. The capsule is then divided over the outer condyle of the femur and along the outer edge of the ligamentum patellæ. By means of the chisel the tubercle of the tibia, along with the ligamentum and periosteum, are displaced inwards. One then proceeds to remove the synovial membrane and semilunar cartilages, and in doing so excellent access is obtained by dislocating the patella inwards, while at the same time the joint is more and more flexed. If it is desired to clear out the posterior pouch of the joint, the femoral attachments of the lateral and crucial ligaments may be separated, along with the periosteum and bone, by means of the chisel. The articular surfaces are carefully inspected, and any suspicious areas are scooped out with the spoon. If there is genu valgum it may be corrected by paring the articular surfaces of the inner condyle and internal tuberosity to the extent required. Iodoform powder is rubbed into the surface and recesses of the wound. The divided capsule and other ligamentous attachments are sutured so as to re-establish the stability of the joint. Drainage may be provided for by means of a rubber tube or a strand of iodoform gauze or worsted. If there is any doubt as to the likelihood of primary healing, the cavity of the wound should be packed with iodoform gauze or worsted. The entire limb from the tuber ischii to the malleoli is then enveloped in plaster-of-Paris, or enclosed in a long splint. When the wound is soundly healed the patient is provided with a Thomas splint, which must be worn for a period of not less than two years in order to prevent flexion of the joint. During the whole of this time the patient should be kept under observation.

*Flexion after Arthrectomy.*—If the patient has been allowed to put the limb to the ground, or has been otherwise neglected, the knee is very apt to become flexed, and this deformity once started is almost certain to increase by the mere weight of the body in walking. In a small number of cases, and especially in rickety children, the flexion may be partly due to a forward curve of the lower part of the shaft of the femur. The deformity may be corrected by linear osteotomy (using a broad chisel) either across the knee-joint from the front, or, in addition, where the femur is curved, by a second osteotomy in the lower third of the shaft.

*Relapse of the disease after arthrectomy* is to be treated by excision or amputation.

*Excision of the Knee.*—Inasmuch as the double object of this operation is to remove every particle of disease, and to secure rigid bony ankylosis, there is no longer any question, as in arthrectomy, of preserving ligamentous connections between the bones, or of preserving the extensor apparatus. The subcapsulo-periosteal method of Ollier, which presents such advantages in other joints, is quite out of place in the knee. The surgeon should aim at removing the antero-lateral portions of the capsule and synovial membrane, along with the patella and its ligament, in one piece, as if he were engaged in the removal of a malignant tumour (A. G. Miller, Kocher). At the end of the operation, the sawn ends of the femur and tibia should be covered by nothing but skin and fascia.

The incision employed should be one giving free access to the whole area of the joint; Kocher's external incision, already described in the operation of arthrectomy, or a large anterior U-shaped flap may be employed. In view of the superfluity of skin in cases where the knee is flexed, or where there is considerable swelling, an elliptical portion comprising that over the patella may be removed. This, which is a matter of choice in most cases, becomes compulsory when this area of skin is the seat of a sinus. By whatever incision the anterior aspect of the joint has been exposed, the next step should be to divide the connections of the vasti and rectus femoris with the upper part of the capsule, so as to allow of exposing the upper limit of the suprapatellar pouch. The more common procedure of sawing across the patella, or of dividing the ligamentum patellæ, and immediately opening the cavity of the joint, is to be deprecated, both because it makes it more difficult to define the upper pouch, and because it exposes the wound from the outset to tuberculous infection. The suprapatellar pouch is then dissected off the femur, in front and

on both lateral aspects, until its reflection on to the cartilaginous surface of the femur is approached. The ligamentum patellæ is divided, and the lower limbs of the capsule and synovial membrane are similarly dissected off the tibia from below upwards until the articular surface is reached. Having divided the lateral ligaments and flexed the joint, the capsule, synovial membrane, patella, ligamentum patellæ, infrapatellar pad of fat, and the semilunar cartilages, are removed in one tumour-like mass. The posterior recess of the joint is then displayed by detaching the crucial ligaments, and by flexing the joint until the femur and tibia are nearly parallel with one another. Not only must the posterior part of the capsule and synovial membrane be removed, but also any disease in the popliteal bursa. The sharp spoon is not so reliable as the scissors or knife. The risk of wounding the popliteal artery during this step of the operations is probably exaggerated. Iodoform powder is rubbed into the raw surfaces and recesses of the wound. Having cleared the ends of the bones, the articular surfaces are removed by means of the saw; skill is required in order to do this, so as to ensure that the sawn surfaces will be capable of being accurately applied to each other in the extended position of the limb. The usual procedure is to saw the bones at right angles to the long axis of the limb, *i.e.* parallel to their articular surfaces, and to employ for this purpose an ordinary amputation saw. If the sawn surfaces fit accurately they are merely placed in contact, otherwise they may be retained in apposition by means of two long steel pins introduced through the skin beyond the excision wound. The pins should not be driven home until the limb is placed in the splint in the extended position. Kocher makes with a narrow butcher's saw a convex section of the femur, and a concave section of the tibia. This method of sawing the femur in the case of growing limbs has the advantage of being least likely to damage the epiphysial cartilage, but it is a little more difficult to carry out successfully. Whatever method is employed for sawing the bones, if tuberculous foci are discovered on the sawn sections, they should be cleared out with the gouge in preference to taking away another slice of the bone. The tourniquet is removed and the blood-vessels are ligatured. The wound is closed, and drainage is provided for by a rubber tube brought through an opening in the skin at the outer side. The limb is maintained in a box or simple posterior (Watson) splint until the wound is soundly healed; plaster-of-Paris is then applied, and the patient allowed to go about on crutches. Three months after the operation the plaster case may be exchanged for a Thomas splint, which should be worn for six months or a year.

*Mortality of Excision.*—Apart from the risks attending any major operation, the chief causes of death following excision of the knee are phthisis pulmonalis and general tuberculosis.

*Results of Excision.*—In the majority of cases the disease is permanently cured, and there is rigid ankylosis at the tibio-femoral junction. The more rigid the ankylosis the more useful is the limb. Very slight flexion, amounting to 5° or 10°, is the best attitude for walking. The shortening directly due to the operation varies with the amount of bone removed; it varies from ½ to 2 inches, and it is easily compensated for by depressing the pelvis on the same side, or by thickening the sole of the boot. If shortening already existed before the operation, the combined shortening may necessitate the use of a high boot. When excision has been performed in a limb which is still growing, and the epiphysial cartilages are removed, the shortening may amount to as much as 6 inches. A very obstinate form of flexion is sometimes observed in young subjects as a result of removing the posterior two-thirds of the epiphysial cartilage of the femur. The anterior portion which is left continues to develop bone, and the original plane of section no longer remains at a right angle to the axis of the limb. In order to correct it a wedge-shaped portion of bone must be removed.

In fibrous ankylosis, unless it is very close and strong, a Thomas splint or other form of apparatus must be worn until the desired stability is acquired. The relapse of tuberculous disease in the shape of abscesses and sinuses is to be treated on the usual lines.

*Amputation* is reserved for severe and usually neglected cases—where the disease extensively involves the bones and is rapidly advancing with evidences of suppuration, where there are septic sinuses (especially after the failure of excision to secure a useful limb), and where the lungs and other internal organs are seriously implicated. It is often remarkable how much the lung disease may improve after the removal of suppurating tuberculous disease of the knee.

The amputation should be performed well above the limits of the infected tissues, whether synovial membrane or cellular tissue.

In view of the unfavourable nature of the cases submitted to amputation it is not surprising that the mortality is a high one, especially if we include those cases which die some time after the operation from phthisis or general tuberculosis.

*Treatment of Deformities resulting from Antecedent Disease of the Knee.*—We are here concerned with cases in which the disease has been recovered from, but the joint has been allowed to assume the flexed position, with or without backward displacement of the tibia.

When the deformity is of the nature of a *contracture*, in which the articular surfaces are

fairly preserved, and the flexion is due to the contraction of the posterior part of the capsule and the soft structures in the ham, extension may be given a trial; but if it fail, all the shortened structures should be divided by the open method, by means of an oblique incision, made from above downwards across the popliteal space. Forcible correction of the deformity is to be avoided unless it be done in stages; with each step towards the extended position the limb is to be encased in plaster-of-Paris.

When there is *fibrous or osseous ankylosis* in the flexed position the procedure varies in different cases. In patients who are still growing, one may succeed with a modified arthrectomy and the removal of a thin slice of bone. In adults, the usual procedure is to remove a wedge of bone. In the bony ankylosis of growing patients, we may either divide the femur above the level of the joint, or wait until the patient is nearly fully grown, and remove a wedge of bone as in the adult. When the flexion is extreme there is a risk of overstretching the popliteal vessels, and of interfering with the circulation in the foot; in these cases it is safer to remove another slice of bone.

When there is a genu valgum deformity, one may practise an osteotomy of the femur as in rickety knock-knee.

### 3. Pyogenic Diseases

Acute and chronic serous synovitis.
Purulent synovitis.
Acute arthritis of infants.
Joint suppurations in pyæmia.
Severer forms of septic arthritis.
Infections from penetrating wounds.
Gonorrhœal affections of knee.
Acute osteomyelitis of lower end of femur.
Acute osteomyelitis of upper end of tibia.
Chronic osteomyelitis—Brodie's abscess.

These include a number of diseased conditions resulting from infection through the blood-stream of the joint, or of the structures in its neighbourhood, with the common pyogenic organisms, or with special bacteria such as the gonococcus or typhoid bacillus. The direct infections resulting from a penetrating wound of the joint may also be conveniently described under this head.

The clinical features vary with the gravity of the infection, and are very similar to those met with in other joints; they may assume the form of an *acute serous synovitis*, which may recover spontaneously, or may subside into a *chronic synovitis* or *hydrops*. Exudation into the joint is always a prominent feature. A characteristic *persistent and relapsing form of hydrops* is met with in the knee, in association with latent forms of staphylococcus osteomyelitis, *e.g.* Brodie's abscess, in the lower end of the femur or upper end of the tibia.

The *purulent forms of synovitis* in the knee present wide variations with regard to their severity and progress. There are certain mild forms called "catarrhal" by Volkmann, in which the joint fills with pus without any periarticular phlegmon, and without any destructive changes in the joint, and in which, if the pus is evacuated, recovery usually takes place with complete restoration of function. This type is most often observed in the "*acute arthritis of infants*," related to staphylococcus osteomyelitis of the tibial or femoral epiphysis, or one of the adjacent ossifying junctions. The *joint suppurations in pyæmia*, which especially affect the knee, are usually remarkably latent; there is, however, no hard and fast line between the milder forms and those which are *serious and progressive*. In addition to the presence of fluid (sero-pus or pus) in the joint, there is a pronounced periarticular phlegmon, œdema of the surrounding skin—and, it may be, of the leg and foot—destructive changes in the articular surfaces in the direction of caries, attended with severe pain, rigidity of the joint, and startings at night; the pus within the joint perforates the capsule and spreads upwards into the thigh beneath the quadriceps, backwards into the popliteal space infecting the bursæ, and downwards into the calf. The septic fever accompanying the severer forms of septic arthritis may readily merge into pyæmia, and cause the death of the patient. The author has observed one case of destructive purulent arthritis in the knee of an adult, which had become stiff from disease in childhood; the relapse in adult life appeared to have originated from a recent pyelitis; both the bones and the soft parts in the region of the knee were riddled with suppuration, and in spite of amputation through the thigh the patient died of septicæmia.

*The septic synovitis and arthritis following upon penetrating wounds of the knee* are usually of a severe and progressive character; they are met with more commonly from accidental wounds with a chisel, or awl, or penknife, or the spike of a railing, from gunshot wounds, or compound fractures involving the knee, but they may follow upon such operations as wiring a fractured patella, removing a loose body or semilunar cartilage.

Practically all the severe forms of pyogenic arthritis result in ankylosis, which is more often osseous than fibrous; in treating them, it is therefore of great importance to keep the bones in a straight line by means of splints and weight extension.

*The treatment in pyogenic diseases of the knee* must be directed to meet the features of each individual case. The general indications are to elevate and immobilise the limb in the extended position on a posterior splint, preferably a gutter of Gooch's splinting reaching from the fold of the buttock to beyond the foot, and to

apply an antiseptic fomentation over the entire area of the joint. If there is exudation into the joint with much tension, the fluid should be withdrawn by means of a trocar and cannula inserted obliquely into the suprapatellar pouch. If the fluid is purulent, a free incision should be made into the joint above and to the outer side of the patella, and a drainage-tube introduced. If this does not arrest the local progress of the disease or the general toxæmia, the patient should be anæsthetised, incisions should be made on either side of the patella, freely opening the capsule and suprapatellar pouch, and drawing through tubes from one side to the other. The drainage may be further improved by pushing a dressing forceps between the bones into the popliteal space, and making an opening there, through which a large rubber tube may be drawn backwards into the joint. Periarticular suppurations must be searched for, and if found, should be opened and drained. The more complete the apparatus for drainage the more thorough is the subsequent irrigation. Saline solution may be employed to wash away pus, blood, and fibrinous material; peroxide of hydrogen and sulphurous acid are the most useful chemical agents for irrigation purposes. Cases are met with, especially those from direct infection through a wound, in which, in spite of all one's efforts in draining and irrigating, the temperature continues to rise, the patient loses ground, and anxiety for the joint yields to anxiety for the life of the patient. The choice of procedure will consist in laying the joint freely open from side to side, dividing the ligamentum patellæ and capsule, and packing the cavity between the bones with gauze, or excising the joint or amputating through the thigh.

The *gonorrhœal affections* of the knee have been sufficiently considered in the general article on Joints; one may refer, however, to the predominance of hydrops, which may prove very obstinate, and in which one may find it necessary to evacuate the fluid through a cannula, and to irrigate the joint with protargol.

*Acute Osteomyelitis of the Lower End of the Femur.*—The lower femoral epiphysis and the adjacent ossifying junctions are very common seats of this disease; at its onset it is frequently mistaken for an affection of the knee-joint, and regarded as rheumatic in nature. The lower end of the bone should be carefully palpated and compared with that of the sound limb, and considerable reliance in diagnosis may be placed on the recognition of the point or points of maximum tenderness. In the operative treatment of femoral osteomyelitis the incision should be made on the outer aspect of the limb in the line of the intermuscular septum; having divided the fascia lata, a grooved director may be pushed inwards to discover the presence of pus beneath the periosteum; the opening thus made may be enlarged with dressing forceps so as to admit the finger, and permit of investigating the locality and extent of the disease; the trigone of the femur is often found to be denuded of periosteum, and is especially liable, in neglected cases, to become the seat of necrosis.

In *acute osteomyelitis of the upper end of the tibia* the superficial situation of the bone is of great assistance in diagnosis and in operative treatment.

*Chronic forms of osteomyelitis, e.g.* Brodie's abscess, attain their maximum frequency in the lower end of the femur and upper end of the tibia; sometimes a sinus may extend from the abscess into the knee-joint, but even then the communication is valvular, so that it is exceptional to have a generalised pyogenic arthritis. More commonly the joint suffers from the formation of adhesions, and the conversion of the articular and interarticular cartilages into fibrous tissue; or it may fill with fluid, constituting one of the forms of relapsing or intermittent hydrops.

5. *Arthritis Deformans. Osteo-Arthritis.*—This may affect the knee only, or may be polyarticular. It may follow upon injury of the joint or of the bones in its vicinity. The changes related to the synovial membrane attain their maximum in the knee, and may assume the form of hydrops with or without fibrinous bodies, or of overgrowth of the synovial fringes, and the formation of pedunculated loose bodies. The changes in the articular surfaces and margins are more easily recognised in the knee than in other joints; fibrillation of the cartilage imparts a feeling of roughness or friction when the joint is firmly grasped during flexion and extension, while lipping of the margins of the trochlear surface of the femur is readily estimated after comparison with the healthy joint. When a portion of the "lipping" is broken off it may give rise to the symptoms of loose body. In advanced cases of hydrops the ligaments become stretched, and there may be lateral movement with grating of the articular surfaces.

Among therapeutic measures applicable to arthritis deformans of the knee, we have observed considerable improvement following tapping of the joint, in cases of hydrops, and injection of iodoform glycerine; there is a sharp reaction and increase of the pain and the swelling for a day or two. Where the patient's sufferings are chiefly due to the presence of hypertrophied fringes, pedunculated loose bodies, or a detached portion of the lipped articular margins, great relief may follow on opening the joint and removal of the offending fringes or bodies. When the disease is of a very aggravated type, is mono-articular, and is the cause of serious crippling in a patient who is otherwise in good health, the question of excising the joint should be considered.

6. *Hæmophylia*—"*Bleeder's Knee.*"—This is a rare but very characteristic affection, chiefly met with in boys and young adult males. The first hæmorrhage into the joint originates suddenly after some trivial injury, and may attract so little attention that it is not thought necessary to seek advice; the appearances are very similar to those of hydrops, and there is little or no pain; the patient is usually anæmic, but is otherwise healthy; the temperature is often elevated (101°–102°), especially if at the same time there are hæmorrhages into the cellular tissue of other parts of the limb or elsewhere in the body. After repeated hæmorrhages the joint becomes uniformly swollen from the deposit of fibrin on the synovial membrane and its subsequent organisation. As the swelling is often associated with flexion and stiffness, the resemblance to white swelling is very close indeed—so much so, indeed, that a wrong diagnosis has been made, and the joint subjected to operation with disastrous results. The treatment of bleeder's knee has been described in the article on Joints.

7. *Neuro-Arthropathies.* — *Charcot's disease* more often affects the knee than any other joint; it is chiefly met with in adult males suffering from lightning pains and loss of the knee-jerks. In the knee it often presents the features of an immense hydrops with œdema of the leg and foot, but whatever the external appearances, the presence of abnormal movements, lateral or rotatory, with cross grating and the utter absence of sensitiveness, are very characteristic; in many cases it is possible to partially or completely dislocate the tibia from the femur.

8. *Hysterical knee* may be regarded as the type of hysterical joints, being the one most commonly affected. It has been described as such in the general article on Joints (see also "Hysteria, Surgical Aspects of," vol. iv.).

9. *Loose Bodies.*—The origin, structure, and clinical features of loose bodies have been discussed in the general article on Joints (p. 84); we may here refer to the operation for their removal. The incision is made directly over the body whenever it can be located to a particular area of the joint. If, on the other hand, the body is free and has to be searched for, the joint must be freely opened, preferably by a vertical incision along the outer border of the patella, so as to admit the finger. The limb must be carefully manipulated during the exploration, or the finger may be severely nipped between the patella and the femur. If the body lies in the posterior recess of the joint one may fail to find it through an incision made on the anterior aspect of the joint; under these circumstances the whole joint must be opened up, and this is best carried out by detaching the tubercle of the tibia, and dislocating the patella inwards, as has been already described in the operation of arthrectomy by Kocher's method. One should always remember that there may be more than one loose body in the knee-joint.

10. *Pathological Dislocation.* — Apart from the backward displacement of the tibia observed in tuberculosis, pathological dislocation is almost confined to cases of Charcot's disease.

11. *Congenital Dislocation of the Knee.*—The tibia is nearly always dislocated forwards, and the patella is frequently absent; when the dislocation is bilateral, it is often accompanied with other errors of development. In congenital dislocation of the tibia forwards, the joint is in a state of hyperextension, which may be increased or diminished by manipulation. The treatment consists in flexing the knee, under an anæsthetic, as nearly to a right angle as possible, and fixing it in this position with plaster-of-Paris or other apparatus. Where the patella is absent it is usually necessary to produce an artificial ankylosis between the femur and tibia.

*Spontaneous dislocation of one or both knees* may be observed in infants; in older children the patient may be able to dislocate the joint voluntarily. J. W. Ballantyne records the case of an infant of eleven months old, in which the right knee was frequently dislocated outwards during attempts at walking; on examining the limb it was found that when the leg was grasped in the position of nearly complete extension, and the upper end of the tibia was pressed outwards, that a partial dislocation of the tibia took place with a slight creaking noise; the dislocated tibia was easily reduced, and the whole procedure did not appear to cause the infant any pain.

In this and similar cases the joint should be fixed on some retentive apparatus until the joint acquires the desired stability.

12. *Congenital Dislocation of the Patella.*—There are several varieties of this lesion. *The complete persistent form*, in which the knee-cap rests on the outer surface of the external condyle in all positions of the joint, is extremely rare, and is usually combined with congenital knock-knee, or with marked external rotation of the leg. Both deformities may be corrected by manipulative treatment if this is begun in early infancy. *The spontaneous or intermittent form*, in which the knee-cap is only displaced outwards when the knee is flexed, is chiefly met with in girls; there is usually a history that the art of walking was acquired with difficulty, and at a later period than in other children. It is frequently associated with imperfect development and flattening of the external condyle, with knock-knee, and with unequal action of the quadriceps. It may occur on one or both sides. The usual complaint is that in walking the patient suddenly falls to the ground and suffers intense pain,

both from the dislocation and from the violent contact with the ground; the knee-cap readily returns to its normal situation when the leg is extended, but the joint may be swollen and painful for a day or two. The dislocation occurs at irregular intervals, and is quite beyond the control of the patient.

The following methods of operative treatment have been practised: (1) detaching the tubercle of the tibia, so as to allow of the insertion of the ligamentum patellæ being displaced inwards; (2) deepening the patellar groove in the trochlear surface of the femur; (3) tightening up the capsular ligament along the inner side of the patella; (4) producing an artificial bow-knee by supracondyloid osteotomy of the femur, as recommended by Professor Chiene, and specially applicable in the female.

If there is knock-knee as well, it should be corrected in the usual way by Macewen's operation.

13. *Diseases of Superior Tibio-fibular Joint.*—These are extremely rare and of little practical interest. The author has observed infection of this joint from a tuberculous focus in the head of the fibula; the disease ultimately spread to the knee by way of the popliteal bursa.

14. *Diseases of the Bursæ in the Region of the Knee.*—The anatomical situation of the bursæ has been described in the article on "Bursæ" in vol. ii. Various types of *acute bursitis* are commonly observed in *the prepatellar bursa*; acute infective forms result in the formation of a circumscribed abscess, or in a spreading cellulitis which may extend upwards into the thigh and downwards into the leg, requiring prompt and energetic treatment by multiple free incisions. The chronic or trade bursitis is familiarly known as *housemaid's knee.*

Inflammation of the *infrapatellar bursa* is a very rare affection; the infective form is liable to spread to the knee-joint.

*The bursæ in the popliteal space* are chiefly liable to a condition in which the sac of the bursa fills with fluid, that is to say, a hydrops; and it may be difficult to differentiate this from the hernial pouchings of the synovial membrane, known as synovial cysts, because the bursæ liable to be affected with hydrops may communicate with the knee-joint and share in its hydrops; and yet the communication may be so narrow that one may not be able to displace the fluid from the bursa into the joint. The bursa between the inner head of the gastrocnemius and the semimembranosus is the one most commonly affected with hydrops; it forms a lax, fluctuating, egg- or sausage-shaped cyst at the inner side of the popliteal space. When the knee is extended and the popliteal fascia is on the stretch the swelling becomes harder and less well defined, whereas in the flexed position it lends itself better to digital examination. The treatment of the various forms of bursitis is carried out on the same lines as in similar diseases elsewhere. The quiescent hydrops of the semimembranosus bursa rarely gives rise to any symptoms, and may therefore be left alone; if treatment is required, the most satisfactory procedure is to dissect it out.

15. *Ganglia in the Region of the Knee.*—These are chiefly met with in working-men and athletes. The commoner variety develops on the outer aspect of the joint, giving rise to a tumour about the size of a pigeon's egg in the interval between the femur and tibia, and in front of the biceps tendon. When the limb is extended, the tumour is hard and but slightly prominent; in the flexed position it becomes more prominent, and fluctuates.

The patient may ignore its existence, or may complain of stiffness, discomfort, and difficulty in extending the limb completely; the disability is greater after working in the kneeling posture, or after football or tennis. If treatment is required, the tumour should be excised; in doing so, some of the fibres of the capsular ligament may require to be sacrificed, and the knee-joint may be opened into. On section, the tumour is found to be a multilocular cyst, the spaces of which are filled with a colourless jelly rich in mucin. The author has observed similar ganglia on the inner aspect of the knee, also in the interval between the bones and in front of the inner hamstring tendons.

16. Tumours in the Region of the Knee

*Of the Bones—*
- Chrondroma and osteoma.
- Sarcoma.
- Hydatids.

*In the Popliteal Space—*
- Enlarged gland and chronic abscess.
- Bursal swellings.
- Synovial cysts.
- Consolidated aneurysm.
- Neuroma.

*The cartilaginous exostosis* is the commonest innocent tumour in the region of the knee; it may be the only one, or there may be a large number scattered throughout the skeleton. Originally developing from the epiphysial junction, the tumour in the case of the femur usually projects on the outer or the inner side of the bone, and may attain a considerable size; in the case of the tibia it more often projects on the antero-lateral aspect between the tubercle and the internal tuberosity, and grows downwards parallel with the shaft. There may be some arrest of the growth of the limb from interference with the epiphysial cartilages. The tumour causes inconvenience by its bulk, or there may be a bursa over the convexity which may become enlarged and sensitive.

As a rule these exostoses may be left alone, as they cease to grow when the skeleton has

attained maturity. If they are causing suffering they are easily removed; a vertical incision is made through the soft parts, and the neck of the tumour is cut through with a stout chisel.

*Cystic tumours* in the interior of the femur or tibia, of the nature of liquefied chondromata, endotheliomata, or myeloid sarcomata, are of very rare occurrence.

*Sarcoma* of the bones in the region of the knee-joint are comparatively common, especially in children and young adults; their general characters have been already described with the "Diseases of Bone," in vol. i. We may again refer to the great difficulty of diagnosis when they are met with at an early stage, before the tumour element has been a prominent feature, and before the advent of such pathognomonic symptoms as egg-shell crackling, spontaneous fracture, and infection of the overlying soft parts. Most difficulty is met with in relation to *tumours of the lower end of the femur*, which sometimes resemble the chronic and especially the tuberculous forms of joint disease; the differential diagnosis has been already discussed under this head.

*Sarcoma of the upper end of the tibia* is nearly always of the central variety; it is less likely to be mistaken for disease of the knee-joint than for other chronic lesions of the upper end of the bone, *e.g.* tubercle, gumma, Brodie's abscess, hydatid, etc.

*Sarcoma of the upper end of the fibula* is of the periosteal type, and appears clinically as a rounded or flattened elastic swelling, practically free from pain or tenderness; the cutaneous veins are increased in number and size over the tumour; the movements of the knee-joint are intact, and the patient is quite able to walk or run about. When the knee is flexed, the tumour may be felt to project towards the popliteal space. A tuberculous mass commencing to liquefy into a cold abscess is the only lesion which at all resembles it. The treatment applicable to sarcomata in the region of the knee has been described in the general article on "Diseases of Bone," in vol. i.

*Hydatids.*—The ends of the bones forming the knee-joint are among those most frequently affected with this rare disease; the clinical features resemble those of the more slowly-growing central sarcomata, *e.g.* deep-seated pains and enlargement of the bone. The swelling may be firm and elastic, or may exhibit egg-shell crackling; spontaneous fracture and suppuration are likely complications.

Treatment consists in making an extensive opening into the bone and clearing out the cysts from its interior; the cavity is then stuffed or drained. The eradication of the parasite must be thorough or the disease is liable to relapse.

17. *Paralysis of Muscles acting on the Knee-Joint and Paralytic Contracture.*—*Peripheral paralysis* from injury or disease of the individual nerves, *e.g.* the anterior crural, the great sciatic, are extremely rare. When the extensor group of muscles is paralysed the disability is considerable, as the patient is unable to bear any weight on the limb except in the position of complete extension.

In *infantile paralysis* involving the lower extremity, the knee-joint may be so unstable and wobbly that the patient may be unable to walk without the assistance of a crutch; artificial ankylosis of the joint is the most satisfactory treatment. As a rule the paralysis is followed by the contraction of certain groups of muscles and by deformity, of the kind usually called *paralytic contracture*; the knee is usually flexed, and although the patient may be able to increase the amount of flexion, he is unable to extend the knee. Along with the flexion there may be a variable amount of genu valgum and inversion of the thigh, especially in patients who have walked with a crutch. The disability is usually aggravated by the addition of paralytic contracture deformity at the ankle. As regards treatment, while a good deal of improvement may follow upon division of the shortened structures and correction of the deformity, arthrodesis of the knee in the extended position yields the best results in the majority of cases.

*Paralytic genu recurvatum* is a rare but very unsightly deformity resulting from infantile paralysis when it chiefly involves the extensor muscles of the thigh; the deformity is the result of the patient using the limb so as to compensate for the muscular weakness. In taking a step forwards, he swings the leg forwards so that when the foot touches the ground the knee is hyperextended, this being the only position in which he is able to bear his weight on the limb without the knee suddenly giving way under him. The hyperextension becomes more and more pronounced as the ligaments and other structures in the ham gradually yield and stretch.

The treatment consists either in fixing the knee-joint by a suitable apparatus or in performing arthrodesis.

## Knee-Joint, Injuries of.


INJURIES

SPRAINS . . . . . 143
BRUISES . . . . . 143
WOUNDS . . . . . 144
GUNSHOT WOUNDS . . . . . 144

FRACTURES IN VICINITY OF KNEE-JOINT

LOWER END OF FEMUR . . . . 145
TIBIA BELOW TUBEROSITIES . . . 145
FROM COMPRESSION OF TIBIA THROUGH ITS ARTICULAR SURFACE . . . . 145
UPPER END OF FIBULA . . . . 146
PATELLA . . . . . 146
SPONTANEOUS FRACTURES . . . 147

EPIPHYSIAL INJURIES

SEPARATION OF LOWER EPIPHYSIS OF FEMUR . . . . . 148
SEPARATION OF UPPER EPIPHYSIS OF TIBIA . . . . . 149
SEPARATION OF UPPER EPIPHYSIS OF FIBULA . . . . . 150

DISLOCATIONS

DISLOCATION OF KNEE-JOINT . . . 150
DISLOCATION OF PATELLA . . . 151
DISLOCATION OF SEMILUNAR CARTILAGES . 151
OTHER INTRA-ARTICULAR INJURIES . . 152

## INJURIES

SPRAINS.—Sprain of the knee-joint is a common injury caused by a twist or wrench of the joint. It is associated in the majority of cases with a varying degree of tearing of ligaments and synovitis. In severe cases a sprain may be accompanied by hæmorrhage into the joint, rupture of tendon sheaths, and displacement of tendons, or injury to a semilunar cartilage. Ligaments may be wrenched from the bone and the synovial membrane may be torn.

*Diagnosis* of sprain is chiefly negative. Bony points should be carefully examined to exclude fracture or separation of an epiphysis, especially that of the lower end of the femur, which in children may easily be mistaken for a sprain. Pain and tenderness is often most marked over the attachments of ligaments. A certain diagnosis that the injury is nothing more than a sprain may be rendered impossible at first by the swollen condition of the joint.

*Prognosis.*—After a sprain the joint is usually weak, and for some time—often months—is not to be depended on for active work. In the severer cases adhesions tend to form, and stiffness results which is troublesome to overcome, and may result in a degree of permanent impairment of the joint movements. Effusion may persist and become chronic. As a rule, hæmorrhage into the joint is absorbed without ill results.

A joint once sprained is liable to subsequent attacks of synovitis consequent on very slight injuries or over-exertion. For a year or two this susceptibility may be a source of constant annoyance to the owner of the joint.

*Treatment.*—Rest, elevation of the limb, and the application of a posterior splint should be accompanied by either cold (in the form of an ice-bag, or Leiter's coils) or hot fomentations. It is a question which is the better. The sooner treatment is commenced after the accident the better is the result obtained. In using heat the best method to adopt is to place the knee at once over a bath or basin of hot water, and sponge it, keeping it as hot as is bearable by the addition of more hot water from time to time. This should be continued for half an hour, and then the limb wrapped in a large quantity of cotton wool, and a bandage as firmly applied as is consistent with comfort. Rest and pressure should be continued so long as swelling and tenderness persist. If ligaments have been torn to any extent, this period of rest should be prolonged to three or four weeks to allow of healing of the torn structures. If the effusion be very great, immediate aspiration may be performed before application of the bandages, every care being taken to ensure asepsis. The subsequent treatment should consist of massage of the limb, gradual movement of the joint, and the use of a support to the knee when the patient commences to go about. Some surgeons advise the immediate application of a plaster bandage, accompanied at first by rest in bed and elevation of the limb, though later the patient may go about on crutches; this bandage to be removed when sufficient time has elapsed to allow of repair of the ligaments, and then massage and movement commenced. A bandage or light leather knee-cap will be necessary when the patient commences to walk. In the treatment of old sprains the use of the Dowsing hot-air baths, accompanied by massage and electricity, often yields very successful results. Manipulation and movement of the joint under chloroform may be necessary to overcome adhesions within the joint and neighbouring synovial sheaths.

BRUISES.—Blows and crushes of the joint may cause severe damage without the skin being torn: synovitis, detachment of articular cartilages, ligaments, or tendons, crushing and splitting of the ends of the bones, hæmorrhage into joint, and rupture of the main vessels may result. Slight contusions may be followed by tuberculous disease, arrest of growth of limb due to injury of epiphysial cartilage in the young, or a form of chronic arthritis with lipping of articular edges, grating and creaking of the joint.

Severer crushes may be followed by sloughing of the skin, the crush becoming compound, or gangrene of limb from injury to the vessels. Suppuration is very prone to occur, followed by necrosis of parts of the bones, acute abscesses, and sequestrum formation, sometimes pyæmia; hæmorrhage into the joint may result in ultimate adhesions and ankylosis.

*Treatment.*—Where the skin remains intact even severe bruises are well recovered from. The treatment consists in elevation and the careful application of splints.

Where there is considerable inflammatory reaction leeching may give excellent results. The ice-bag may be used unless the skin is much bruised, in which case it is better avoided owing to the risk of death of the skin. If suppuration occur, free drainage should be provided, small fragments removed, and continuous irrigation or immersion in a warm boracic bath employed. Excellent results frequently follow this method of treatment. In cases where the ends of the bones are implicated, or the main vessels torn, amputation is necessary.

WOUNDS OF KNEE-JOINT.—All wounds of the knee-joint are grave injuries owing to the presence of the large synovial membrane and its pouches, and to the risk of septic infection, the instrument causing the injury being rarely aseptic.

*Symptoms.*—The chief difficulty, especially in *punctured* wounds, is to decide whether or not the joint has been opened. The escape of synovial fluid is certain evidence of penetration of the joint. Fluid may, however, come from a synovial sheath or a bursa, though not in the same amount. If there be no escape of synovial fluid the rapid swelling of the joint is a very suggestive sign. Where there is doubt, great caution should be exercised in the use of a probe; frequently the instrument enters at a distance from the joint and produces a more or less valvular wound, thus preventing the ready escape of fluid.

In GUNSHOT wounds the joint is usually unmistakably involved. Bullets striking the large and cancellous extremities of the bones in the neighbourhood of the knee-joint frequently drill cleanly through them. In the past war in South Africa cases have been recorded in which the condyles of the femur have been drilled through without fracture taking place. The patella is usually drilled, but may be fractured, and numerous cases have occurred in which the bullet has passed through the knee-joint and perforated the femur or tibia as well. The small bore and the high velocity of the bullets are responsible for this peculiarity.

The range appears to have little effect in determining the extent of the injury. Hæmarthrosis is a frequent symptom, but the swelling usually subsides rapidly. In the Chitral campaign, where bullets of larger calibre and less velocity were used, the injuries were much more severe. A bullet striking the patella and femur produced extensive fracturing of the bones, and sometimes the cavity of the knee-joint was converted into a mere bag of comminuted fragments of bone. The upper end of the tibia is much more liable to splinter than the lower end of the femur, and the fracture may extend into the joint.

*Treatment.*—All wounds should be carefully cleansed with soft soap and turpentine, and then washed with corrosive or biniodide of mercury lotion 1-1000 or 1-2000. A clean incised wound may be sutured at once; ragged edges should be trimmed, and if much bruising, it is better not to aim at immediate union.

In severe wounds and in gunshot wounds, accompanied by injury to bones, the treatment used almost invariably to be amputation. The experience of the past war has been very different—the recoveries from gunshot wounds of the knee being numerous, and with useful limbs.

These excellent results have been due, not so much to the facilities for antiseptic surgical practice, which was often very difficult to carry out efficiently, but to the general favourable surroundings of the patients.

The after-treatment of wounds of knee-joint should consist in rest on a splint until the wound is healed and any effusion has subsided, and then gradual movement and light support of the knee.

*Complications.* — 1. Acute septic arthritis; the symptoms are rapid swelling of the joint, with redness, heat, pain, œdema, and fever. If limb is not controlled the knee becomes flexed. Erosion of cartilage gives rise to agonising pain when the joint is moved, and the characteristic starting pains at night. Pus tends to burrow among the muscles, and secondary abscesses and pyæmia may result. If sepsis is suspected the joint should be aspirated, and if the fluid is becoming purulent the joint should be freely incised on both sides and drained after irrigation with 1-2000 corrosive. The pouches, especially that beneath the quadriceps, should be carefully washed out and a tube inserted into each. Continuous irrigation is of great value. Amputation may become necessary.

2. Impaired mobility or ankylosis will result from adhesions in severe injuries, or after sepsis, in many cases but not in all.

3 Injury to, or tearing of the popliteal nerves from the instrument causing wound, or from splintering of bone. These will require suture.

4. Injury to popliteal artery and resulting aneurysm may occur.

5. Osteomyelitis is a very rare complication.

*Foreign Bodies.*—Sometimes nothing can be felt, even on the most careful examination. In these cases a skiagraph will often show the position of the foreign body. It is often of great assistance to have a lateral view of the joint as well as an antero-posterior.

*Treatment.*—A lateral incision will often be sufficient to reveal the foreign body. If lodged between the condyles, it may be brought into view by alternately flexing and extending the joint, and thus enable one to remove it with a sharp hook or forceps. Should this fail, the patella will require to be turned aside or sawn across to allow of a full view into the joint cavity. The body frequently is found between the condyles, and may be attached to the intercondyloid notch by dense fibrous tissue. To avoid having the cicatrix adherent over the patella, and to have it well removed from possible pressure in the act of kneeling, it is best to make a curved incision across the knee with the convexity upward, the extremities being well over the condyles, and the middle above the upper margin of the patella. A vertical lateral incision may be converted into the more extensive one if required. The joint should be closed without drainage, and such

cases recover with perfect movement if asepsis has been preserved.

### Fractures in Vicinity of Knee-Joint

Fractures of Lower End of Femur.—The varieties to be distinguished are: (*a*) supracondyloid, (*b*) oblique and T-shaped fractures of the condyles, (*c*) detachments of parts of the articular surface.

(*a*) *Supracondyloid* fractures may be transverse just above the condyles, or oblique; sometimes spiral, due to twisting of the bone. When transverse or oblique the displacement of the lower fragment is typical, the powerful gastrocnemius tending to flex it toward the popliteal space, so that the upper fragment overrides the lower; the knee-joint may be involved as a result of this injury. A serious complication may be found in pressure on or rupture of the popliteal vessels by the lower fragment when the displacement is extreme, resulting in thrombosis of the artery or gangrene of the leg.

*Diagnosis.* — The signs are: shortening of thigh, crepitus, abnoral mobility, and projection of the displaced fragment. If the knee-joint be involved there will be effusion into it. Pulsation in the tibial vessels should be sought for; its absence renders the condition much more serious.

*Treatment* consists in giving an anæsthetic and reducing the displacement by extension. If the fragments can be easily controlled, the limb should be placed in a straight splint with extension. If the tendency to displacement be well marked, the knee should be bent and the limb placed on a double-inclined plane, with extension in the line of the thigh, or a Hodgen splint may be used. If the displacement persists, the tendo Achillis should be divided.

The joint may be aspirated if much effusion of blood is present. Massage and passive movement should be commenced in fourteen days, by which time sufficient callus should be formed.

Injury to the popliteal artery may result in a traumatic aneuryism and arrest of the circulation in the leg, in which case it will be necessary to incise the popliteal space, turn out the clots, and ligature both ends of the artery. The removal of the pressure of the clot may allow of a sufficient collateral anastomosis through the articular branches to save the limb. This anastomosis is not, however, good, because the blood must pass through two sets of capillaries —from profunda and anastomotica—into the articulars, and thence into the tibials. If, therefore, the leg still remains cold after the pressure of the aneurysm has been removed, gangrene is imminent, and amputation just above the seat of the fracture is necessary.

*Oblique or T-shaped Fractures of the Condyles.* —Oblique fractures of one or other condyle may occur from severe violence; the fracture runs from the intercondyloid notch obliquely upwards. Displacement is usually slight, as the fragment remains attached to the lateral ligament.

In the T-shaped fracture the split between the condyles is more vertical, and both are separated from the shaft.

*Diagnosis* is made by the recognition of lateral mobility and crepitus, increase of width of the knee, pain on pressure about the condyles, effusion of blood into the knee, and the presence sometimes of sharp projections of bone.

*Treatment.* — Is best treated in a slightly flexed position on a Hodgen splint. Extension and elastic compression to the joint should be applied; if necessary, the joint may be aspirated first. Great care should be taken to get the limb straight in putting up this form of fracture, as there is considerable tendency to genu valgum or varum after such an injury. Early massage, and in a fortnight passive movement, should be carried out to avoid ankylosis.

*Fractures of Parts of the Articular Surface.*— Very rarely the attachments of the lateral ligaments may be detached along with part of the articular edge in dislocations of the knee.

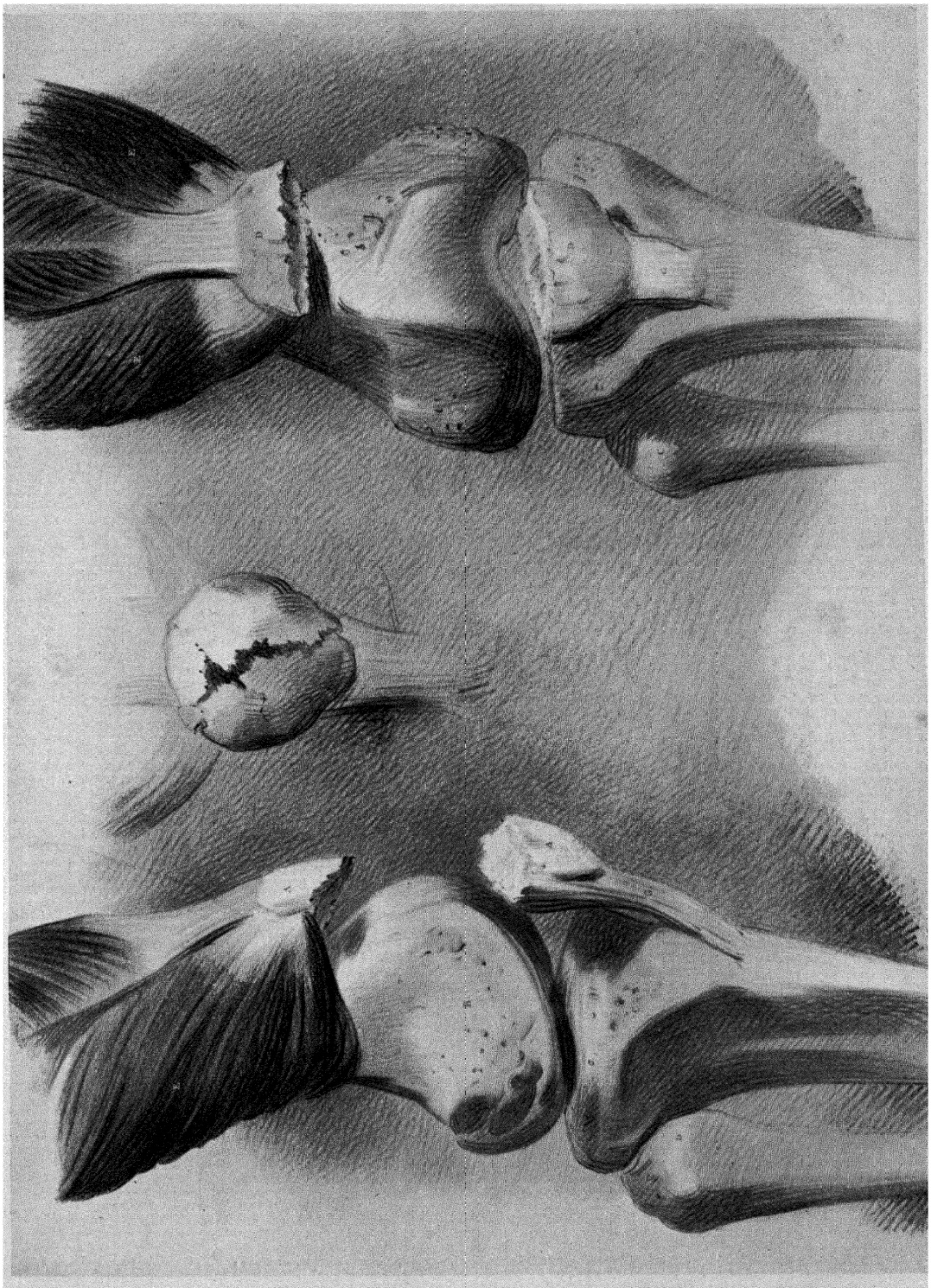
Fracture of Tibia below Tuberosities.— Also a rare injury, and results from direct violence as a rule, *e.g.* a kick; indirect violence, such as might lead to fracture of the lower end of femur, or a dislocation of the knee, may be responsible for it. The line of fracture may be oblique, and may enter knee-joint and cause synovitis.

*Diagnosis* depends chiefly on the increased width of the bone, tenderness on pressure, abnormal mobility, and crepitus. The use of an anæsthetic or the X-rays will aid the diagnosis.

*Treatment.* — Weight, extension, with the injured region left exposed to allow of massage, and compression by a bandage. Any tendency to varus or valgus should be watched for and corrected at once. After four weeks plaster-of-Paris may be applied.

Fracture from Compression of Tibia through its Articular Surface is caused by the forcible pressure of the tibia against one or other of the femoral condyles in a fall from a height on to the feet, occurs in falls from a dogcart, during mountaineering, or jumping off a bicycle. There may be simply a fissure traversing the joint surface, but in bad cases the end of the tibia may be crushed into two or more fragments, between which the shaft is impacted.

*The Signs* are great tenderness with increased width of upper end of tibia, perhaps abnormal lateral mobility. If fracture involves only one-half of tibial articular surface there may be a tendency to varus or valgus, the former being more frequent as the inner tuberosity is more



Fracture of Patella.

VOL. V. *To face page* 146.

mentous union, even if the fragments are kept close together.

*Treatment.*—1. *Non-operative Procedure.*—The hip should be flexed and knee fully extended to relax the quadriceps, and the limb placed on a straight splint, which may be in the form of an inclined plane, or of felt or poroplastic moulded to the back of the limb, and supported or slung to continue the relaxation. If tension in the joint be great the effused blood may be removed by aspiration. The fragments should then be brought together, and the torn aponeurosis and clots displaced as far as possible by rubbing the surfaces against each other. The limb should be fixed to the splint, and the fragments kept approximated as nearly as possible by two strips of plaster placed one above each fragment, the ends being crossed and fixed to the splint. Another method is by means of a strip of plaster 8 in. × 3 in., with one end curved to fit above the upper fragment. This is applied, and two elastic bands are fixed to each corner of the plaster, and then stretched on each side of the leg to be fixed to splint lower down. The plaster should be held in position by the bandage fixing the thigh to the splint. The quadriceps should be gently massaged daily, and occasionally a weak faradic current may be applied. After eight weeks the patient may be permitted to walk with crutches, a light splint or leather knee-cap being worn for some months; massage, and gradually increasing passive and active movement of the joint should be carried out daily. Fractures from direct violence, with no displacement of the fragments, should be kept on a splint for three to four weeks, and then passive movements commenced.

2. *Operative Procedure.* — (*a*) Malgaigne's hooks, which are now hardly ever used, are inserted into the fragments after preliminary puncture of the skin, and then screwed together. The result is generally fibrous union.

(*b*) Mayo Robson's method consists in the passage of two needles transversely — one through the quadriceps, and the other through the ligamentum patellæ, both close to the bone, and wiring the ends together.

(*c*) Twynam, by means of a special curved needle, passes subcutaneously a suture of silk or silver wire round the margin of the fragments, through the quadriceps and ligamentum patellæ. The fragments are placed in contact, the encircling suture drawn tight, knotted, and cut short.

(*d*) Barker's method of subcutaneous suture is carried out as follows:—A tenotomy knife is passed through skin and middle of ligamentum patellæ close to edge of lower fragment. A curved needle on a handle is passed through this track and behind the two fragments, being brought through quadriceps close to the edge of upper fragment to the skin. The skin is incised on the needle, and the knife introduced so as to split the tendon vertically between the needle and the edge of patella. The needle is threaded with strong silver wire, and withdrawn with one end of the suture. The needle is then passed between the skin and patella from the first puncture to the other, threaded with the other end of the suture, and again withdrawn. The fragments are approximated and rubbed together to remove clots, the blood in the joint is squeezed out through one of the incisions; the suture is then tied firmly, and the ends cut short. Passive movements may be commenced in three or four days, and the patient should be able to walk in five or six weeks. No splints should be used.

(*e*) Direct suture by opening the joint is the best procedure, so far as perfect coaptation is concerned. Must be done only under rigid aseptic conditions; is especially suited for cases which have not done well under treatment by splints. If it is decided to employ this method in a recent fracture, a few days should be allowed to pass to permit the swelling to subside, and to enable one to purify the skin. The fragments are best exposed by turning down a flap, which done, the joint should be cleared of clots and dried. The fragments should then be examined, and any drooping of aponeurosis over the edges raised up. The fragments may be sutured in various ways; silk, silkworm gut, or silver wire may be passed vertically round the fragments, or they may be drilled in two places, and a double suture employed. The ends of the wires should be twisted once or twice, and pressed level with the bone surface. The torn edges of aponeurosis should be united with a few catgut sutures. In old-standing cases the fractured ends should be sawn off before the fragments are united.

The result of such an operation is as a rule good, the patient being able to bend the knee freely in two weeks, and able to walk in three to four weeks. A knee-cap is unnecessary. Though the patella forms callus more slowly than any other bone, union is usually bony. The risk of refracture is comparatively slight.

Owing to unfortunate results from septic inoculation at the time of operation that have occurred from time to time, and also to the very fair results often obtained from non-operative treatment, wiring the fragments is far from becoming the routine practice. No other method gives such complete approximation of fragments and such firm union, but whether risk of operation is justified is still an open question.

Spontaneous Fractures are those which occur in a bone which is diseased; the bone, being weakened by the presence of the disease, breaks on the application of very slight violence.

The chief causes which lead to spontaneous fractures are:—

1. Atrophy of bone from age, disease; thinning

from pressure of an aneurysm, or simple growth, from tabes, general paralysis, and chronic brain diseases.

2. Fragilitas ossium, a fragile condition of bones, not associated with obvious atrophy, and often with a hereditary history.

3. Inflammation of bone, with subsequent necroses, abscess, or caries, of pyogenic, tuberculous, or syphilitic origin.

4. Rickets. Ossification is irregular and feeble, resulting bone being spongy and fragile. Union is much delayed, and may not occur until disease is almost cured.

5. Osteomalacia. The change consists in decalcification of bone and subsequent absorption of part of the constituents by the marrow. Bone gets thinner and thinner till it becomes a mere shell and disappears altogether, being replaced by the marrow.

6. New growths. Of simple tumours, the chondroma is the only one that ever results in fracture. Sarcoma, which is usually primary, and carcinoma, which is usually secondary, are much more frequent causes of spontaneous fracture. A hydatid cyst may be responsible for the fracture. (Rupture of ligamentum patellæ and quadriceps, *vide* Muscles and Tendons.)

### Epiphysial Injuries

Separation of the Lower Epiphysis of the Femur results from extreme *direct violence*, as the passage of a wheel over the lower end of the femur, and from *indirect violence*, giving rise to over-extension of the knee, together with violent twisting and traction on the leg, as when it is entangled in the spokes of a wheel in motion, and is carried round by it. This is the common mode of production (Hutchinson, jun., Barnard). Lateral flexion, or a force applied in a lateral direction, is best calculated to produce a separation of the epiphysis (Henry Morris). In young children a slight fall may cause the injury, especially if they are the subjects of some disease, *e.g.* syphilis, rickets, tubercle. It is sometimes met with in railway and lift accidents.

The majority of cases are met with in children and young adults between seven and fourteen years of age, although, theoretically, it may occur up to the end of the twentieth year, *i.e.* before complete bony consolidation between the diaphysis and the epiphysis has taken place. It is nearly always in boys.

In *partial separation* the line of cleavage between the epiphysis and diaphysis is incomplete, the periosteal sheath is intact, and there is no displacement. With *complete separation* the line of cleavage passes right across the bone between the epiphysis and diaphysis. This may be *simple* or *compound*.

*Simple without Displacement.*—In this condition the periosteal sheath is usually untorn, the epiphysis being merely loosened. If, however, the periosteum is torn the synovial membrane will be injured (for the epiphysis includes the whole articular surface), and acute synovitis of, or effusion of blood into, the knee-joint will follow. The breadth of the femur and the strength of the periosteum lessen the occurrence of displacement. There is swelling about the joint, pain on attempting to move the limb, and tenderness along the epiphysial line.

*Simple with Displacement.*—In this variety the thin posterior periosteum is perforated by the end of the diaphysis, which projects beneath the skin to one or other side of the popliteal space, usually the outer. The periosteal sheath is strongest on the front of the femur, and this band is rarely torn; hence, when displaced, the epiphysis is carried forwards, taking with it the tibia, to which it is attached by the popliteus and the strong crucial ligaments; at the same time it is drawn upwards and inwards by the quadriceps and adductors, and rotated backwards by the strong gastrocnemius, the two heads of which are in part attached to the epiphysis. The fractured end of the diaphysis is convex, and that of the epiphysial cartilage, which usually remains attached to the epiphysis, is concave, and when reduced it is not easy to displace them. If, however, the anterior band of periosteum is torn, the epiphysis is displaced backwards; it is then difficult to keep in position after reduction.

*Symptoms.*—There is marked deformity about the joint, increased girth around the knee, and shortening of the limb from two to four inches. Great swelling and ecchymosis in the popliteal space soon develop, with effusion into the knee-joint of extravasated blood and synovial fluid. The broad lower end of the diaphysis is felt at the outer side of the popliteal space. It does not move with the leg when the latter is moved laterally. The epiphysis is felt in front and to the inner side of the displaced diaphysis, and it moves with the leg. Soft cartilaginous crepitus may be felt when the two ends of the bone are in contact. There is abnormal mobility with hyperextension of the leg on the thigh, readily obtained under an anæsthetic.

*Compound Separation.*—With very severe injury the lower end of the diaphysis is forced through the skin, and projects to one or other side of the popliteal space, usually the outer.

*Immediate Complications.*—Separation may be complicated by a fracture of the diaphysis or an intercondyloid fracture of the epiphysis, or one or other condyle may be broken off, or a fracture of the upper end of the tibia. When there is displacement the popliteal vein may be pressed upon, producing œdema of the leg, and subsequent hæmorrhage from ulceration; or it may be wounded, giving rise to extravasation of blood into the surrounding tissues. If the artery is compressed the injured limb will be colder than its fellow; if wounded, an aneurysm

will form; when completely ruptured the pulse will be absent in the dorsalis pedis and the posterior tibial arteries, and gangrene will set in. The popliteal nerves may be pressed upon, producing pain in the leg and foot, or one or other nerve completely torn across.

*Later Complications.*—The vascular growing epiphysial cartilage when damaged is liable to be the starting-point of tuberculous disease or acute infective osteomyelitis. When the injury is compound, suppuration of the wound with acute periostitis and necrosis (and suppurative arthritis) accompanied by septic phlebitis and pyæmia frequently result. After union has taken place there may be limited flexion at the knee from incomplete reduction of the deformity and permanent hyperextension; fibrous or bony ankylosis, especially if there was previous suppuration in the joint; shortening of the femur from impaired growth, and premature ossification of the epiphysial disc, with secondary spinal curvature.

*Diagnosis.*—A partial separation is not easily distinguished from a *contusion of the bone*; tenderness localised along the epiphysial line is in favour of separation. In complete separation it may be necessary to administer an anæsthetic in order to make out the true nature of the injury. "Sometimes the displacement is so slight that the injury may easily escape notice, or be mistaken for a *traumatic synovitis*" (Howard Marsh).

From a *dislocation* of the knee make out the exact relation between the patella and the head of the tibia and fibula, also the movements in the joint. Note the abnormal mobility. Dislocation of the knee is rare in young subjects, and the joint is usually stiff and fixed, flexion and extension being difficult—a skiagram will settle the difficulty.

*Supracondyloid fracture* is rare in children, and the lower end of the upper fragment is more pointed and oblique, and it is more distant from the joint. The characteristic soft cartilaginous crepitus is pathognomonic of separation if it be present. In favour of separation we have the age of the patient as a guide, the absence of obliquity of the fragments, the nearness of the fracture to the joint, the smoothness of the fragments, and the great difficulty in effecting reduction.

*Prognosis.*—This is a very serious injury. It is attended when compound by a high mortality from shock and pyæmia. The ultimate result, however, in most cases that recover is good.

*Treatment.*—The following is advocated by Hutchinson, jun., and Harold Barnard:—

*Reduction.*—"Under complete anæsthesia an assistant makes steady but strong traction on the tibia in the line of the limb. This overcomes the upward pull of the quadriceps extensor and brings the epiphysis down to the line of the separation.

"The operator then clasps his hands beneath the lower part of the thigh and draws it steadily upwards, gradually flexing completely the knee and hip joints, while the assistant still keeps up the traction on the leg.

"This manœuvre causes the epiphysis to move back upon the fractured surface of the diaphysis until it has reached its normal position, and further movement is prevented by the periosteum coming into contact with the anterior surface.

"A bandage is then applied around the thigh and ankle, fixing the knee at an angle of about 60°.

"The limb is laid on its outer side on a pillow, and an ice-bag applied to the front of the knee to limit the effusion. This position is maintained for a fortnight.

"After fourteen days the limb can be extended under gas if necessary, and put up in plaster in a position about 30° short of the straight line, or it may be put on a MacIntyre splint and gradually extended. The plaster remains on from a fortnight to three weeks, and a little massage restores movement."

They conclude "that in extended position of the knee, even with an anæsthetic, reduction of the fragment is very difficult if not impossible.

"With method of full flexion reduction is always easy, the treatment is short, and it is the rule to obtain perfect movement in the knee without shortening or deformity of the leg."

Aspirate the joint if there be much effusion into it, and apply elastic pressure by means of a bandage.

When compound, the greatest care must be taken to procure asepsis. The adjacent skin and the projecting diaphysis must be thoroughly cleansed with soft soap and water, next with spirit, and lastly swabbed with and the wound syringed out with 1 in 2000 perchloride or biniodide of mercury, and an antiseptic dressing applied.

The wound may be completely closed, or a drainage-tube left in. A Hodgen splint will be found most convenient for redressing the wound, should this become necessary, without removing the apparatus. It may be necessary to resect the end of the diaphysis in order to effect reduction; this is rarely required in simple displacement.

Ligature of the popliteal artery or vein, or both, may be necessary to control hæmorrhage. When gangrene occurs the thigh must be amputated through its lower third, but only after all efforts to save the limb have failed. Should suppurative arthritis supervene, the joint must be freely laid open on each side of the patella and drained. Amputation for pyæmia may become a necessity later.

SEPARATION OF UPPER EPIPHYSIS OF TIBIA is rare, but its possible occurrence must be remembered in any case of severe injury of the

knee-joint in a child. Its rarity is probably due to the fact that the ligamentum patella, internal lateral ligament, and semimembranosus tendon are inserted partly into epiphyses and partly into diaphyses, thus strengthening their relations.

Complete separation of upper epiphysis is usually the result of a violent wrench of the leg, and is most liable to occur between twelve and sixteen years of age.

*Signs* are abnormal mobility, cartilaginous crepitus, and displacement, which is slight and consists of overriding of the epiphysis, usually forward, but occasionally inward and outward. Effusion into the knee-joint almost invariably occurs.

*Diagnosis.*—Free movement of joint, which is present in separation of epiphysis, serves to distinguish it from dislocation of the knee, which, moreover, hardly ever occurs in childhood.

Mobility at epiphysial level below the articulation is conclusive. When little or no displacement exists, it may be mistaken for a sprain.

*Prognosis.*—Good union, usually osseous, follows. Ankylosis from synovitis or suppuration in the knee-joint. Deformity may occur from incomplete or non-reduction. Premature arrest of growth as a result of this injury is rare; when it does occur the fibula is bowed out.

*Treatment.*—Any displacement present should be remedied by flexing the knee and manipulation, under an anæsthetic. When there has been little or no displacement, the limb may be put up in plaster-of-Paris at once, or lateral and posterior splints followed by plaster after two or three weeks. Where much displacement has existed, the better position is probably that of flexion on a MacIntyre or Hodgen splint. Primary amputation is only necessary when the injury is compound and accompanied by severe laceration of the soft parts. Secondary amputation may be called for by gangrene or suppuration in the joint.

Separation of Tubercle of Tibia.—The epiphysis includes the tubercle of the tibia, which is frequently developed from a separate centre, and may be torn off by a violent contraction of the quadriceps.

Separation of the tubercle occurs when springing from the ground, as in vaulting; the commonest time of life is between sixteen and twenty years. It may be mistaken for fracture of the patella. The fragment is drawn up by the quadriceps, and is freely movable in all directions. Active extension is impossible. Blood may be effused into the knee-joint; the fragment should be fixed in position by a steel peg; good union and use of limb result.

Separation of the Upper Epiphysis of the Fibula usually takes place before its union with the diaphysis, and between the ages of seven and fourteen, but it may occur after. Frequently its detachment accompanies that of the upper tibial epiphysis.

This injury may result from *indirect violence*, such as forcible contraction of the biceps while the knee is in a flexed position, or from *direct violence*, it having occurred while a case of knock-knee was being straightened.

The prominent *symptom* is pain on pressure over the head of the fibula. The fragment, which is readily movable, can be felt on the outer side of the knee-joint, being displaced upwards by the biceps, which is inserted into it. The external popliteal nerve is liable to injury, giving rise to pain along its distribution, and partial or complete paralysis of the peronei and extensor muscles.

*The diagnosis* depends chiefly on the age of the patient and the extreme mobility of the fragment. There may, however, be difficulty in distinguishing it from a sprain, especially if severe bruising be present, in which case a skiagram would be of assistance.

*The treatment* consists in the reduction of any displacement, and putting up the limb in the flexed position, to relax the traction of the biceps on the fragment. Massage of the joint should be commenced on the day following the injury, and passive movements in seven to fourteen days.

### Dislocations

*Dislocations of the Knee-Joint* are of very rare occurrence, and when met with are due to extreme violence. Are found in machinery accidents, where the leg has been violently twisted or wrenched, the thigh often being more or less fixed; also when men have fallen from a height.

The varieties of *complete* dislocation are—*forwards*, occurring during hyperextension of the leg, the head of the tibia lying in front of the condyles and drawn upwards sometimes as much as four inches; *backwards*, usually due to violence to front of leg or back of thigh, the head of the tibia resting behind the condyles. In both these forms the soft parts are extensively torn.

*Incomplete* dislocations are not likely to occur, and may be forwards, backwards, laterally, or oblique. In this group the articular surfaces are still partly in contact, and there is less destruction of soft structures.

Dislocation may occur laterally, combined with rotation of the leg on its long axis, usually outward.

*The soft parts* suffer very severely in complete dislocations, the lateral and crucial ligaments being extensively torn especially in the anterior and posterior varieties. The hamstring muscles may be torn across, and injury or rupture of the popliteal vessels and nerves may lead to gangrene of the leg. The dislocation is frequently compound.

Mr. Eames, in the *Brit. Med. Jour.*, April 21, 1900, mentions five cases of complete forward dislocation of knee all occurring at the same time, and caused by a fall down the shaft of a mine, and describes the appearances. The following were prominent signs:—

Great deformity.

Condyles of femur prominent and projecting back.

The skin behind joint on the point of bursting.

The skin of popliteal space may be torn across without opening joint cavity.

Head of tibia and fibula on anterior surface of femur and drawn up 2 inches.

A varying amount of effusion and extensive ecchymosis.

A fracture of tibia or femur near joint may accompany dislocation.

Reduction as a rule is easily accomplished under an anæsthetic by combined traction and direct pressure.

*Treatment.*—The limb should be placed in a flexed position on a splint or simply on pillows, arranging the limb in a position of greatest comfort. Hot anodyne fomentations or ice-bags should then be applied until the synovitis and extravasation have disappeared, which usually takes place in eight to fourteen days. During this time the toes should be watched for any sign of gangrene. Then a well-fitting leather knee-cap, extending well above and below the joint, should be fitted on, and at the same time gentle massage and passive movements commenced and continued daily. Any return of synovitis and pain would necessitate a cessation of massage until it subsided. After three to five weeks active movement may be gradually carried out. The splint should be worn for five or six months or longer, the recovery of full strength in the limb being often long postponed. Recovery may be accompanied by persistent œdema and eczema of foot and leg. Compound dislocations and those cases where gangrene threatens may require amputation.

*Spontaneous Recurrent Dislocation of the Knee-Joint.*—This very rare condition may be met with in infants. Dr. J. W. Ballantyne records a case in which the child, by simple pressure of one leg on the other, could slip the knee in and out laterally. A retentive apparatus was applied, and at sixteen months the child could walk well and had no tendency to dislocation. The condition is ascribed to congenital laxity of capsular and other ligamentous structures.

*Pathological dislocations* occur in connection with advanced disease of the knee-joint, the head of the tibia commonly passing backward and outward.

*Congenital fixed dislocations* may be met with, and are associated with rudimentary development or absence of the patella, or with obvious deformities of the articular surfaces of the femur and tibia.

Dislocation of Patella.—The patella is a sesamoid bone developed in the quadriceps tendon, and is not firmly fixed at the sides. Dislocation, which is rare, appears frequently to depend on some congenital defect in the patella or femur, especially of the external condyle, and usually takes place to the *outer side*, on account of the patella resting more on the outer than inner condyle, more especially if there be a tendency to knock-knee. The dislocation is *incomplete* when the joint surfaces remain in contact, and *complete* when the patella rests wholly on the outer surface of the condyle, the edge or one or other of the surfaces being in contact with the condyle. The dislocation may occur when the *knee is extended*, from strong contraction of the quadriceps causing the patella to glide directly over the outer condyle, or during *flexion* from a blow on the inner side of the bone, the force causing the patella to slide laterally in the groove between the femur and tibia. *Vertical dislocation* consists in the rotation of the patella on its vertical axis through an angle of 90°, so that one or other border rests in the groove between the condyles. It is described as *inward* or *outward* according as the cartilaginous surface of the patella is directed to the inner or outer side of the joint. It is usually due to direct violence, though occasionally to muscular action. The inward variety is perhaps the more common. The bone may be twisted completely round, the articular surface becoming anterior.

*Diagnosis* is usually easy, except perhaps in the rare condition of a complete rotation of the bone.

*Treatment.*—Reduction as a rule readily effected by direct pressure after relaxation of the quadriceps, by extending the knee and flexing the hip. There may, however, be considerable difficulty in vertical dislocations. After reduction a leather knee-cap should be worn, well padded over external condyle to prevent return of the dislocation. Operative treatment has until lately been as a rule unsuccessful. Recently mooring of the patella to the inner side of the joint after division of the capsule on the outer side has been followed by permanent success.

*Dislocation of the Semilunar Cartilages.*—Of the two cartilages, the internal is displaced twice as often as the external, and this is ascribed to the following facts: (*a*) that it is more firmly fixed than the external; (*b*) that a greater degree of rotation outward is possible, thus greater strain can be brought to bear on its attachments; (*c*) that the ordinary position of the foot and leg lends itself to a greater liability to the production of outward rotation. Usually the anterior attachment is torn; very rarely is the cartilage completely detached or divided.

*Production.*—The injury is produced by strong rotation of the lower end of the femur when the

knee-joint is bent and the tibia fixed, as when playing a stroke at golf. Less frequently, violent rotation of the tibia with the femur fixed may result in displacement of a cartilage.

The cartilages move with the tibia in flexion and extension. In rotation the tibia rotates beneath the cartilages, one or other being fixed.

During external rotation the external cartilage is fixed, and the internal is apt to slip through the gradually increasing gap that is formed between the tibia and the internal condyle of the femur.

The reverse occurs during internal rotation; but the external cartilage being smaller, rounder, and more mobile than the internal, is less frequently nipped between the bones when it slides into the gap.

This injury rarely occurs in a perfectly normal joint, being commonest among those whose knee-joints are liable to have great strains thrown suddenly upon them, such as football players.

*Symptoms and Diagnosis.*—After a twist of the leg there is sudden and intense pain in the knee, often causing the patient to fall, with the joint fixed in the flexed position. The knee rapidly becomes swollen, and on movement being attempted the joint locks on extension, but can be flexed quite freely. On extension the pain is increased, and the patient may feel that something has become "jammed" in the knee. There is pain on pressure over the joint line, and a projection is often felt which may be slightly movable. In old-standing cases a clicking may be felt on flexing and extending the joint. If the detached end of the cartilage remains in the centre of the joint a depression may be felt in the position of the cartilage, but the diagnosis of this variety from a loose body presents considerable difficulty, especially if there be much effusion.

*Treatment.* — In recent cases the cartilage should be replaced under an anæsthetic, if necessary. The procedure consists in flexing the knee completely, and then rotating the leg inwards or outwards according as the internal or external cartilage respectively be displaced. While rotation is maintained, suddenly extend the leg; at the same time press the projecting edge of the cartilage into the joint. After reduction, rest on a splint and elastic pressure are necessary. If the patient will permit, a plaster-of-Paris case should now be applied, and the limb used as little as possible for several weeks. This method, which gives the best chance of cure short of operation, is rarely tolerated, and one has then to resort to a knee-cap to control the movements of the knee as much as possible while allowing the patient to go about. He should be warned to avoid any rotating movement of the limb, walking with his toes in if the internal cartilage has been affected, and out if the external. The knee-cap should be worn for three or four months.

Recurrence is frequent from slight twists, and subsequently the cartilage is apt to slip out on the slightest provocation, thus interfering with an active life. These are the cases for operative interference. An apparatus such as Ernst's may be tried, but it is often very irksome and not always efficient.

The *operative* procedure may consist in either suture or removal of the cartilage. The results of both are about equal, though probably removal is the better, because a cartilage which has been sutured may get loose again. Either operation may be performed through an incision over the joint line on one or other side, according to the cartilage affected. The incision extends between the ligamentum patellæ and the internal lateral ligament for the internal cartilage, and between the ligamentum and biceps tendon for the external. The line of this incision in the capsule should be above the position of the cartilage. The detached portion of cartilage, which may be doubled over, is either placed in position and stitched to the fibrous capsule with catgut, or removed. It is advisable to explore joint for a possible loose body at the same time. The incision should be completely closed in layers. After-treatment consists in controlling the joint for five or six weeks, at first in bed with a posterior splint. When the wound has healed, use plaster-of-Paris or a moulded poroplastic splint. At the end of four weeks, movements should be commenced, and the patient gradually allowed to walk with a bandage on the knee. Longer confinement is necessary after suture than after removal.

*Detachment of part of articular cartilage from the femur* is an injury that may occur as the result of very slight violence. During flexion of the knee it is possible to produce a certain amount of internal and external rotation, and also some abduction and adduction, the knee-joint not being a simple hinge. If, when the knee is bent, the bones are pressed together with a lateral twisting, a portion of cartilage with spongy bone attached may be forced off the femur. This portion may become completely loosened, and form a foreign body in the joint, or may remain hanging as a loose body; in either case it should be removed.

*Rupture of posterior crucial ligament* may occur as the result of a violent blow on the anterior surface of the head of the tibia. The injury is followed by synovitis and subsequent weakness of the knee. On examination the head of the tibia can be slightly displaced backward into the popliteal space, when the knee is bent at right angles and the foot steady on the ground. For such a condition, supporting apparatus should be worn for some months.

**Kneippism.** *See* CURE (*Kneipp Water Cure*).

**Knock-Knee.** *See* DEFORMITIES (*Genu Valgum*); HIP-JOINT, INJURIES OF (*Coxa Vara, Results*).

**Knots.** *See* CLOVE HITCH; PREGNANCY, DISEASES OF PLACENTA AND CORD (*Knots on Cord*); REEF KNOT; STAFFORDSHIRE KNOT; etc.

**Koch Treatment.**—The use of tuberculin in the treatment of tuberculosis.

**Kocher's Method.**—A plan for the reduction of a dislocated shoulder. *See* SHOULDER, DISEASES AND INJURIES OF (*Dislocations, Treatment*).

**Kocher's Operation.**—An operation for the radical cure of inguinal hernia (*see* HERNIA, *Radical Cure*); also an operation for carcinoma of the tongue (*see* TONGUE, *Carcinoma, Treatment*).

**Koenig's Rods.** *See* AUDITORY NERVE AND LABYRINTH (*Localisation of seat of Lesion producing Nerve-Deafness*).

**Koilonychia.**—Spoon-nails, nails concave on the surface. *See* NAILS, AFFECTIONS OF THE (*Alterations in Curvature*).

**Kola Nut.**—The fruit of *Sterculia acuminata*, known also as gourou, kokkorokou, or bissy-bissy; a native of the West Coast of Africa; used as a digestive tonic, in diarrhœa, and in heart disease.

**Kolpo.** *See* COLPO.

**Kombé.**—An African arrow poison, made from strophanthus (*q.v.*).

**Kopftetanus.**—A special form of traumatic tetanus in which the facial nerve is involved, causing facial paralysis in addition to trismus; cephalic tetanus. *See* TETANUS (*Clinical Features*).

**Koplik's Spots.**—Red spots with bluish centres sometimes seen on the mucous membrane of the cheeks and lips before the cutaneous eruption of measles appears. *See* MEASLES (*Symptoms*).

**Korsakoff's Syndrome.**—A combination of symptoms, or *Symptom-complex*, met with sometimes in chronic alcoholics in which there is loss of memory, "pseudo-reminiscences" (such as stories of people seen or journeys undertaken), and occasionally peripheral neuritis; psychosis polyneuritica.

**Kötschenowa.** *See* BALNEOLOGY (*Russia*).

**Koumiss.**—Fermented mare's milk (lactic and vinous fermentation), easily digested in weak states (convalescence) and useful in phthisis. *See* DIET (*Milk and its Products*); HEART, AFFECTIONS OF MYOCARDIUM AND ENDOCARDIUM (*Treatment, Diet*); INFANT FEEDING (*Milk Preparations, Koumiss*); INVALID FEEDING (*Koumiss*); MILK (*Therapeutic, Koumiss or Kumys*).

**Kousso.**—Also known as Cusso; is obtained from the dried flowers of *Brayera anthelmintica*. It contains a neutral principle called Koussin, and tannic acid. *Dose*—$\frac{1}{4}$-$\frac{1}{2}$ ʒ.

It is used as an anthelmintic for all kinds of tapeworm, administered in the form of an infusion. It is rarely prescribed in this country.

**Kra-Kra.** *See* CRAW-CRAW.

**Krameriæ Radix.**—Rhatany Root. The dried root of *Krameria triandra*. It contains Rhatanhia-tannic acid, to which it owes its astringent properties. *Preparations*—1. Tinctura Krameriæ. *Dose*—$\frac{1}{2}$-1 ʒ. 2. Liquor Krameriæ Concentratus. *Dose*—$\frac{1}{2}$-1 ʒ. 3. Infusum Krameriæ. *Dose*—$\frac{1}{2}$-1 ℥. 4. Extractum Krameriæ. *Dose*—5-15 grs. 5. Trochiscus Krameriæ. 1 gr. of the extract in each. 6. Trochiscus Krameriæ et Cocainæ. 1 gr. of the extract and $\frac{1}{20}$ gr. of hydrochloride of cocaine in each.

The extract is added to tooth powders as an astringent when the gums have a tendency to bleed readily. The powdered root may be insufflated to check epistaxis. The trochisci are employed in relaxed conditions of the throat, and the infusion can be used as a gargle for the same purpose. Internally, krameria is administered as an astringent for the purpose of checking diarrhœa. It is of no value as a remote hæmostatic.

**Kraske's Operation.** *See* RECTUM, DISEASES OF (*Cancer, Treatment, Excision of Rectum*).

**Kraurosis.**—An atrophic condition affecting the vulva characterised by drying and shrivelling (κραῦρος, dry) of the parts, with pain in and irritation of them. *See* VULVA, DISEASES OF (*Kraurosis Vulvæ*).

**Krause, Glands of.** *See* LACHRYMAL APPARATUS (*Anatomy*).

**Kreatin and Kreatinin.** *See* CREATIN.

**Kresamine.**—An antiseptic preparation containing trikresol and ethylendiamin (25 per cent of each); it has been used in skin diseases (as solution ($\frac{1}{2}$ per cent) or ointment).

**Kresol.** *See* URINE, PATHOLOGICAL CHANGES IN (*Aromatic Substances*).

**Kreuznach.** *See* BALNEOLOGY (*Germany*); MINERAL WATERS (*Muriated Waters containing Bromine and Iodine*).

**Krishaber's Disease.**—Vertigo with sensory illusions, cardiac irritability, etc.

**Krohne and Sesemann's Apparatus.** *See* Anæsthesia, General Physiology of (*Regulating Chloroform Inhaler*).

**Kromskop.**—An apparatus for the reproduction in colours of photographed natural objects (*see International Clinics*, s. x., vol. ii. p. 1, 1900).

**Krypton.**—A gas found in small quantities in the atmosphere.

**Kubel's Method.**—A method of estimating the amount of organic matter in water, from the amount of oxygen required to oxidise it.

**Kubisagari.**—Endemic paralytic vertigo occurring in Northern Japan.

**Kudowa.** *See* Balneology (*Germany, Silesia*).

**Kusatsu.** *See* Balneology (*Japan, Sulphurous Thermal Waters*).

**Kutubuth.** — Melancholia with restless and inconsequent wandering to and fro; an old term used by the Arabian physicians for a morbid mental state said to occur chiefly in the month of February.

**Kyestein.** *See* Kiestein.

**Kyphosis.** *See* Spine, Surgical Affections of (*Kyphosis*); *see also* Chest, Deformities of (in *Kyphosis*); Deformities (*Cerebral and Spinal Paralysis*); Labour, Prolonged (*Pelvic Deformities, Kyphotic*); Rickets (*Clinical Features*); Syringomyelia (*Symptoms, Kyphosis*).

**Kythmos.** *See* Balneology (*Greece*).

**Labia.**—Lips (plural of *labium*), especially *labia oris* (lips of the mouth), and *labia pudendi* or *labia vulvæ* (margins of the vaginal orifice). *See* Generation, Female Organs of (*Vulva*); Gonorrhœal Infection (*Œdema of labia*); Hernia (*Inguinal, Diagnosis, Labial Swellings*); Pregnancy, Physiology (*Changes in External Genitals*); Pruritus; Puerperium, Physiology (*Evidences of Recent Delivery*); Syphilis (*Acquired, Primary Sore, Situation*); Uterus, Malformations of (*Hypertrophy of Labia Minora*); Vulva, Diseases of.

**Labidometer.** — An instrument for measuring the fœtal head; it consists of a pair of obstetric forceps with a graduated scale attached.

**Labio-.**—In compound words *labio-* (from Lat. *labium*, a lip) means relating to the lips, *e.g.* labio-dental, labio-nasal, labio-glossal, labio-mental, etc.

**Labis.**—The forceps (Gr. λαβίς, a forceps).

**Labitome.**—An instrument for diminishing the size of the fœtal head in cephalotomy; a pair of cutting forceps or a cephalotome.

**Labium.** *See* Labia.

**Labour.**—This will be described in the following sections:—

A. PHYSIOLOGICAL SECTION

1. **Physiology.**
2. **Progress and Duration.**
3. **Diagnosis and Mechanism.**
4. **Management.**
5. **Labour in Multiple Pregnancy.**

B. PATHOLOGICAL SECTION

6. **Precipitate and Prolonged Labour.**
7. **Faults in the Passenger.**
8. **Accidental Complications affecting Child only.**
9. **Retention of Placenta.**
10. **Post-Partum Hæmorrhage.**
11. **Injuries during Labour.**
12. **Operations.**

*See also* Achondroplasia (*Clinical Features, Labour in Achondroplasic Dwarf*); Asphyxia (*Causes, During Birth*); Cocaine (*Uses, in Labour*); Coccygodynia (*Etiology, Traumatism in Labour*); Fœtus; Generation, Female Organs of; Hypnotism (*Obstetrical*); Medicine, Forensic (*Signs of Delivery*); New-born Infant; Pregnancy, Diagnosis; Pregnancy, Affections of Generative Organs; Pregnancy, Hæmorrhage; Pseudo-cyesis; Puerperium, Physiology; Puerperium, Pathology (*Puerperal Infection*); Puerperium, Pathology (*Puerperal Insanity*).

## A. PHYSIOLOGICAL SECTION

### Physiology of Labour


Definition . . . . . 154
Causes of . . . . . 154
Difficulties of . . . . . 155
Stages . . . . . 155
Factors—
(1) *Powers* . . . . . 155
*General Description* . . . 156
*Action in various Stages* . . 156
(2) *Passages* . . . . . 159
*Hard and soft* . . . . 159
(3) *Passenger* . . . . . 161
(*a*) *As a whole* . . . 161
(*b*) *The Fœtal Head* . . 161
Relation of Passenger to Passages . 162


Labour may be defined as the separation and expulsion of the contents of the gravid uterus, and is the physiological termination of pregnancy.

Causes of Labour.—The normal period of

human gestation is probably 273 days, as evidenced by the statistical records of Leuchardt and Leuwenwardt; and the period of expulsion is conveniently calculated to occur at the tenth menstrual period missed, or 280 days from the first day of the last period. As is well known, this date is by no means exact, as it is impossible to determine in most cases the date of fertilisation of the ovum.

The reason why labour should occur at a specific time has been attempted to be explained by many elaborate theories, each and all of which can be met by insuperable objections; so it must still be considered as one of nature's many mysterious secrets.

The theories advanced may to some extent explain the causation of labour, but give no clue to its onset at a given time. Thus the researches of Friedlander, Leopold, and Kundrat have demonstrated that the penetration of multinucleated cells into the placental sinuses during the later months of pregnancy lead to coagulation of the blood, and to the formation of young connective tissue which obliterates the sinuses, and thus tends to increase the amount of venous blood in the remaining active portion of the placenta, which causes irritation and uterine contraction.

Brown-Séquard has tried to show that the excess of $CO_2$ circulating the veins of the gravid uterus, acts in a like manner.

Others assert that labour is induced by a fatty degeneration of the decidua vera which predisposes to separation of the ovum and its subsequent expulsion, while some authors consider that there is an increasing irritability of the uterus with strengthening contractions, which acquire a special strength at the tenth menstrual period missed, and cause separation and expulsion.

Doubtless there is much that is true in many of the theories advanced, and probably several acting in unison may account for the onset of labour, but it is needless to say that none give even the slightest evidence of why it normally occurs at a given time. Natural selection seems alone to direct us on reasonable lines. Children born before this period are puny and ill able to lead an independent existence; while children born later are so large that their expulsion has incurred risks both for the mother and themselves. By a process of heredity it will be evident that the survivor of the fittest, or the majority of survivors, will be born on the 273rd day of gestation, and will thus develop and fix a period which will represent the habitual period of human gestation, or, in other words, assure the onset of labour at a given time.

Difficulties of.—In the human female labour is an extremely finely-balanced complex process, and thus as a rule requires many hours for its completion; the smallest hitch in the normal mechanism tends towards indefinite delay and serious complications.

The difficulty of labour in woman as compared with the lower animals is mainly to be accounted for by the difference in the pelvis and pelvic floor necessitated by the erect posture, although at the same time the comparatively large size of the fœtal head must also be taken into account. As will be noted upon the description of the factors of labour, the passages through which the ovum has to pass are curved and irregular in shape, while the pelvic soft parts or floor are thick and compact to afford support to the abdominal and pelvic viscera. In the lower animals, on the other hand, the parturient canal is straight and regular, while the pelvic soft parts are lax and thin. If proof were wanting of the difficult nature of labour in woman, it is to be found in the marked thickness of the uterine wall as compared with that of the lower animals, which is evidence of the greater force required for the expulsion of the contents. The difficulties and dangers of labour vary greatly in different types of the human race, and it may be generally stated that the higher the grade the more difficult does the process tend to become. This is probably due to the coexistent increase in the size of the fœtal head dependent upon intellectual development. The higher social grades of the same type seem also to have more difficult labour, as evidenced by the higher mortality; this, though perhaps due to a slight extent to a similar cause, is doubtless exaggerated by the want of physical development. The expulsion of male children, from their larger size, is more difficult and dangerous than female children, the mortality to the mother being about 40 per cent greater. As is natural to expect, first labours are more difficult than subsequent ones, from the want of previous dilatation of the canal.

Stages.—For the sake of description the period of labour is differentiated into three stages:—

(1) The stage of preparation, from the commencement of pains till the full dilatation of the cervix, or, in other words, till the complete canalisation of the genital canal (1st stage).

(2) The stage of expulsion of the child, from the full dilatation of the cervix till the birth of the child (2nd stage).

(3) The stage of separation and expulsion of the placenta and membranes from the birth of the child till the birth of the secundines (3rd stage).

### Factors of Labour

Embraced in the process we have to consider three factors, viz., the powers, the passages, and the passenger. The powers are threefold: (1) the uterine contractions, so-called primary powers; (2) voluntary muscles, specially those of the abdomen, so-called secondary powers; and (3) the weight of the ovum.

*Primary Powers, or Uterine Contractions.*—

These must be looked upon as by far the most important factor in expulsion, in so far as they alone are able to complete the process as evidenced in cases of paraplegia and complete anæsthesia. In their action they are intermittent; each contraction lasts for a period of from thirty to a hundred seconds, with a varying interval which is most regularly marked in the second stage. By means of this intermittency exhaustion of the mother is prevented, the placental circulation is not embarrassed, and accommodation of the passenger to the passages is favoured. Uterine contractions are purely involuntary, although they may be influenced mentally—a point of importance in the management of labour, in so far as we know that encouragement stimulates, while on the other hand depression tends to diminish their action.

Doubtless the centre of nervous stimulus lies in the sympathetic ganglia, although a spinal centre has been described in the lumbar enlargement of the cord and a cerebral centre in the medulla. It has been stated that the intermittency is due to paralysis of the terminal nerve filaments in the uterine wall, induced by contraction of the uterus, and probably influenced by the resulting anæmia. The contractions are usually associated with painful sensations, hence the common expression of "pains" applied to them. These painful sensations vary in the different stages of labour. In the first stage they are of a cutting nature, while in the second stage they may be described as of a bearing-down, tearing character. Along with the temporary contractions of the uterine walls and their intervening relaxation we have at the same time a permanent shortening of the muscular fibres, known as retraction. By this means not only are the individual fibres permanently shortened, but also there is a redistribution of their arrangement.

We have thus in the action of the primary powers a double effect: (1) a temporary marked shortening of the individual fibres, "contraction"; and (2) a permanent slighter diminution in length, "retraction."

Uterine contractions are not peristaltic.

*The Secondary Powers.*—As has already been stated, these are mainly supplied by the contractions of the abdominal muscles and diaphragm. They not only are of value in assisting the primary powers in their expulsive efforts, but also are beneficial in preventing the effects of excessive retraction, and maintaining the long axis of the uterus in the axis of the pelvic brim. Though not absolutely essential, their absence or impaired action seriously delays the completion of labour, a point of great practical importance in reference to the question of anæsthesia during parturition. To a great extent the secondary powers are reflexly stimulated to act, but at the same time their force is markedly influenced by mental control, the parturient being able to voluntarily assist in the expulsive efforts.

*The Weight of the Viscera.*—This is a factor of very minor importance, but may have a slight effect when the patient is in the erect posture by assisting dilatation of the cervix. Thus the parturient should be encouraged to walk about during the first stage.

*Action of the Powers in the different Stages of Labour.*—During the first stage the uterus is practically alone concerned in the dilatation of the cervix, and for a clear conception of the manner in which this is completed a general knowledge of the disposition of the muscular fibres of the organs is necessary. At full time the uterus is to be considered as consisting of three distinct portions, viz., the body, lower uterine segment, and cervix (see Fig. 1). They are in a general manner to be differentiated from each other by their relationship to the peritoneal investment; on the body the peritoneum is closely adherent, on the lower uterine segment it is loosely attached, while the cervix has practically no peritoneal covering whatever. Of these three portions the body alone actually contracts, and is to be considered a power; the lower uterine segment and cervix are purely passive, and are in truth passages. The muscular fibres of the body are irregular in their distribution; those of the lower uterine segment are mainly disposed in a longitudinal direction, while in the cervix the disposition of the pelvis is mainly circular.

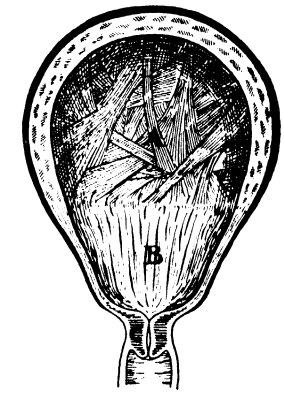



FIG. 1.—Diagram of full-time gravid uterus showing main disposition of fibres. A, Body proper; B, lower uterine segment (longitudinal); C, cervix circular.

With the onset of labour retraction of the body of the uterus commences. This is at first associated with the painless uterine contractions which are ever present during pregnancy. As the result of retraction the cavity of the body proper is permanently decreased in size, and the muscular wall at the same time becomes thicker. The retraction of the body pulls upon and lengthens the lower uterine segment, and through it upon the cervix. The cervix is thus pulled upon in an upward direction, and its passive circular fibres (commencing at the os internum) gradually yield from above downwards, with the result that the canal of the cervix is dilated, and now forms part of the general uterine cavity which thus compensates for the diminution in the body proper (Figs. 2, 3, 4). During this period the patient is usually unaware of any active changes occur-

ring, although she experiences a sensation of the uterine tumour having fallen somewhat; this is generally associated with a feeling of more easy respiration, which has been called the "lightening" before labour. Vaginal examination at this stage will show the cervix to be shortened, but the os externum usually closed in primiparæ.

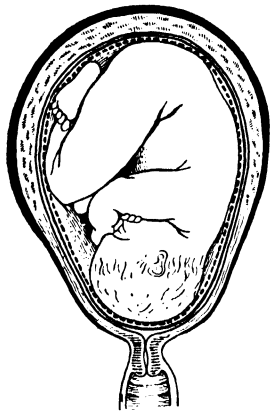


FIG. 2.—Full-time gravid uterus. Primiparæ, showing cervix closed. The dotted line demonstrates fœtal ovoid.

In due course uterine contractions become stronger and are associated with painful sensations; the patient now passes into the active first stage of labour. During this stage a similar though increased action of the uterus continues; the body proper contracts and retracts with a corresponding increase in the traction on the lower uterine segment, which results in a slight lengthening of this portion of the uterus and a gradual dilatation of the os. Should there be difficulty in the dilatation of the cervix through rigidity or other causes, an increased strain is thrown on the lower uterine segment, which causes it to become more and more lengthened and correspondingly thinned (*see* Rupture of Uterus, *under* INJURIES DURING LABOUR). In consequence of the continued retraction of the body proper with the associated increased thickness of its walls, and at the same time the thinning of the lower uterine segment, there is formed a sharp line of demarcation between these two portions of the uterus throughout its circumference; this is known as the "retraction ring" (Fig. 5). As is to be expected, the greater the retraction and corresponding thinning of the lower uterine segment, the more marked does this ring become; its detection, therefore, may be of much practical value as evidence of impending rupture. With the completion of the first stage and full dilatation of the os there is an entire absence of any constriction formed by the cervix. The uterus and vagina now form one smooth, continuous canal—"complete canalisation." During this stage but slight descent of the ovum occurs, the main effect having been the stripping of the cervix and lower uterine segment off the lower pole.

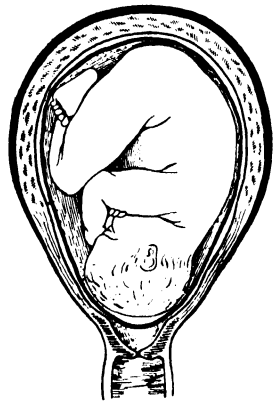


FIG. 3.—Commencing dilatation of cervix before the actual onset of labour pains; stage of "lightening" before labour.

Full canalisation having been completed, expulsion and descent of the ovum now take place, and the second stage of labour commences.

The uterine force is now assisted by contractions of the abdominal and thoracic muscles, and by their combined efforts the child is expelled. The combined force has been variously estimated at from 17 to 57 lbs. to the square inch. The direction of the applied forces is downwards and backwards in the axis of the brim of the pelvis.

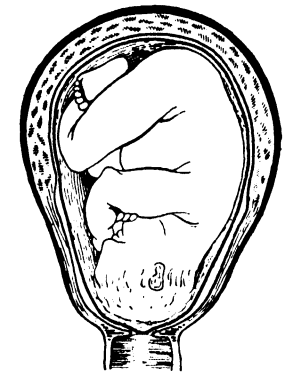


FIG. 4.—Commencement of first stage with early dilatation of os externum.

*Third Stage.*—For the completion of this stage both the primary and secondary powers are called into action, though not in combination. By contraction and retraction of the uterus the placenta and membranes are separated and expelled from its cavity, their further expulsion and birth to be completed by the unaided action of the secondary powers.

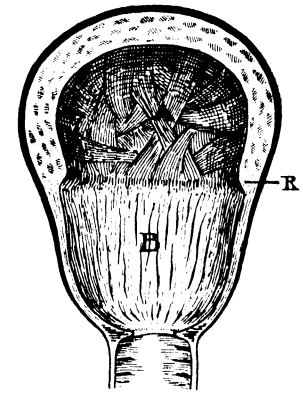



FIG. 5.—Gravid uterus towards end of first stage, showing A, body proper retracted; B, lower uterine segment lengthened; and R, retraction ring.

In the first stage there is exercised on the ovum by the contractions of the body of the uterus a general pressure, which is transmitted at right angles to its surface. The entire superficial area of the ovum is therefore acted upon, except the lower pole, which is in contact with the passive lower uterine segment. Pressure is by this means conducted through the ovum upon the lower uterine segment, which yields and lengthens; at the same time the uterus retracts, and, by dragging on the cervix, opens up its canal; into this the ovum bulges, and transmits pressure laterally on its walls, and thus assists in the further dilatation. Also, from the pulling-up of the cervix through retraction the cervix is further dilated, and the

lower pole of the ovum separated from the uterine wall. As a result, the lower pole of the ovum is exposed and presents. This normally consists of the membranes and a quantity of contained liquor amnii, the so-called bag of forewaters. After full dilatation of the cervix the bag of forewaters usually ruptures. From the close adaptation of the soft parts of the pelvic canal around the presenting part (girdle of contact), the liquor amnii which surrounds the fœtus in the uterine cavity is prevented from escaping *en masse* along with the forewaters. During the subsequent stages of labour it escapes gradually. Its retention is of much value in preventing a complete moulding of the uterine wall to the body of the fœtus, which would thus seriously compress the placenta and obstruct the circulation within it.

Full dilatation of the cervix and rupture of the membranes, with escape of forewaters, terminates the first stage, and descent of the fœtus now commences.

The duration of the first stage varies greatly. As can only be expected, it is normally much longer in primiparæ from want of previous dilatation. Calculating from the time when pains occur at fairly regular intervals of from five to seven minutes, the average first stage may be described as occupying eight to ten hours in a primipara, and five to seven hours in a multipara.

Descent of the fœtus now commences, and opposition is offered by the bony pelvis and pelvic floor. The former is overcome by a process of accommodation of the presenting part to the irregular passages, in the course of which a complicated though definite mechanism is undergone and described (see p. 174). This mechanism of accommodation is entirely due to the combined efforts of the powers and resilient pelvic floor. The presenting part is impelled downwards during the pains, and through the resiliency of the pelvic floor recoils after each contraction ceases. By this means a constant up-and-down movement is maintained, which favours and secures the transit of the presenting part through the most available channel. At the same time, by means of the pliability of the presenting part it becomes moulded, and thus adapted to the varying available space. The passage of the child through the compact pelvic floor is rendered possible by the mobility of its pubic and sacral segments. The former is drawn upwards by the retraction of the uterus, while the latter is forced downwards by the pressure of the advancing part, as if by a folding-door mechanism; the door is thus opened up and the expulsion of the child facilitated.

The descent of the fœtus is almost entirely confined to the presenting head; the breech or upper pole of the fœtal ovoid may be found to be exactly at the same level when the head appears at the vulva as at the commencement of labour. This is explained by the pliability of the fœtal ovoid, which, from the pressure exercised upon it from all sides except at its lower pole, is elongated by the straightening of its vertebral column (Figs. 6, 7, 8). After the birth of the head a short interval of rest occurs, after which contractions recur, and the body is expelled. The duration of the normal second stage may be said to average three hours in a primipara, and two hours in a woman who has previously borne children.

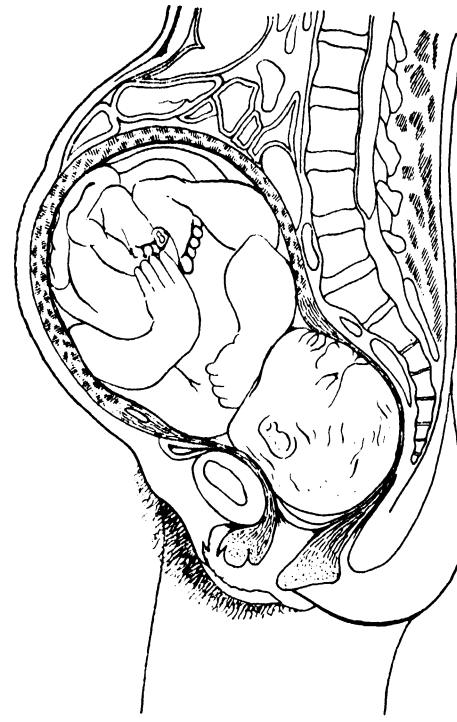


FIG. 6.—Commencing labour, showing complete attitude of fœtal flexion.

During and after the expulsion of the fœtus the tonic retraction of the uterus causes it to firmly compress the decreasing uterine contents. Thus, after the birth of the child the uterus closely surrounds the secundines (placenta and membranes). Intermittent uterine contractions continue, and cause their separation by diminishing the area of attachment, and after separation expel them into the vagina, from which they are forced by the unaided action of the secondary powers, and born. The tonic retraction of the uterus permanently maintains the closure of the uterine sinuses at the site of the separated placenta, and thus prevents excessive hæmorrhage, while recurring intermittent contractions expel from the uterus any blood which may ooze.

Separation of the placenta does not commence till after the birth of the child, and is attained by a process of what is known as detrusion. This consists in the extensive diminution of the

placental site by contraction, and retraction of the uterine wall to such a small area (4 in. × 4½ in.)

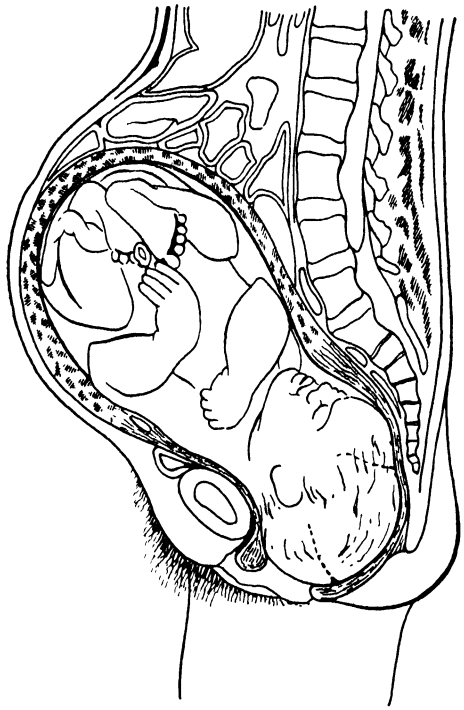


FIG. 7.—Labour, second stage, showing commencing straightening of fœtus.

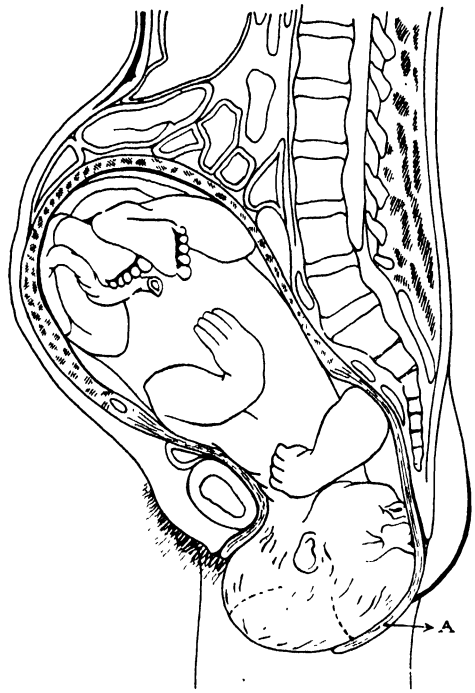



FIG. 8.—Birth of head, showing extension of fœtal ovoid. A, thinning of pelvic floor projection.

that the placenta, though semi-elastic in consistence, is torn from its attachment. Detachment, as observed from a series of frozen sections of the third stage of labour, would appear to occur gradually from below upwards, and thus when completely separated the organ is expelled, doubled up in an elongated form, the entire process of separation and expulsion from the uterus being due to the same cause. Separation of the membranes from the body proper also occurs only during the third stage. They are partially detached in a similar manner to the placenta by diminution of their area of attachment through contraction or retraction of the uterus; by this means they are thrown into a series of wavy ridges. Their complete separation is only attained by the traction of the placenta during its expulsion.

The amnion and chorion are of different elasticity, and thus form ridges independently of one another, the intervening layer being stretched and lacerated. If strong adhesions exist, therefore, between the chorion and the uterine wall, it is no uncommon thing to have the amnion expelled entire with the placenta, leaving the entire chorion *in utero*, a condition very apt to be overlooked when examining the secundines to ascertain their complete expulsion. The average duration of the third stage is about twenty minutes.

After labour, retraction and contraction are so complete that no space exists in the uterine cavity; the uterine walls, which are 1¼ to 1½ inches thick, are firmly apposed to one another. Occasionally a blood-clot, continuous with the thrombi in the vessels at the placental site, may be present. The post-partum uterine cavity from the external os to the fundus measures about 7½ inches.

### PASSAGES

The passages, for convenience of description, may be divided into hard and soft—the former are represented by the bony pelvis, and the latter by the lower uterine segment, cervix, and vagina.

The bony pelvis, which forms the boundaries of the hard canal through which the uterine contents pass during labour, is restricted to what is known anatomically as the true pelvis. This, from its irregular shape, is difficult to describe so as to give a clear conception of its nature. For this purpose three planes may be drawn at different levels, the dimensions of which will serve to show the varying nature of the contour of the canal: (Fig. 9) the first, at the upper level, which is known as the brim, inlet, or superior strait; the second, at the level of a line drawn from the middle of the symphysis pubis to the junction of the second and third sacral vertebræ, is known as the cavity; and the third, known as the outlet or inferior strait, from the lower border of the symphysis pubis to the tip of the coccyx. So as to estimate the irregular nature of the canal

the dimensions of each plane are determined by measuring the antero-posterior, the transverse, and oblique diameters.

The antero-posterior or conjugate diameter at

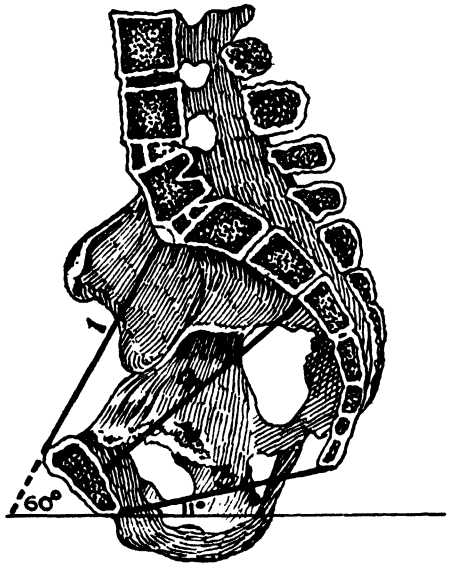



FIG. 9.—Vertical mesial section of bony pelvis, showing planes at which measurements are taken (1, 2, 3), and angles of plane of brim 60° and outlet 11°.

the brim extends from the upper border of the symphysis pubis to the sacral promontory, and measures 4 inches; at the second plane (cavity) it measures 4½ inches; and at the outlet, with the tip of the coccyx firmly pushed back, it is 5 inches in length.

The transverse diameter of the brim is measured at the widest distance between the iliac bones, and measures 5 inches. In the cavity it is 4½ inches, and at the outlet, from one ischial tuber to the other, its dimension is 4 inches. The two oblique diameters of the brim are taken from the sacro-iliac joint on one side to the ilio-pectineal eminence on the opposite side, and are called right and left respectively, according to the joint from which they are taken. They measure 4½ inches. In the cavity and at the outlet they are measured parallel to those at the brim, and are of the same length, viz., 4½ inches.

In considering the diameters of the pelvis as a whole (see table), it will be noted that the conjugate from above downwards is increased by an inch, the transverse is decreased by an inch, while the obliques remain the same throughout.

TABLE

| | Conjugate. | Oblique. | Transverse. |
|---|---|---|---|
| Brim . . | 4 | 4½ | 5 |
| Cavity . . | 4½ | 4½ | 4½ |
| Outlet . . | 5 | 4½ | 4 |

Another internal diameter which is of much practical value remains to be described, viz., the diagonal conjugate. It is measured from the lower border of the symphysis pubis to the sacral promontory, and is 4¾ inches. It is of importance as being a measurement which can be readily taken by the examining finger, and from which by the subtraction of three-quarters of an inch the length of the conjugate of the brim (conjugata vera) can be estimated.

In a general description of the normal pelvis the brim is considered as heart-shaped. The cavity, as the diameters show, is circular, while the outlet is diamond-shaped.

In the erect posture the plane of the pelvic brim forms an angle of 60° with the horizon, the sacral promontory is about 3½ inches higher than the upper border of the symphysis pubis. Without the soft parts the plane of the outlet forms an angle of 11° with the horizon; the tip of the coccyx being about half an inch higher than the lower border of the symphysis pubis. With the soft parts *in situ* the plane of the outlet is very materially changed.

In the measurement of the pelvis there are three external diameters which are of practical importance:—The external conjugate from the spine of the last lumbar vertebra to the upper border of the symphysis; it is 7½ inches, and is of value in estimating the true conjugate. The interspinous diameter from one anterior superior iliac spine to the other (9½ inches), and the intercristal, between the widest portion of the iliac crests (10½ inches), are of more value as regards their comparison with each other than in the estimation of their actual length. Under normal conditions the interspinous should be at least an inch less than the intercristal; any approximation between them is indicative of flattening of the iliac bones, a condition usually met with in rachitic pelvic deformity (see p. 215).

The soft structures within the pelvis modify to a greater or less extent its various diameters; this is most evident at the outlet, which is filled by the pelvic floor. The pelvic floor may be described as a thick, compact, musculo-membranous diaphragm traversed by three slit-like canals—the vagina, rectum, and urethra. The former, which mainly is concerned in parturition, traverses the floor in the erect female at an angle of 60° to the horizon, or, in other words, parallel with the plane of the pelvic brim. On its external or skin aspect the pelvic floor bulges in a convex manner beyond the plane of the bony outlet to the extent of nearly 3 cm.; this is described as the pelvic floor "projection" (Fig. 8).

For descriptive purposes the floor may be considered as composed of two segments, divided from one another by the transverse vaginal slit, and known respectively as the anterior or pubic segment and the posterior or sacral segment. The former consists of the anterior vaginal wall,

bladder, urethra, and retropubic fat, is loosely attached to the bony canal, and is freely movable. The latter consists of the posterior vaginal wall and structures posterior to it, is firmly attached and less mobile—features of the greatest value in considering the method by which this seemingly impenetrable barrier to the passage of the child is overcome during labour.

Viewed as a whole the parturient passage may be considered as a bony canal merely lined by soft structures in its upper half, but from the thickness and consistency of these soft structures in its lower half materially modified by them as regards direction. As has already been stated, the plane of the pelvic brim and direction of the vagina respectively form an angle of 60° with the horizon, and are thus parallel; the axis of the brim and direction of the vaginal canal which forms the exit through the outlet must, therefore, be at right angles. Before expulsion of the uterine contents can be accomplished, therefore, a curved path must be traversed equivalent to half a circle. This curvature of the pelvic canal is described as the "curve of Carus," or axis of the pelvic canal. For clinical purposes the axis of the inlet may be roughly considered as the direction of a line drawn from the umbilicus to the tip of the coccyx.

Third Factor.—The Passengers.—These are represented by the fœtus, placenta, and membranes, and liquor amnii.

*The Passenger or Ovum.*— For descriptive purposes the ovum may be divided during labour into three parts: (1) The free or presenting part, which can be felt by the examining finger. (2) The obstructed part, which is in contact with the girdle of resistance, that is to say, the portion which is in contact with the genital canal; in the first stage the girdle of contact is formed by the cervix, and in the second stage by the vagina which lines the bony pelvis. (3) The part which is directly acted upon by the powers, and lies above the girdle of resistance. As a whole the ovum when entire is of an ovoid shape, and under normal circumstances the lower end of the ovoid is the smaller; by this means it is accommodated to the normal ovoid contour of the uterine cavity. After rupture of the membranes the fœtus alone is to be considered; this also, from the marked flexion of its parts upon one another, is of an ovoid shape, the smaller end of the ovoid being the cephalic extremity. This accounts for the frequency of cephalic presentations, 96 per cent; the fœtal ovoid conforming with the shape of the uterine cavity (Fig. 2). Though forming the smaller end of the fœtal ovoid, the fœtal head in itself forms the largest and least compressible portion of the uterine contents, and, as has already been stated, is the portion of the fœtus which offers the greatest difficulty to expulsion. This is not merely from the size of its diameters, but also from the fact that it is less compressible. Its incompressibility, however, is not absolute; the bones are incompletely ossified, and offer between them membranous interspaces, so-called sutures, which allow of a considerable amount of overriding or moulding as it is called.

The bones of the cranial vault — occipital, frontal, and parietal—are separated from one another by one longitudinal and two transverse sutures, respectively named sagittal, lambdoidal, and coronal. Where the sutures cross each other, membranous interspaces of considerable dimensions are present, and are called fontanelles; thus where the sagittal and lambdoidal sutures meet is the posterior fontanelle, and where the sagittal intersects the coronal the anterior fontanelle or bregma (Fig. 10).

The posterior fontanelle is triangular in shape, and has three sutures running from it. The

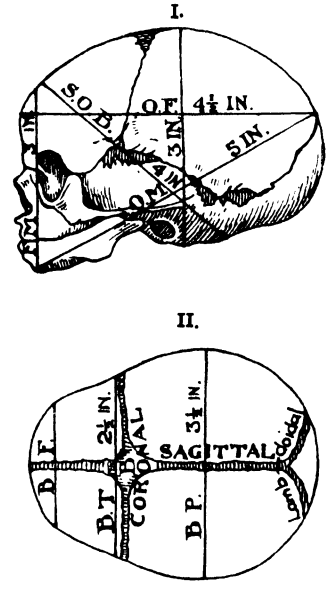



Fig. 10.—Diagrams of fœtal head, showing sutures and diameters. B, Anterior fontanelles: I. longitudinal and vertical sutures; II. transverse sutures.

anterior fontanelle is larger than the posterior, lozenge-shaped, and has four sutures entering into its formation. The space between the anterior and posterior fontanelles, and bounded laterally by the parietal eminences, is known as the vertex. The regions of the head are the occiput, vertex, brow or sinciput, and face.

*Diameters.*—For practical purposes a series of measurements of the fœtal head, known as diameters, are taken; these may be tabulated as longitudinal, transverse, and vertical.

A. *Longitudinal.*—Occipito-mental, from the chin to the occipital protuberance, 5 inches. Occipito-frontal, from the glabella or root of the nose to the occipital protuberance, 4½ inches. Suboccipito-bregmatic, from the anterior angle of the anterior fontanelle to the junction of the

occiput with the neck, 4 inches. This diameter may be shortened almost half an inch by taking the measurement from the posterior angle of the bregma, a point of importance in the movement of flexion in the mechanism of labour, which see (p. 174).

B. *Transverse.*—Biparietal, joining the parietal eminences, 3½ inches. Bitemporal, between the widest points of the coronal suture, 3 inches. Bifrontal, the widest part of the head anteriorly, 2½ inches.

C. *Vertical.*—Fronto-mental, from the chin to the upper part of forehead, 3 inches. Trachelo-bregmatic, from the foramen magnum to the bregma, 3 inches.

The circumference of the head in the occipito-mental plane is 16 inches, in the occipito-frontal plane 14 inches, and in the suboccipito-bregmatic plane from 11 to 12 inches.

As a whole the fœtal head is wedge-shaped; viewed from above, it slopes away forwards from the parietal eminences and back to the occiput.

During labour the diameters of the head are considerably diminished by the overriding of the bones, equitation. The occiput passes beneath the parietals, and the posterior parietal bone is driven beneath the anterior.

From the measurements of the fœtal head it will be seen how closely they correspond with the available capacity of the bony parturient canal, and how finely balanced, therefore, must be the mechanism to allow of expulsion through its lumen.

As has already been shown (see "Fœtus," vol. iii.), the fœtus lies in a membranous sac, surrounded by liquor amnii, and attached to the uterus by the placenta and umbilical cord, through which it derives its nourishment and oxygen, and excretes waste products.

As the fœtus forms by far the largest portion of the ovum, its expulsion is undoubtedly the main feature in the mechanism of labour. It has, therefore, to be closely studied: 1st, as regards the manner it is disposed in the uterine cavity at the onset of labour; and, 2nd, with reference to the measurements of the largest diameters which pass through the parturient canal during labour, the fœtal head.

"*Disposition*" *of Fœtus in Utero.*— By this is meant the general relation of the fœtus as a whole to the uterine cavity. This embraces (*a*) the relation of the fœtal parts to one another, "attitude" of the fœtus; (*b*) the relation of the long axis of the fœtus to the uterine cavity, with special reference to the most dependent part or "presentation" of the fœtus; and (*c*) the relation of the presenting part to the parturient canal, "position" of fœtus. The "attitude" of the fœtus is one of almost complete flexion. The head is flexed on the chest so that the chin is in contact with the sternum; the vertebral column is bent on its ventral aspect, and the thighs, knees, and elbows are all acutely flexed. By this means the fœtus forms an ovoid, and occupies the least possible space, and at the same time is a compact mass which is acted upon by the powers during expulsion to the greatest advantage. Such a marked degree of flexion is obtained that the length of the fœtal ovoid *in utero* is barely half of the actual length of the child when born. Thus a full-time child of 20 inches measures *in utero* scarcely 10 inches from pole to pole. The cephalic extremity or pole is the smaller (Fig. 2).

In its disposition *in utero* the long axis of the fœtal ovoid naturally corresponds to the long axis of the uterine cavity, which is usually vertical. Thus one or other pole of the fœtus usually presents. In over 96 per cent of cases the cephalic pole presents; this is due to the accommodation of the fœtal ovoid to the uterine ovoid, the smaller end of the ovoid uterine cavity being normally the lower (Fig. 2). In 3 per cent of full-time labours the podalic extremity or breech of the fœtus presents; in these instances the change of presentation is probably due to some change either in the fœtal ovoid or in the shape of the uterine cavity. In less than ·5 per cent the fœtus lies transversely in the uterus (shoulder presentations).

The relation of the presenting part to the parturient canal, so-called "position," varies considerably before the onset of labour. In vertex presentations the different positions are named according to the situation of the occiput. Under normal conditions the longest diameter of the vertex (occipito-frontal) lies in one or other oblique diameter of the brim; thus the occiput may be to the front or back, either on the left or right side. Four positions are thus described, viz., left occipito-anterior, right occipito-anterior, right occipito-posterior, and left occipito-posterior—the first, second, third, and fourth positions of Naegelé in the order named. The relative frequency of these positions is L.O.A. 65 per cent, R.O.A. 10 per cent, R.O.P. 20 per cent, L.O.P. 5 per cent. It will then be seen that in 85 per cent of vertex cases the occipito-frontal diameter lies in the right oblique diameter of the pelvis, and 65 per cent with the occiput forwards. This is accounted for by the fact that the right oblique is the most available diameter of the pelvic brim, the left oblique being encroached on by the full sigmoid flexure of the colon. That the occiput is so frequently forwards is to be explained by the accommodation of the fœtal ovoid to the uterine ovoid, the convex back of the fœtus becoming accommodated to the markedly concave anterior aspect of the uterine cavity. From its frequency, therefore, the normal presentation and position is the vertex L.O.A.

Further reference to the *position*, *presentation*, and *attitude* of the fœtus will be found on p. 169 *et seq.*

## Stages and Duration of Labour

DEFINITIONS . . . . . 163
PREMONITORY STAGE . . . . . 163
FIRST STAGE . . . . . 163
SECOND STAGE . . . . . 166
THIRD STAGE . . . . . 167

The process of labour is divided into three stages: the first stage, or *stage of dilatation*; the second stage, or *stage of expulsion*; and the third stage, or *placental stage*. Further, for clinical purposes it is convenient to include an additional stage—the *premonitory stage*, inasmuch as labour is ushered in by a train of symptoms and physical signs of sufficient definiteness to warrant such an addition.

THE PREMONITORY STAGE.—*Duration.*—The premonitory stage of labour is most irregular, both in the time of its onset and the degree of its symptoms. As a rule the symptoms first show themselves one or two days before labour —properly so-called—starts. In primiparæ the symptoms are well marked; in multiparæ they may be slight or even entirely absent.

*Phenomena.*—The principal phenomena associated with this stage are as follows:—

(1) The Occurrence of False Pains.—The commonest phenomena of commencing labour consists in the occurrence of irregular pains, distributed over the abdomen generally. These pains, which may be considered as amplifications of the painless contractions of the pregnant uterus, are known as "false pains" or *dolores presagientes*. They occur at widely separated intervals, and are distinguished from true labour pains by their irregularity, and by the fact that they are felt over the abdomen generally and not in the back.

(2) The Descent of the Fœtal Head into the Pelvic Cavity.—This sign is of value in multiparæ, as in their case the head does not as a rule descend until about the commencement of the first stage. In primiparæ, on the other hand, it is valueless, as in them the fœtal head can, as a rule, be found in the pelvic cavity during the last three weeks of pregnancy. It must also be borne in mind that descent of the head may be prevented by disproportion between its size and the size of the pelvic brim or cavity, or owing to some intra-uterine obstruction to the descent. The commonest causes of disproportion are contracted pelvis, mal-presentations of the head, hydramnios, and tumours growing from or occupying the pelvis. The commonest causes of intra-uterine obstruction are low situation of the placenta, hydramnios, twins, and myomata obstructing the cervical canal or lower uterine segment.

(3) Partial Dilatation of the Cervical Canal.—The changes which occur in the cervix during this stage differ in the case of primiparæ and of multiparæ. In primiparæ, the internal os usually commences to dilate at the beginning of labour, while the external os may remain closed for some time after labour has started. In multiparæ, on the other hand, the external os is as a rule dilated for some days before labour starts; and, in some cases, the internal os may share in this dilatation, though, as a rule, its dilatation commences during this period. In both primiparæ and multiparæ, the operculum or plug of mucus which fills the cervical canal is expelled

(4) Swelling of the Vulva.—A slight degree of swelling of the vulva very constantly occurs. It is due to the increased obstruction offered to the return of blood, owing to the pressure exerted upon the veins by the descending head.

(5) The Occurrence of a Blood-stained Discharge.—The discharge or show—as it is generally termed—which occurs at this period consists of viscid mucus from the cervix, and a small quantity of blood; it is probably closely connected in its quantity and onset with the commencement of dilatation of the cervix.

The falling of the fundus of the uterus is sometimes given as one of the phenomena of this stage. At the end of the thirty-sixth week the fundus reaches to the ensiform cartilage, while at the commencement of labour it is found to be midway between the ensiform cartilage and the umbilicus. As, however, this change gradually occurs during the last three or four weeks of pregnancy, it can hardly be considered as one of the symptoms of this stage.

*Diagnosis.*—It is by no means easy in all cases to determine whether the patient has reached the premonitory stage of labour or not. It is a question which frequently can only be settled by carefully looking for the various symptoms and physical signs which have been described. The fixity of the head is a tolerably reliable guide in multiparæ, if it is present; on the other hand, it is of no value in primiparæ. If the head is not fixed, and other signs point to the likelihood of the patient being in labour, an attempt must be made to ascertain if there is any cause sufficient to prevent such fixation. The occurrence of irregular pains is sometimes deceptive, as they may be due to flatulence, etc. A considerable degree of dilatation of the cervical canal is a tolerably certain sign. Slight dilatation, on the other hand, is but of a negative value.

FIRST STAGE.—*Duration.* The first stage, or stage of dilatation, commences with the onset of true uterine contractions, and ends with the full dilatation of the os and the rupture of the membranes. Its average duration is in primiparæ from eleven to twelve hours, in multiparæ from six to eight hours.

*Phenomena.*—The chief phenomena of the first stage are—the uterine contractions, the taking up and dilatation of the cervix, and the rupture of the membranes.

The contractions of the uterine muscle fibres, or the "labour pains" as they are generally termed, are involuntary, occur intermittently, cause a varying degree of pain, and sweep over the organ as a peristaltic wave. The effect of a contraction upon the shape of the uterus is to cause a diminution of the transverse diameters, and an increase in the longitudinal diameters and in the thickness of the walls; the effect upon the cavity of the uterus is to cause a diminution in the size of the latter. The result of this diminution is to cause increased pressure upon the ovum, and, as the latter is incompressible, to force it in the direction of least resistance. Various factors combine in making the region of the internal os the area of least resistance to the advance of the ovum, and consequently the lower pole of the ovum tends to advance in this direction. The duration of a contraction is from three to ten seconds, and the interval between two contractions may at the commencement of labour be an hour or more, while, as the second stage approaches, they may occur every ten to twenty minutes.

While the uterine contractions are at work intermittently diminishing the size of the uterine cavity, there is another change taking place in the fibres which results in the permanent diminution of the cavity. This is the occurrence of retraction, and, as it is a most important process, it is well to devote a few lines to a description of it. The uterus consists of two distinct regions or segments—the upper uterine segment and the lower uterine segment. The upper segment—whose main function is to expel the fœtus—contains the contractile fibres of the uterus; the lower segment—whose main function is to expand in order to allow the passage of the fœtus—contains but a very small proportion of contractile fibres, and so may be regarded as the non-contractile segment of the uterus. The junction between the two is known as the contraction ring, or sometimes as the ring of Bandl. The latter term, however, implies the acceptance of Bandl's theory as to its origin, and unless we are prepared to accept this, it is better to use a term which does not tie us to a fixed theory. At the commencement of labour the contraction ring is situated slightly above the internal os, and during the whole labour it is rising progressively higher on the uterus, so that—in an extreme case where some obstruction to delivery existed, and labour was consequently much prolonged—the contraction ring might be found at the region of the umbilicus. The gradual rising of the ring upwards is associated with an equally gradual thickening and shortening of the upper or contractile segment, and a similar thinning and lengthening of the lower segment. This change is the effect of retraction, and retraction itself may be described as a process by which the muscle fibres do not return to their full length after each contraction, but remain slightly shortened. There is probably also an actual change of position of the fibres, at least in their relationship to one another; so that those which at the commencement of labour were lying end to end, after some little time lie with their ends overlapping, and after a longer time may even lie side by side. The retraction of the fibres always occurs towards the fundus, or, in other words, the contraction ring always tends to move upwards towards the fundus. The ring can be felt through the abdominal wall, as a depression running obliquely across the uterus, in those cases in which labour has been very strong or unduly prolonged. In normal labours it can rarely be felt, as it does not rise sufficiently high above the symphysis pubis. It is most essential to be able to recognise the presence of the contraction ring, as it furnishes an absolute indication of the effect of the uterine contraction upon the uterine wall.

The taking up and the dilatation of the cervix are the essential phenomena of this stage, as is shown by the name usually given to it—the stage of dilatation. The taking up of the cervix is the term applied to the process by which the cervical canal is made continuous with, and so part of, the lower uterine segment. The extent to which this process occurs differs in primiparæ and in multiparæ, as will be seen by reference to the diagrams.

In primiparæ, at the commencement of labour the cervix is long, and presents more or less its original outline, having both the external and the internal os closed. The first step consists in the dilatation of the internal os, then of the supra-vaginal portion of the cervical canal, and then of the infra-vaginal portion. As soon as this last has occurred the taking up of the cervix is complete, and the uterine and cervical cavities are continuous. The os externum, which now forms the uterine orifice, is still undilated.

In multiparæ, on the other hand, at the commencement of labour the external os is as a rule sufficiently dilated to admit one or two fingers, and the cervical canal is somewhat everted as the result of former lacerations, etc. Consequently, when we examine vaginally the finger passes through the external os, and first is obstructed by the internal os. As soon as labour commences the internal os dilates, and also the supra-vaginal portion of the cervical canal. This as a rule completes the degree of taking up of the cervix which occurs, and the remainder of the cervix, *i.e.* the lower portion and the already somewhat dilated os externum, retracts synchronously when the time comes for the uterine orifice to dilate. The result of this difference between primiparæ and multiparæ, is that in the former when the taking up of the cervix is complete, the uterine orifice is encircled by extremely thin, paper-like edges, formed by

the borders of the original os externum alone. In multiparæ, on the other hand, the uterine orifice is surrounded by blunt, comparatively thick edges, formed by the portion of the

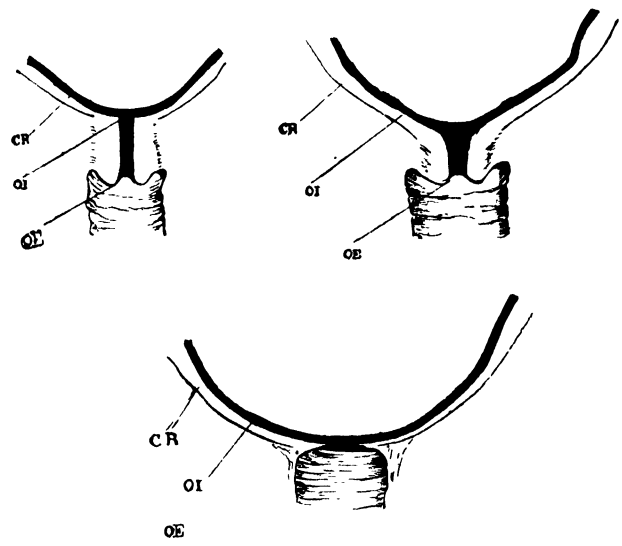



FIG. 11.—Diagrammatic representation of the manner in which the cervix is taken up in the case of a primipara. OE, os externum; OI, os internum; CR, contraction ring. (Schroeder.)

cervical wall which has not been taken up, as well as by the margin of the os externum.

As soon as the taking up of the cervix is complete, the next step is the dilatation of the uterine orifice. This is brought about by the downward pressure of the advancing ovum

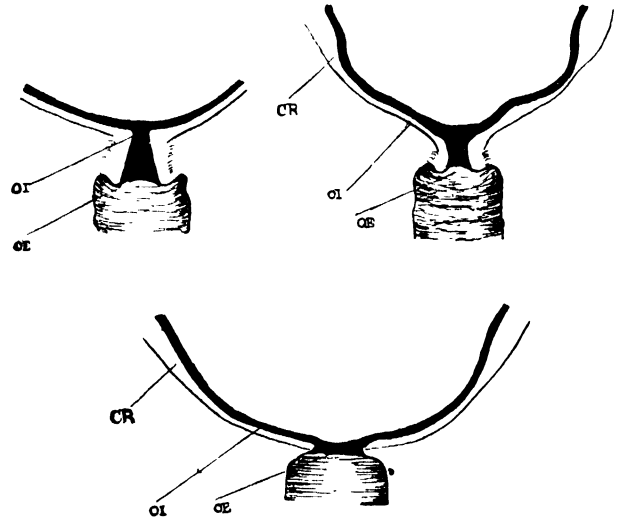



FIG. 12.—Diagrammatic representation of the manner in which the cervix is taken up in the case of a multipara. OE, os externum; OI, os internum; CR, contraction ring. (Schroeder.)

and by the gradual retraction upwards of the remainder of the cervix. As soon as this retraction is so complete that all traces of cervical projection have disappeared, and the vaginal and uterine cavities have become practically continuous, the cervix is said to be fully dilated.

The final phenomenon of the first stage is the rupture of the membranes. This event, which is due to the loss of support experienced by the membranes owing to the retraction of the cervical walls, usually synchronises with the full dilatation of the os. In certain cases, however, owing to a failure of adaptation between the presenting part and the lower uterine segment, the membranes at an early period in the first stage have to withstand the full force of the uterine contractions, transmitted to them through the liquor amnii, and consequently rupture almost at once. In such cases all the liquor amnii escapes with a rush, an occurrence which never happens under normal circumstances, when there is due adaptation between the presenting parts and the lower uterine segment. In these cases, only the liquor amnii in front of the head escapes, as the remainder is dammed up by the presenting part.

*Constitutional Symptoms.*—The constitutional symptoms of the first stage are very slight. At the commencement the patient in many cases pursues her ordinary occupations, save when a pain occurs. As the stage advances, the pains become more frequent and of longer duration. The pulse and temperature are as a rule unaffected, save for a slight increase in frequency in the rate of the former during a pain. Gastric disturbance associated with vomiting is of common occurrence, especially towards the end of the stage.

*Diagnosis.*—As a rule, it is easy to determine the onset of the first stage. All the symptoms which have been given under the premonitory stage are present, but are more marked. The painless contractions of the uterus disappear and are replaced by painful contractions. The latter can be recognised by laying the hand flat upon the abdomen of the patient, and determining the fact that the onset of a pain is associated with an easily perceptible hardening of the uterine muscle. The character of the pains serves to distinguish between the first and the second stage, even without going into the determination of the condition of the cervix and of the membranes. In the first stage the pains are constituted solely by involuntary contractions of the uterine muscle fibres; in the second stage, as will be seen, the patient accompanies each uterine contraction by voluntary contractions of the abdominal muscles—bearing down, as it is termed. (For Management, see p. 193.)

SECOND STAGE.—*Duration.*—The second stage, or stage of expulsion, commences with the full dilatation of the os and the rupture of the membranes, and ends with the expulsion of the child. Its average duration is from one to two hours in primiparæ, and from ten to fifteen minutes in multiparæ.

*Phenomena.*—The chief phenomena of the second stage are the continuance of involuntary contraction and retraction of the uterus, the addition of voluntary contraction of the abdominal muscles, and the consequent expulsion of the fœtus.

The nature of the uterine contractions remains unchanged, save that they become more violent, and last for a longer time. The interval between them is also lessened. They vary in length from thirty to sixty seconds, and occur every five to seven minutes up to the actual time of expulsion, when they are almost continuous. Retraction of the muscle fibres also continues; and its importance is now seen, as it enables the uterus to reduce the size of its cavity to suit its lessening contents. The voluntary contractions of the abdominal muscles impart to the second stage pains their expulsive character. As each contraction commences, the patient fixes her diaphragm by closing the glottis after a deep inspiration, and, contracting her abdominal muscles to the utmost, brings all the force she can to bear upon the uterus and its contents. The reason that these voluntary expulsive efforts do not occur during the first stage is obvious. At that time, the undilated cervical canal offers a bar to the advance of the uterine contents, and hence the effect of the contraction of the abdominal muscles is merely to drive the entire uterus downwards into the pelvis without in any way furthering the expulsion of the ovum. In the second stage, this obstruction is removed, and the compression of the uterus by the contractions of the abdominal muscles materially assists in hastening the delivery of the fœtus.

The expulsion of the fœtus commences as soon as the membranes rupture. The presenting part is driven downwards through the vagina until it reaches the perineum, where there is usually some little delay. Then as each fresh contraction occurs, the presenting part advances a little, and can be seen at the vulva separating the labia; and as the contraction passes off, it again recedes into the vagina. Finally, it descends so far that it does not recede, and then the next contraction will in all probability cause its expulsion. As the presenting part is passing over the perinæum, the pain caused is so severe that the patient is compelled to cry out. This act, by opening the glottis, checks all efforts at bearing down, and so slows expulsion. In this way a longer time is given to the perineum to dilate, and the tendency to laceration is diminished.

The necessary dilatation of the vagina, vulva, and perineum is permitted by the softening these tissues undergo as the result of serous infiltration of the connective tissue. This is due, first, to the active hyperæmia of the vessels which occurs during labour; and secondly, to the fact that the return flow of blood being obstructed by the pressure exerted upon the veins by the presenting part, there is a consequent increase of intra-vascular tension.

*Constitutional Symptoms.*—The constitutional symptoms of the second stage are more marked than are those of the first, owing to the fact that the uterine contractions are stronger, and that the descent of the fœtus through the vagina increases the patient's suffering. The frequency of the pulse-rate and of respiration is increased during the pains, and profuse sweating may occur. As the fœtus presses more and more upon the rectum, the patient experiences a strong desire to go to stool, although there is usually nothing in the bowel to evacuate.

*Diagnosis.*—The diagnosis of the onset of the second stage can, as has been mentioned, be made by the change in the character of the pains. Further, the patient herself, or her attendants, can usually inform us whether the membranes have ruptured or not, so obviating the necessity of making a vaginal examination. If the latter is made, the fact that the cervical canal is fully dilated can be determined.

The progress which the fœtus is making through the vagina can be determined by abdominal palpation or by vaginal examination. By the assistance of the former we can follow the progressive descent of the presenting part by noting the rate at which it travels downwards behind the symphysis. In the early part of the second stage, the height above the symphysis of some portion of the presenting part—for instance, the chin in vertex presentations—can be measured in finger-breadths. As labour advances, the portion which we have taken for our guide will be found to approach the level of the symphysis, and then to sink below the latter. The rate of advance can then be followed by sinking the finger-tips into the true pelvis; while by the time we can no longer reach the chin even in this manner, the presenting part will be pressing upon the perineum, and almost or quite visible from below. If a vaginal examination is made in order to determine the progress of the presenting part, a gradual diminution in the distance between the latter and the perineum can be determined. But here we have to guard against a possible fallacy. In all cases of delayed labour with strong uterine contractions the caput succedaneum hourly increases in size, and bulges more and more downwards towards the perineum. Consequently, it is easy to attribute the diminished distance between the caput and the perineum to the descent of the presenting part

instead of—as may be the case—to the increasing size of the caput. (For Management, see p. 194.)

THIRD STAGE.—*Duration.*—The third stage commences with the birth of the fœtus, and ends with the expulsion of the after-birth. It is impossible to estimate its average duration, as the latter depends entirely upon the manner in which the stage is conducted. If the expulsion of the placenta is left to the natural efforts, the average duration is from two to three hours. If, however, the usual method is adopted of waiting until the placenta is detached by the uterine contractions and expelled into the vagina, and then expressing it after the Dublin method, the average duration of the stage is from ten to fifteen minutes.

*Phenomena.*—The principal phenomena of the third stage are the continuance of intermittent contractions and permanent retraction of the uterine muscle fibre, the detachment of the placenta, and the expulsion of the latter, first from the contractile segment of the uterus into the lower uterine segment or the vagina, and then from the latter position externally. It is most convenient to consider the third stage as consisting of two periods. In the first period, the placenta is detached and expelled below the contraction ring; in the second period, it is driven outside the genital passages. The mechanism by which the placenta is detached from the uterus is still a matter of some dispute. The most commonly accepted theory is that of Schultze. He considered that the placenta was first partially detached owing to the shrinkage of the placental site, which occurs as the uterus contracts down after the birth of the fœtus; that then blood escaped from the uterine vessels into the retro-placental space thus formed, and constituted a hæmatoma, the pressure of which completed the detachment of the placenta and drove the latter downwards into the membranes with its fœtal surface lying lowest. As a result, the placenta is the first part of the secundines to leave the uterus, and, subsequently, as it descends still farther, it pulls the membranes after it and so causes their detachment. Matthews Duncan, on the other hand, considered that the placenta after its detachment was expelled from the uterus with its lower border first, and that it passed through the contraction ring as a button goes through a button-hole. Its expulsion with the smooth fœtal surface forwards, he considered to be due to premature traction upon the cord. Schultze's mechanism usually occurs in about three-quarters of all cases, but then there is frequently a slight amount of traction upon the cord during the birth of the child.

The Edinburgh school, in the persons of Hart and Barbour, brings forward two theories as to the cause of placental separation and expulsion, which differ from the foregoing. Barbour considers that he has proved that the placental site can be reduced to a space of $4\frac{1}{2}$ in. × 4 in., without causing the separation of the placenta; he also considers that if the uterus contracts firmly down upon the placenta it will tend to expel the latter, and during this process separation will naturally occur. Accordingly, he attributes the separation of the placenta to the diminution of the placental site to an area less than $4\frac{1}{2}$ in. × 4 in., plus the action of the uterus as a whole on the placental mass. Hart, on the other hand, while agreeing that the main cause of the separation of the placenta is disproportion between its area and the area of the placental site, considers that the cause of the disproportion is, not the placental site becoming smaller than the placental area, but its becoming larger than the latter. His reason for his belief is as follows: so long as the placenta has either or both its blood supplies from the maternal or fœtal vessels intact, it can diminish or increase in size *pari passu* with the portion of uterine wall to which it is attached. When, however, the supply from both mother and fœtus is cut off, the placenta can diminish *pari passu* with the uterine wall, but cannot again expand as the wall relaxes. Consequently, separation occurs during the relaxations of the uterus which occur in the third stage after the fœtal circulation has ceased—owing to the ligation of the cord or other cause, and after the maternal supply has been cut off by the retraction of the uterus.

The descent of the placenta below the contraction ring, *i.e.* the commencement of the second period of the third stage, can be recognised by certain changes which take place (Figs. 13 and 14). They are as follows:—

(1) The Funis lengthens.—As the placenta

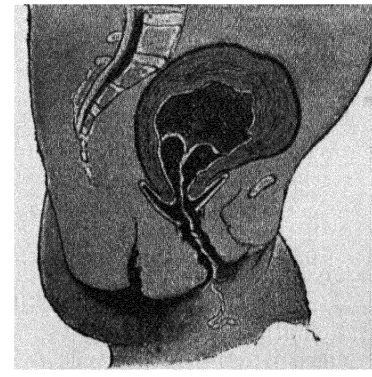


FIG. 13.—Before the expulsion of the placenta (diagrammatic).

leaves the uterus and comes to lie in the vagina, the cord will also descend, and there will be an increase in the length of the portion which is outside the vulva. This increase in length will be most easily recognised if, when tying the cord, the ligature which is placed next the mother is tied as close to the vulva as possible.

It thus forms an indicator on the cord, and enables any elongation of the latter to be readily detected.

(2) The Fundus of the Uterus rises upwards almost to the Umbilicus.—At the birth of the child the portion of the uterus above the contraction ring sinks downwards into the thinned-out lower uterine segment and vagina, under the pressure of the abdominal muscles and of the controlling hand of the assistant. Later, as the placenta is expelled from the uterus, it

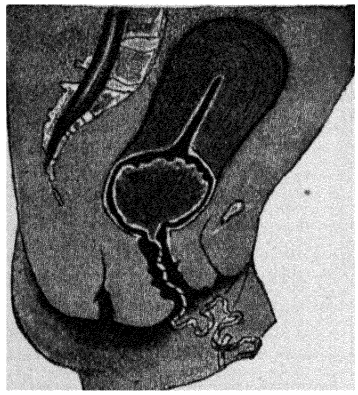


FIG. 14.—After the expulsion of the placenta (diagrammatic).

comes to occupy the place where the body of the uterus formerly lay, and so dislodges the latter upwards out of the pelvis. As a result the fundus rises from its former position—slightly above the pelvic brim—to almost the level of the umbilicus.

(3) The Mobility of the Uterus is increased.—This change also depends upon the alteration in the position of the body of the uterus. When the latter lay in the pelvic cavity with the placenta inside it, it was supported all round by the walls of the pelvis, and consequently it could not be readily moved from side to side. As, however, it rises out of the pelvis this support is lost, and consequently it becomes more mobile.

(4) The Abdominal Wall bulges forward above the Pubis.—This change is due to the presence of the placenta in the lower uterine segment or in the upper part of the vagina. The placenta, lying in one of these positions, pushes forward the structures in front of it, and so causes a prominence above the pubis which is not unlike that caused by distended bladder.

The expulsion of the placenta from the vagina, if left to the natural efforts, is a somewhat lengthy process. There is no very efficient natural mechanism for obtaining this expulsion, as the unnatural position in which the patient is placed, *i.e.* on her back in bed, prevents her from forcing the placenta out, by straining, as readily as she would do if she could get into a squatting position. Consequently the placenta lies in the vagina for some time, until it finally works its way downwards helped by any contractions of the abdominal muscles which may occur. In consequence of the unnecessary delay which such a tedious process would cause, this period of the third stage is invariably artificially shortened. The most usually adopted way of doing this is by the method originated in Dublin during the early years of the last (the nineteenth) century, *i.e.* by substituting firm pressure over the uterus for the natural efforts, and so by driving the uterus downwards into the vagina effecting the expulsion of the placenta.

As has been mentioned, the loss of a certain amount of blood is almost an invariable accompaniment of the third stage. The average amount is said to be four ounces before the placenta is delivered, and six ounces with the placenta and membranes (Dakin).

*Constitutional Symptoms.*—Immediately after delivery, the patient experiences a marked sense of relief due to the almost complete cessation of pain. The temperature may be slightly higher than during labour, while the pulse-rate may be somewhat less than it was during the latter portion of the second stage. The subsequent condition of the patient depends entirely on the amount of blood which is lost. In some cases there may be a slight increase in the pulse-rate and a depression of temperature of one or two degrees, owing to the amount of blood lost, and to the chilling of the patient, which may occur during the delivery of the after-birth and the necessary cleansing of the parts. The degree of pain caused by the uterine contractions is, as a rule, not very severe. (For Management, see p. 196.)

## Diagnosis and Mechanism of Labour


GENERAL DIAGNOSIS OF NORMAL LABOUR 169
PHYSICAL EXAMINATION . . . . . 169
*Abdominal* . . . . . 169
*Vaginal* . . . . . 172
*Bimanual* . . . . . 172
GENERAL MECHANISM OF NORMAL LABOUR 172
*Factors of* . . . . . 172
VERTEX PRESENTATION—
*General Principles of Mechanism* . 173
*Flexion* . . . . . 174
*Internal Rotation* . . . . . 175
*Extension* . . . . . 176
*Head Moulding* . . . . . 177
DIAGNOSIS AND MECHANISM IN SPECIAL VERTEX POSITIONS . . . . . 177
*Moulding* . . . . . 178
PERSISTENT OCCIPITO-POSTERIOR MECHANISMS . . . . . 178
*Moulding* . . . . . 180
DIAGNOSIS AND MECHANISM IN FACE PRESENTATIONS . . . . . 180
*Moulding* . . . . . 181

PERSISTENT MENTO-POSTERIOR MECHANISMS . . . . . 181
*First Face Position* . . . . 181
*Second Face Position* . . . . 182
*Third Face Position* . . . . 182
*Fourth Face Position* . . . . 182
BROW PRESENTATIONS . . . . . 183
DIAGNOSIS AND MECHANISM IN PODALIC LIES . . . . . 185
DIAGNOSIS AND MECHANISM IN TRANSVERSE LIES . . . . . 187
*Spontaneous Delivery* . . . . 188

## DIAGNOSIS OF NORMAL LABOUR

In describing the various relations which the fœtus and its parts may assume to the pelvis of the mother and to one another, the following terms will be used, viz., *lie*, *position*, *presentation*, and *attitude*.

By the *lie* is meant the relation of the long axis of the child to that of the mother. The fœtus may lie with its long axis approximately in that of the mother, in which case the lie is a longitudinal one; or the child may lie across the mother's trunk, and is then said to be a transverse lie.

In the case of a longitudinal lie the head may be directed downwards, cephalic lie; or the breech may be downwards, podalic lie.

The term *position* means the relation a given part of the fœtus—the one taken is usually on the posterior aspect—has to the anterior, lateral, or posterior aspect of the mother.

In the case of the head presenting by the vertex, the posterior end (occiput) may be directed to the front, occipito-anterior; to the side, head transverse; or to the back of the mother, occipito-posterior; or to some other point on the circumference of the pelvic ring.

The word "orientation" is frequently used by French authors to express this meaning, and is, in fact, a more exact and unambiguous expression of it.

*Presentation* is a term which has been used in a very loose way by various authors. It really means that part of the fœtus which is first touched by the finger of the person making a vaginal examination.

If the child is in a cephalic lie it may present by the vertex, or by the face, or by a surface of the head intermediate between these areas. Also, in a podalic lie the feet or the breech may be the presenting part. Tyler Smith defines the presentation as that part of the child which is "felt most prominently within the circle of the os uteri, the vagina, and the ostium vaginæ, in the successive stages of labour."

The relations which the trunk, the head, and the limbs of the child have to one another constitute the *attitude* of the fœtus. This is considered quite independently of any relation of the fœtus to the maternal parts.

The usual attitude is one of flexion—the head is flexed on the trunk, the thighs are flexed on the abdomen, and the legs on the thighs.

Or the head may be in a state of extension, as in face presentations; or the legs may be extended on the thighs, as in certain kinds of breech presentation.

## PHYSICAL EXAMINATION

In endeavouring to determine the relations of the child to its mother's pelvis in order that the course of labour may be intelligently watched, and any assistance, in cases where it is necessary, given to the greatest advantage, the most satisfactory results will be obtained by pursuing a routine course of examination in every instance.

The following plan should be adopted, and the sections taken in the order given:—

Abdominal examination—
Inspection.
Palpation.
Auscultation.
Vaginal examination.
Bimanual examination.

Other points also, such as the shape of the bag of membranes, the escape of meconium from the cervix, and any peculiarities in the way the liquor amnii comes away after rupture of the membranes, are to be observed, since they may assist in the diagnosis.

ABDOMINAL EXAMINATION.—For this purpose the woman must lie on her back in as comfort-

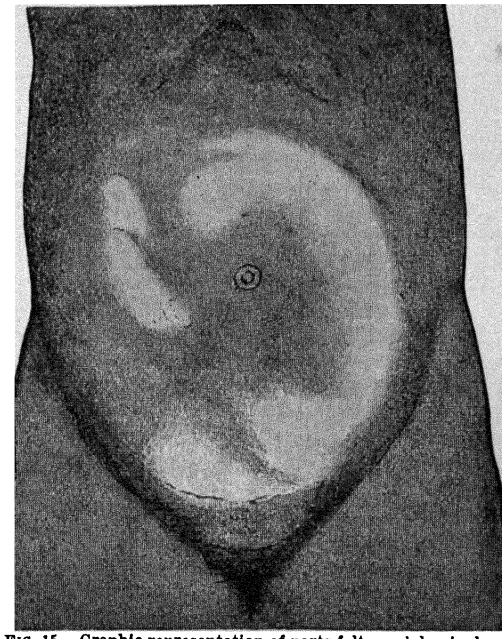


FIG. 15.—Graphic representation of parts felt on abdominal palpation in the case of cephalic lie (first vertex position).

able a posture as possible, with the abdomen thoroughly exposed to view.

The bladder must be empty, a catheter having been used if necessary, and the bowels should have been well cleared out.

*Inspection and Palpation.*—The uterus at the beginning of the examination may be found in a state of contraction or of relaxation. In either case valuable evidence can be gained.

*During Contraction.* — If it is tense, the general outline of the uterus can be readily seen and felt, and its long axis made out; but the parts of the child cannot be recognised.

The lie of the child, longitudinal or transverse, however, can be ascertained; and at the same time, although it may not be a question of importance as regards the future mechanism, the presence of a fibroid tumour in the accessible parts of the uterus would probably be discovered.

Deviations of the uterus from the normal axis, which, as will be seen later, influence the mechanism of labour, are easily made out, and steps may be taken now or later to diminish or change the side of any such deviation if it is likely to interfere with the normal course of labour.

*During Relaxation.*—It is when the uterus is relaxed, however, that the most valuable information can be gained. The exact relation of the child to the mother's pelvis, and its attitude, can in a large majority of cases be distinctly made out.

*Position of Observer.*—It is best to stand at first on the right hand of the patient, looking towards her head. The hands should be laid flat on her abdomen, one lying over each side of the uterus. Each hand thus makes counter-pressure against which the other can work (Fig. 16).

Supposing the child to be in a longitudinal lie, and that the hands fall about the middle of the uterine length, the first thing noticed in a case favourable for examination is that on one side, the left (of the woman) in the most common position of the child, there is a firm, even surface; on the other side the feeling is that of a somewhat soft elasticity. The resistant feeling is caused by the underlying back; the softer one is produced by a space filled with liquor amnii, existing between the two incurved poles of the fœtus. If the two hands are now moved up higher on the abdomen, still lying opposite to one another (Fig. 17), the back of the child can be traced up to the fundus uteri, feeling pretty much alike in all its length, and following the curve of the fundus on merging into the breech; while on the right side of the mother the left hand is able to make out some irregular knobs, which are the feet, and perhaps the knees of the child. The limbs may often be felt and seen to move, both by the observer and by the mother. It will be noticed that the mergence of back into breech, as the right hand is moved upwards, is an imperceptible one, differing considerably from the sensation conveyed where the head is at the fundus.

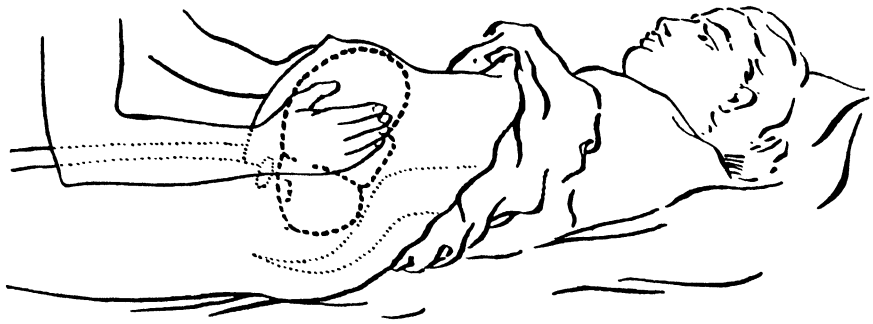


FIG. 16.

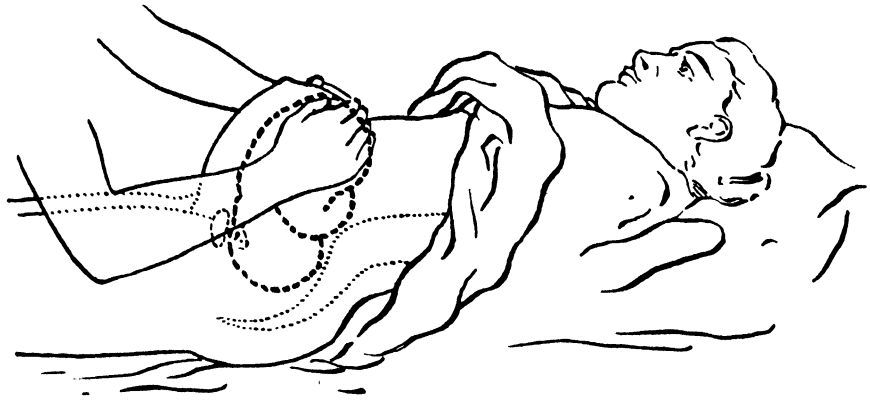


FIG. 17.

An attempt may now be made to feel the head of the child as it lies on the brim (multipara), or slightly dipping into it (primigravida), by placing the right hand on the abdomen, just above the level of the symphysis (Fig. 18). The thumb and middle finger will usually be able to grasp the base of the skull, and the

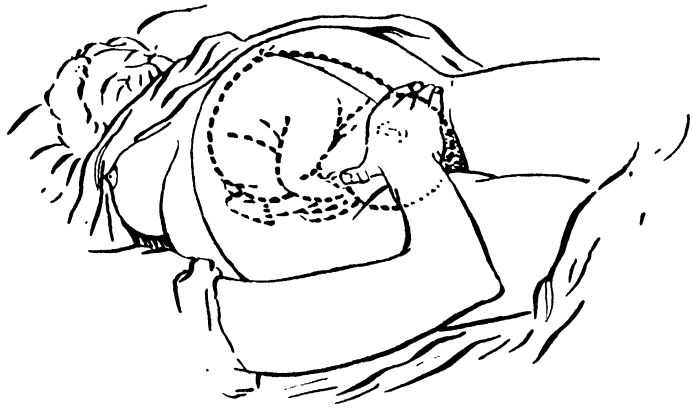


FIG. 18.

hardness, roundness, and mobility of the head can be recognised.

The head can be more clearly identified and its position made out by the observer's next turning so as to look towards the woman's feet, using his hands in combination as before. To grasp the head between the tips of the fingers they will have to be pressed somewhat deeply downwards and backwards towards the pelvic

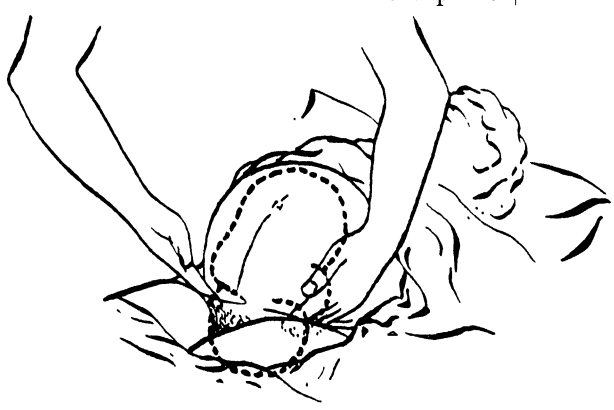


FIG. 19.

inlet—a superficial palpation will yield no results of value. When the head is felt to be well grasped by the tips of the fingers it can, in the usual attitude of the child, be made out without much difficulty that the back, traced from above, slopes into the nape of the neck without any abrupt curve; whereas on the right side of the woman the prominent forehead makes a fairly distinct relief from those parts of the fœtus, namely, the arms folded on the chest, to be felt just above it.

If the head is extended, as in face presentations, the occiput will be felt to project somewhat abruptly from the curve of the back as this is traced down; but the chin of the child will lie pretty much on a plane with the front of the child's body-surface. The arms are frequently not clearly recognised; if they are, there is no mistake as to which way the child is facing; but it may be remembered that the lower limbs, which can, if any part can, always be made out, point to the anterior surface of the child.

In performing any of the above manipulations the best results are got in a multipara whoseabdominal walls are thin, and who is able to bear such an examination without tightening her abdominal muscles. In cases where these conditions are not found the fœtal parts can often be recognised if, instead of making steady pressure on the parts under the hands, this pressure is made in slight jerks, in the same way as a liver may be mapped out in an ascitic abdomen. The slight jerk overcomes momentarily the resistance of the interposed layers, whether they be of elastic tissues or of fluid, though, of course, more perfectly in the latter case. It will be found, however, that with practice the consecutive sensations obtained by "dipping" all over the surface of a uterus, even if there be some obstacle of the kind named in the way, are mentally combined into a fairly reliable impression of the lie and position of the child.

The importance of abdominal examination of pregnant and parturient women cannot be over-estimated. In fairly practised hands they give more reliable results than are to be obtained from vaginal examinations alone, though it is not recommended that abdominal examinations should be considered sufficient for practical work. With the desire of avoiding all possibility of septic infection, a long series of cases were examined at one lying-in hospital by the abdominal method alone, and no accident is recorded; but it is evident that such a condition as prolapse of the cord, which gives no sign externally save that of slowing of the fœtal heart if compression of the cord is becoming fatal, would remain unrecognised if this means of examination only were used.

The results of abdominal examination in each kind of lie, position, and presentation will be given in describing the special mechanism belonging to each.

*Auscultation.*—This method of examination is applicable in practice to the abdomen alone;

for though attempts have been made to utilise it by the vagina, and a stethoscope has been contrived for the purpose, no information of value can, for many reasons, be obtained by this route.

For the purpose of diagnosis of the relations of the child to the pelvis, the only sound which is of any value is that of the fœtal heart.

In the commonest position of the child in the cephalic lie, namely, when it lies with its head flexed and the occiput pointing to the mother's left and somewhat forwards, the fœtal heart is best heard over a point about the middle of a line joining the left anterior spine to the navel; that is, at the spot where the right upper part of the child's back lies in contact with the uterine wall immediately under the abdominal parietes.

A layer of fluid between the child and the surface cuts off all possibility of hearing the sound; and this may be heard better over parts of the child, even if they are more remote from its thorax, so long as such parts are touching the uterine wall in front, than over the cardiac area of the child if this be separated by fluid from the end of the stethoscope. The heart-sounds of the fœtus are, therefore, never heard over the front of its chest unless it is in an attitude of extreme extension, such as is found in presentations of the face.

The sounds are more distinctly heard when counter-pressure is made on the opposite side of the uterus to the stethoscope, so as to bring the conducting surface into closer contact with the abdominal wall.

Vaginal Examination.—By vaginal examination the condition of the pelvis and its contents are more or less distinctly made out, according to the stage of labour. It is really complementary to the abdominal examination, and should not be undertaken until as much as possible has been already determined by the latter means.

As far as the mechanism of labour is concerned, and we are dealing here with this alone, the points to be observed are the relations of the parts within the canal of the cervix—or vagina, as the case may be—to the pelvic walls and to one another.

Thus it is ascertained how far the bag of membranes, if this exists, protrudes in front of the presenting part; whether the cord is presenting or prolapsed; what the presenting area is; and its relations, in position and size, to the pelvic walls.

After a careful abdominal examination, there is no difficulty in very rapidly making one's self quite certain on all these points. If no presenting part can be discovered it will easily be decided to what cause this must be referred, for a transverse lie will have already been made out by the abdomen, as will a high-lying breech or a hydrocephalic head; and, in the absence of these conditions, placenta prævia will be thought of and recognised. (Another cause of absence of presenting part, namely, rupture of the uterus and escape of the fœtus into the abdominal cavity, need not be considered here.)

If necessary to complete diagnosis, the whole hand may be introduced into the vagina under an anæsthetic.

As a matter of course, antiseptic measures must be rigidly practised.

Combined Examination.—A most accurate determination of the mechanism to be expected can be made by the bimanual method. The part occupying the pelvis or its inlet can be held between the fingers of the opposing hands, and all or nearly all its surfaces explored and recognised. In the case of the head the amount of flexion, and the relative size of the head and brim, can be infallibly demonstrated and the previous diagnosis confirmed. In breech cases the presenting part can be brought within easier reach of the vaginal finger, and its disposition clearly ascertained; or if a shoulder is presenting, this can be identified.

The other points in diagnosis may be mentioned, namely, the escape of meconium, which, in any quantity and unmixed with liquor amnii, strongly suggests a breech presentation; and the discharge of an excessive amount of liquor amnii, which may be due to hydramnios and may also indicate a podalic lie, or may mean a transverse lie or a contracted pelvis. In this category comes also a prolapsed cord, as showing that the presenting part does not accurately fit the pelvic inlet.

## Mechanism of Normal Labour

The mechanism of labour, by which is meant the movements which the fœtus makes in its passage through the parturient canal, is a process almost entirely belonging to the second stage. Some attention, however, will have to be paid to the first stage, that of dilatation of the cervix, and even to periods anterior to this. Three factors combine to constitute the mechanism of labour. They are:—

A. The expelling force.

B. The passage through which expulsion is effected.

C. The body to be expelled.

The *expelling force* is provided by the contractions of the uterine muscle, the muscles of the vagina, and those of the abdominal walls, as has been already described.

This force must act, as far as it is an effective one, in the axis of that portion of the canal occupied by the part of the child actually engaged in it.[1]

[1] The head is said to be "engaged" in the pelvis when it has entered sufficiently for its movements to be influenced by the pelvic walls; and any other part of the child is "engaged" in that region of the pelvis which is influencing the movements of the said part by its shape.

The *passage* consists of a short tube with a bent axis (axis of the parturient canal). The walls of this tube vary in rigidity at different cross-sections; and the shape of its cross-section, taken at right angles to its axis at that level, varies at different points along its length in a definite manner. (See Physiology of Labour, p. 154.)

The *body to be expelled* consists of two ovoids, the trunk and the head, connected by a joint which allows of almost "universal" movement. Of these two ovoids the head is comparatively rigid, the body very plastic. The head is therefore the more important of the two ovoids in the matter of mechanism.

There is, however, another property of the fœtus, and that is its elasticity. When the head is fully flexed, for instance, the child has a certain tendency towards extension; and, on the other hand, when the fœtus is extended there is a still greater tension produced in its body which makes for flexion. This curve-tension, as it may be called, has not received any attention from writers on obstetrics; but it has, as will be shown, an important influence in the mechanism of labour. The elasticity of the child is mainly due to its muscular cone, and in a less degree to the ordinary elasticity of the bones, ligaments, fasciæ, and other connective tissues.

The fit of the child to the pelvis is a close one, even when the two ovoids of which it consists are accommodated to the passage in the most advantageous way.

The most advantageous way is that, in the first place, the long axis of the child shall lie approximately in the axis of the passage—delivery in a transverse lie is impossible.

This being obtained, there remain the sections of the fœtus at right angles to the long axis to be adapted in the best way to the cross-sections of the passage—that is, the width of the shoulders has to go into that diameter of the canal where there is most room for it; and still more imperatively, the longest of those diameters of the head which lie across the canal must find themselves in the widest diameters of that part of the canal in which they lie, or labour will be delayed or arrested.

Since the tube has its greatest diameters at one level transverse, at another oblique, and at another antero-posterior, these longest diameters of the head and trunk will, as the child descends, be constantly endeavouring to follow them.

This endeavour on the part of the fœtal mass to find the *path of least resistance* is the cause of the mechanism of labour.

### Presentations of the Vertex

*General Principles of Mechanism.*—The movements of the child in the commonest kind of presentation, namely, that of the vertex, and in the position in which the head lies with the occiput directed forwards and to the left, will now be described in somewhat full detail. They will serve as a standard, and the points in which other mechanisms agree with or differ from this type can then be easily understood.

The child is found in the cephalic lie, in about 96 per cent of all cases, at the end of pregnancy. Of cases in the cephalic lie, about 75 per cent are presentations of the vertex in the position just mentioned. There are three main reasons for this: they are (1) the position of the centre of gravity of the fœtus at term; (2) the relative shapes of the fœtus and the uterus; (3) the movements of the fœtus in the uterus.

(1) *The centre of gravity of the child* at term is found to lie about the level of the shoulders, rather to the right side on account of the liver lying to the right, and nearer to the back than to the front of the thorax. A fœtus suspended in a fluid of its own mean specific gravity would thus tend to lie slightly on its right side with its head downwards.

The uterus is inclined to about an angle of 60° with the horizon when the woman is in an upright posture, and, in addition, its anterior surface is rotated slightly round to the right. Thus the left side of the front of the lower segment of the uterus is the lowest part of its cavity; in consequence the head tends to fall into this part, with the right shoulder in front of it—that is, into the position above mentioned. The natural attitude of the head is one of partial flexion, and so the vertex comes to lie lowest.

(2) *Relative Shapes of the Fœtus and the Uterus.*—The widest part of the uterus is the fundus, and the widest part of the fœtus is its breech, and so the breech tends to lie in the fundus. As a proof of the value of this as a cause it may be mentioned that in the case of hydrocephalic children, in which the head-end is the larger, the child lies with its breech downwards far more commonly than where it is normally shaped.

Also, it will be remembered that when the uterus is relaxed there is a well-marked convexity of the posterior uterine wall forwards, owing to the projection forwards of the lumbar spine. The normally flexed child has its concavity on its ventral surface, and, in consequence, obtains the most comfortable fit to the uterus by lying with its dorsal surface forwards.

(3) The movements of the fœtus in the uterus are mainly of its legs; and in any case the legs, acting at the end of the body, will have more influence than the arms in bringing about changes of lie. The child up to the seventh or even the eighth month is able to change its lie without much difficulty, as is well known.

If we take a child lying with its feet downwards, any sudden extension of its lower limbs will bring them against the brim of the pelvis

and tend to throw the lower end of the body upwards. If this displacement is so great as to bring the child into a transverse lie, the shape of the uterus will soon tend to convert this into a longitudinal one, either cephalic or podalic. If the child falls back into its original lie, the same process may be repeated again and again; but if the longitudinal lie happens to be the cephalic, the child with its legs uppermost has nothing resistant to kick against, and movements of its legs will have little effect in moving its lower end away from the fundus.

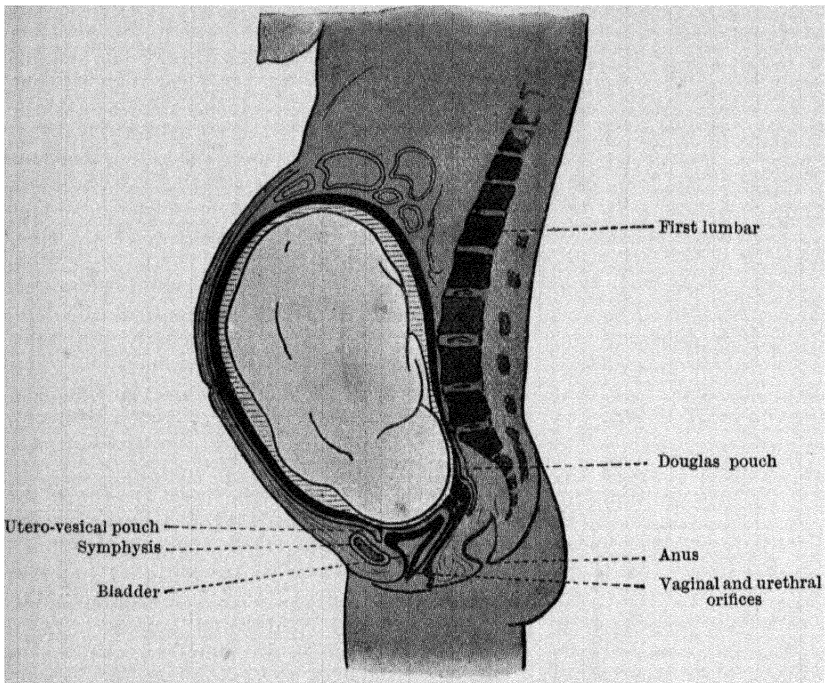


FIG. 20.—Uterus at term.

The child is then, to start with, lying with its head flexed and its occiput forwards and to the left. It does not, in ordinary cases, engage till the membranes have ruptured.

At this moment a diameter near the occipito-frontal diameter is lying in the plane of the brim.

The expelling force at this stage, that is, after rupture of the membranes, consists in the uterine contractions, exerted, since the fundus uteri is not yet in contact with the breech of the child, as a general intra-uterine pressure acting over the whole surface of the child excepting the vertex, which is in contact with the lower uterine segment.

Downward pressure comes to bear on the area of the vertex which overlies the os, and is, therefore, unsupported, and this pressure is acting in the axis of the uterus practically through the centre of the child's head.

*Flexion.*—The head is lying with a diameter near the occipito-frontal across the lower uterine segment. In this relation to the parturient canal the head is comparable to an egg lying in an elastic tube with its long axis not coinciding with the axis of the tube. If the egg is moved backwards and forwards, and friction reduced as far as possible by lubrication, it will soon come to lie with its long axis pretty exactly in that of the tube, and its small axis across it.

In the case of the head the long axis is the mento-vertical, and any move in the direction of accommodation, such as is made by the egg, would mean, in the state of partial flexion in which the head is now, a further flexion. The head then becomes more flexed, and the suboccipito-frontal diameter, the smallest available diameter of the head, owing to the attachment of the head to the trunk, takes the place of one nearer the occipito-frontal, and lies across the tube (Fig. 21).

This movement of flexion is assisted by the obliquity of the uterus. The uterus is, in the greater number of instances, inclined to the right, and any pressure acting on the base of the skull, and not at right angles to its surface, will tend to depress that end of the skull towards which the line of pressure is directed. In the position of the head under consideration the occiput is to the left, and therefore becomes depressed; that is, the head is flexed. It is likely that the uterine obliquity has comparatively little power in this direction before the fundus comes into contact with the breech, and fœtal-axis-pressure[1] comes into play; but that it has some is shown by the fact that left dorsal positions predomi-

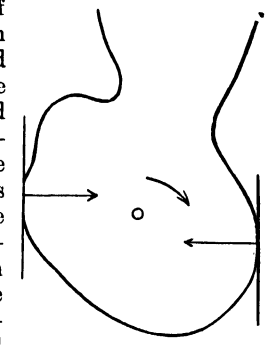


FIG. 21.—Effect of inward pressure of walls of birth-canal on moderately flexed head.

[1] *Fœtal-axis-pressure.*—This is a downward pressure exercised by the fundal end of the uterus on the fœtus in the long axis of the latter. It is made possible by the stiffening of the child produced by the contraction of the circular fibres of the uterus. It occurs after the greater part of the waters has drained away, and the fundus comes into contact with the breech.

nate in the proportion of about 9 to 3 in vertex presentations, whereas in face presentations the proportion is only 4 to 3. This shows that with the uterus in the usual inclination to the right there is a greater tendency for the head to be flexed in left dorsal positions than in those where the occiput is to the right; and a greater tendency for it to become extended (face presentation) in right dorsal positions than in left (Fig. 22).

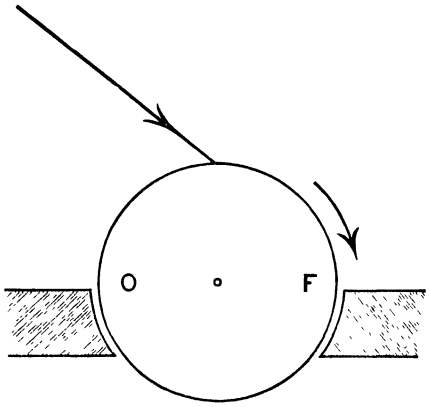



FIG. 22.—Effect of obliquity of uterine axis on head at brim. O, occipital; F, frontal end.

Still, the question of flexion or extension has in normal cases been decided before the head engages, and engagement in practically all cases precedes the establishment of fœtal-axis-pressure. But when the liquor amnii has drained away sufficiently to allow the axis-pressure to act, there appears an additional factor in retaining and possibly increasing the flexion of the head; for now the axis-pressure acts through the spinal column on the base of the skull at the condyles. A line continuing the direction of this pressure through the condyles to the level of the centre in bulk of the head, falls when the head is only a very little flexed somewhere between the centre of the head and its posterior end (Fig. 23). The occiput is thus further driven down; the occipital end of the head under the circumstances is, therefore, the first part of the head to encounter the resistance of the pelvic floor, and to be influenced by its slope.

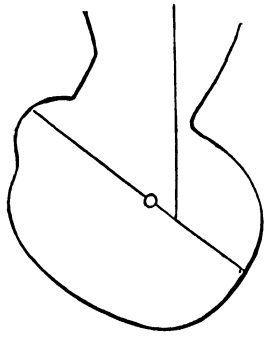


FIG. 23.—Fœtal-axis-pressure on slightly flexed head.

If a vaginal examination is made after the biparietal diameter has passed the brim, and before the head has come into relation with the pelvic floor, the first part touched is a point somewhere near the posterior upper angle of the right parietal bone. The sagittal suture is further back in the pelvis, and appears to lie close to the sacrum; the biparietal diameter is therefore oblique as regards the plane of the outlet of the pelvis, which is practically a plane at right angles to the axis of the vaginal canal. Naegelé observed this obliquity, and came to the conclusion that the biparietal diameter passed the plane of the brim with the sagittal suture nearer to the sacrum than to the pubes; and he described this as the relation of the head to the brim in normal labour. It is, however, not so, for the head passes the brim with its biparietal diameter lying in the plane of the brim. The explanation is that the head continues to lie in the same relation to the plane of the brim for some little distance after it has passed through the brim, and must therefore lie obliquely to the plane of the outlet (to which plane the results of a vaginal examination are referred), and this is nearly at a right angle with the plane of the brim.

The obliquity of Naegelé is an important part of the mechanism of labour in certain forms of contracted pelvis. It is known also as Anterior Parietal Obliquity.

*Internal Rotation.*—Owing to the shape of the pelvis, whose widest diameter in the cavity is the oblique (5 inches), the suboccipito-frontal diameter of the head (4½ inches) turns into the oblique, in which it already approximately lies. This movement of rotation on the axis of the fœtus is performed in the main by the head alone, though the shoulders take a certain share in it. Rotation in the pelvis is called INTERNAL ROTATION to distinguish it from a rotation of the head, which occurs after this part has escaped from the vulva, and is no longer under the influence of the maternal parts.

The head, descending a little lower, brings its occipital end into contact with the left half of the pelvic floor. This slopes inwards and downwards, and the occipital end of the head glides along its surface inwards and downwards to the anterior edge of the pelvic floor, thus finding its way under the pubic arch, in the middle line almost. The occiput by this movement along the pelvic floor carries on the rotation already begun till the suboccipito-frontal diameter is in the antero-posterior diameter of the outlet. This, it will be remembered, is the widest diameter (5 inches) of the outlet, and the suboccipito-frontal diameter would naturally tend to rotate into this, the oblique and the transverse diameters of the outlet being respectively 4½ inches and 4 inches, even if it were unaided by the slope of the left half of the pelvic floor.

This movement of internal rotation may be put in another way, perhaps more simply. Again, comparing the fœtal head to an egg in a tube, the long axis of the egg corresponding to the mento-vertical diameter of the head,

and the short axis to the suboccipito-frontal diameter, then an egg passing down a curved tube, like the lower end of the birth-canal, and having started with its long axis lying somewhat obliquely to that of the tube, would tend to place this long axis in exact coincidence with the axis of that part of the tube in which it happened to lie, and one of its poles would first emerge from the lower end.

If the head were not attached to a trunk, the mento-vertical diameter would come to lie exactly in the axis of the lower end of the genital canal. But the shoulders are now in the

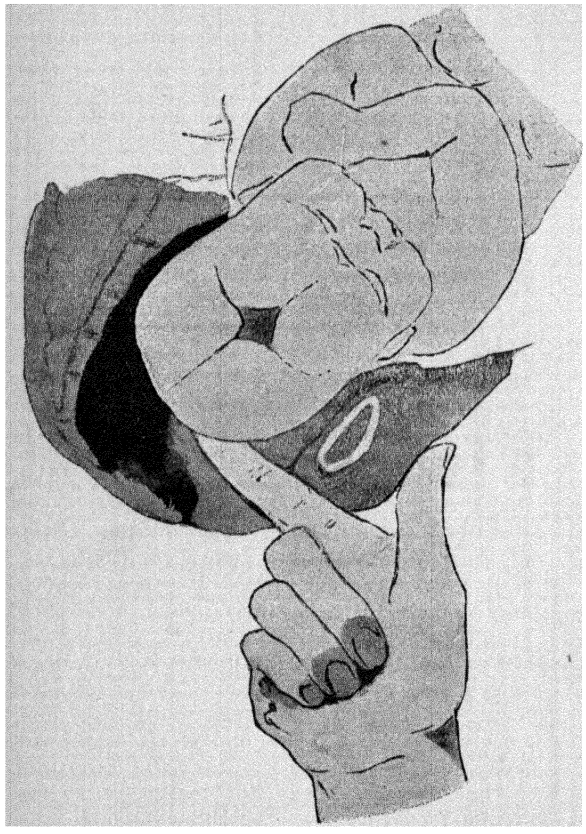


FIG. 24.—Relation of head to finger when occiput lies to the left.

brim, and are lying in the left oblique diameter; consequently there is a twist of the neck produced by the head rotating towards the antero-posterior diameter, and the tension caused by the twist prevents the rotation from being quite complete.

There is another reason for the incompleteness of the rotation. It will be seen later that the head in passing through the tightly-fitting tube of the parturient canal becomes moulded—that is, squeezed and diminished in whatever diameters happen to lie in the cross-section of the tube, and lengthened in those diameters which coincide with the length of the tube. While the head is lying obliquely as it engages, the poles of its ovoid, after moulding, will not be the anatomical poles of the mento-vertical diameter, but, in the position and mechanism under consideration, those of a diameter whose posterior pole is to the right of the middle of the vertex, and whose anterior pole is slightly to the left of the middle of the chin. This is the real ovoid with which we have to deal as the head passes under the pubic arch; and its lower pole, the point to the right of the middle of the vertex, is the one which will lie in the centre of the canal, and will first emerge from the vulva.

*Extension.*—It will be remembered that at the level of the pelvic floor the posterior wall of the genital canal takes a rather sudden bend forwards, and the axis has a corresponding bend. In consequence of this, the path of the head is changed from one in the axis of the pelvic brim to one in that of the pelvic outlet. Now the trunk is still lying in the upper part of the parturient canal, and is therefore in the axis of the inlet; therefore the head makes now a different angle with the trunk from the one existing before it entered the lower part of the canal. Since its dorsal surface is looking forwards the head necessarily becomes less flexed than before, and finally extended. The nape of the neck is at this time applied to the back of the symphysis, and its movement along this surface, which is the inner side of the curve, is very restricted compared to the large movement made by the anterior part of the head along the posterior wall of the canal formed by the pelvic floor and perinæum. The chin probably leaves the sternum to some extent during this extension.

The movement of extension begins to take place before the head has rotated into the nearly antero-posterior diameter of the canal, and there is on this account some inclination of the head towards the child's right shoulder.

As the head continues to advance, following the still curving axis of the canal, it becomes more extended, and the occiput moves upwards and forwards in front of the symphysis until the chin has escaped over the anterior border of the perineum, and the neck alone occupies the orifice of the vulva. The head is now out of the control of the canal, and any further rotations of it are produced by the influence the diameters of the canal have upon the shoulders.

The shoulders entered the pelvis with their bis-acromial diameter at right angles to the occipito-frontal diameter of the head, and therefore in the left oblique. They descend in this diameter till they come to the pelvic floor; the posterior shoulder then slides along the left half of the floor backwards till it lies in the bottom of the gutter formed by the two halves of that structure, and, the antero-posterior

diameter of the pelvic canal being at this level the largest of any, the bis-acromial diameter rotates into it.

Now, at the moment at which the head clears the vulva the shoulders are still in the left oblique, and the natural movement of the head to place itself at right angles with their width causes it to rotate immediately after emergence so as to face slightly to the right (Fig. 25). This first rotation is made very frequently with a jerk; then as the shoulders rotate completely into the antero-posterior diameter of the outlet the head moves farther round, so as at last to face the mother's right thigh. This is the movement of *external rotation or Restitution*, the latter name indicating that the head is now restored to the position it had at the moment of entering the pelvis, that is, facing to the right.

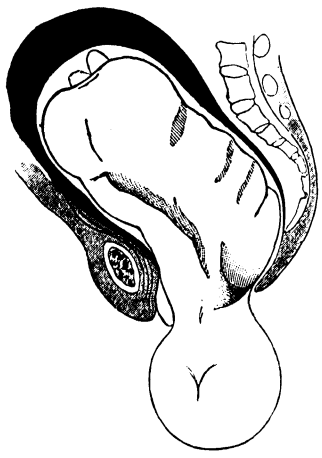


FIG. 25.—Relations of child to pelvis during delivery of shoulders. (From Winckel's frozen section.)

The delivery of the shoulders takes place in the same kind of way as that described for the head. The anterior shoulder appears first below the symphysis, and there forms a centre round which the posterior shoulder revolves. Both shoulders are born practically at the same time.

The arms are folded across the chest with the hands under the chin. The upper part of the thorax now lies in the outlet and the lower part in the brim, so that there is some lateral flexion of the trunk.

The hips come down in much the same way as the shoulders, their bitrochanteric diameter turning into the antero-posterior diameter of the outlet.

*Moulding of the Head.*—During the passage of the head through the birth-canal some of its diameters become altered owing to the considerable pressure to which the head has been exposed. The different ways in which alterations of the shape of the head by pressure are permitted by its structure have been already explained.

The head passes through the canal with its longest axis, the mento-vertical diameter, coinciding with the axis of the canal as nearly as the attachment of the trunk permits. This would mean that all diameters at right angles to this undergo compression if the coincidence were exact. Such is, however, not the case, for the leading point of the head is not found on the sagittal suture, but on the right parietal bone close to the suture. The compression takes place in a series of rings bounding planes at right angles to the line joining this point to the chin. Lengthening takes place along this line, and the head is obliquely distorted.

When the other positions of the vertex are considered it will be seen that when the left parietal bone is to the front, in the second and fourth positions, the end of the new long axis of the head is shifted to the left side of the vertex.

Each mode of delivery of the head has, according to the relations prevailing between the head and pelvis, a special moulding. These varieties will be described in order after each variety of mechanism.

### Diagnosis and Mechanism in Special Vertex Positions

*First Vertex.*—This is the one already described.

*Diagnosis.*—The occipito-frontal diameter is nearly in the transverse diameter of the brim with the occiput a little forwards. Per abdomen, the back of the child and the occiput lie to the mother's left, and the fœtal heart is heard on this side a little below the level of the navel. The limbs are to the right.

Per vaginam, the woman lying on her left side, the sagittal suture is felt through the sufficiently dilated os to run downwards and forwards, and to end in the posterior, triradiate fontanelle. The anterior fontanelle may be felt at the other end of the suture; and possibly the right ear, with the pinna directed downwards and forwards, can be reached.

*Mechanism.*—The head passes the brim, becoming more flexed. It then rotates completely into the right oblique. As it descends the occiput is directed towards the middle line, thus coming to the front, and passes under the pubic arch. The suboccipito-frontal diameter now lies in the antero-posterior diameter of the outlet very nearly. The nape of the neck is pressed against the lower border of the pubic arch, and the birth of the head is completed by extension. The shoulders come down in the left oblique, the right shoulder being in front.

When the head has completely escaped, the face makes a small movement towards the mother's right thigh, and this movement is continued as the shoulders rotate into the antero-posterior diameter of the outlet; so that the head lies at the end of restitution, with the face and occiput squarely to right and left respectively.

*Second Vertex.*—In this case it is only necessary to substitute left for right throughout the above description of the first vertex mechanism. The sagittal suture, being in the left oblique, runs upwards and forwards.

*Third Vertex.*—The occipito-frontal diameter lies in the right oblique nearly, with the occiput backwards.

*Diagnosis.*—Per abdomen, the back of the child lies to the mother's right, and the limbs to her left. The fœtal heart may be slightly more difficult to hear in this position, since the back of the child is directed rather away from the anterior abdominal wall; it is heard in the same place as in second vertex positions.

Per vaginam, the sagittal suture runs downwards and forwards as in the first position, but the posterior fontanelle is found at the end of the suture near the back of the mother's pelvis.

*Mechanism.*—The head descends as before, and, on meeting the pelvic floor, rotates through three-eighths of a circle to the same place as in second vertex positions. The case then proceeds as if the position had been a second vertex originally; it is thus said to have been "reduced" to a second vertex.

*Fourth Vertex.*—The occipito-frontal diameter lies nearly in the left oblique diameter with the occiput backwards.

Substituting left for right, the description of the mechanism of the third vertex will answer for this. The sagittal suture runs upwards and forwards, the posterior fontanelle being towards the back of the mother's pelvis. The head rotates so that it lies in the same position as if it had begun by being a first vertex; it is therefore "reduced" to a first vertex.

The mechanisms of the first and fourth, and of the second and third positions respectively, are the same except for the fact that in the two where the occiput lies backwards the rotation by which it comes to the front is one which describes three-eighths of a circle, instead of, as in the occipito-anterior positions, only one-eighth.

*Moulding.*—The way moulding in vertex positions is brought about has just been described. The diameters reduced are those at right angles to the long axis of the head, one near the mento-vertical. In all cases the suboccipito-frontal, suboccipito-bregmatic, and biparietal are diminished, and the mento-vertical lengthened. The occipito-frontal is in nearly all cases diminished somewhat. In first and fourth positions the prominent part of the vertex is on the posterior superior angle of the right parietal bone, and over a varying area around this; in the second and third positions the prominence is on a corresponding area on the left side. The caput succedaneum is over the prominence in each case.

The moulding in these, as in other vertex cases to be immediately described, is assisted by the movements of the flat bones of the vault on one another. Under the compression of the resistances encountered by the head their edges overlap to varying degree. The bone most pressed upon is the posterior parietal bone—that is, in first and fourth positions the left; and in second and third, the right. In consequence of the pressure, the posterior bone is flattened and slides under the anterior one. Since the frontal and occipital bones are attached to the base of the skull, and so cannot move so freely, they always go under the edges of the parietal bones.

*General Character of Labour.*—In the above mechanisms the course of labour may be considered as absolutely favourable for mother and child. Other presentations and positions have in their mechanisms elements which modify the prognosis for either mother or child, or both.

### Persistent Occipito-posterior Mechanisms

In certain cases, beginning with the occiput backwards (third and fourth vertex), the labour does not result in a reduction of these positions to second and first respectively, but the head is born with the face still looking to the pubes. This occurs in rather more than 1 per cent of vertex cases.

The *cause* of this irregularity is *want of flexion.* Flexion to a sufficient degree, as has been shown, is necessary to bring the occiput down low enough to be the first part of the head to come into relation with the pelvic floor; for if this does not happen, there is no more reason why the occiput should rotate to the front than that the forehead should, since both ends of the head ovoid reach the floor of the pelvis at the same time, and both are equally directed forward by the slopes on which they impinge. Further, there is, owing to the absence of flexion, no longer the suboccipito-frontal diameter of 4 inches to easily rotate through the transverse diameter of the pelvic cavity (4½ inches), but the occipito-frontal of 4½ inches has now to be reckoned with. This cannot move through the transverse, and so comes to be acted on by the shape of the pelvis at this level. The direction of least resistance for it to move in is for the occiput to rotate into the hollow of the sacrum.

Want of flexion is brought about in several ways. It is in some instances due to one or other of the causes which, when acting to a far greater extent than at present, produce face presentations. Thus a slightly contracted pelvis may have just enough want of space in its antero-posterior measurement at the brim to retard the biparietal diameter, which is near the hinder end of the head, for a time, and to allow the forehead to come down more than is normal. The obliquity of the uterus may interfere with the necessary amount of flexion, if the inclination happens to be such that the line of the expelling force is directed along the abdominal surface of the child; as when the uterus has its normal obliquity to the right and the head is lying with its occiput to the right (see Fig. 22, p. 175).

Flexion is apt to be interfered with in all

cases of occipito-posterior position more than in those with the occiput forwards, for after the head has descended somewhat into the brim the child will lie with the promontory of the sacrum fitting into the nape of its neck, and this tends to keep the cervical spine extended rather than flexed. Still, in the large majority of cases the causes leading to flexion manage to right this.

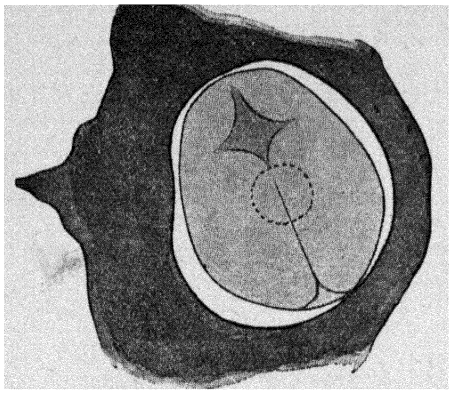


FIG. 26.—First vertex. Pelvis of woman lying on her left side, seen from below. Biparietal diameter free.

Also, as Herman points out, the sacro-cotyloid diameter in a normal pelvis is less than the full oblique diameter. Now when the occiput lies backwards the biparietal diameter occupies this sacro-cotyloid diameter, and is more retarded than if it lay in the full oblique (Figs. 26 and 27). In consequence flexion is somewhat interfered with, just as happens in similar

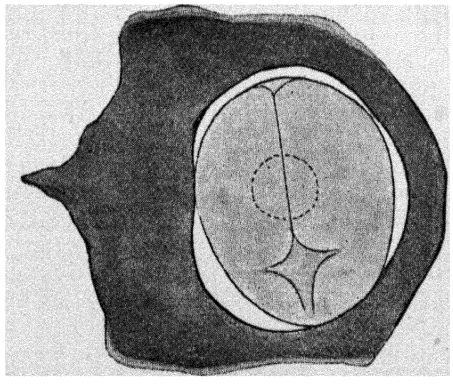


FIG. 27.—Third vertex of same pelvis. Biparietal diameter gripped.

conditions in certain forms (elliptic brim) of contracted pelvis.

*Diagnosis.*—It can in most cases, by the unusual lowness of the anterior fontanelle, be determined after the head has passed the brim and is engaged in the cavity, that the occiput is going to rotate backwards. In well-flexed cases this fontanelle cannot be reached at this stage without some difficulty.

*Mechanism.*—The description of this may begin when the occiput has just rotated into the sacrum. The forehead lies against the back of the symphysis. The head revolves round this, the occiput descending, probably much influenced by the fœtal-axis-pressure (see p. 174). The flexion is in most cases assisted by a slight gliding upwards of the forehead behind the pubes. As the head advances the occiput is pressed on to the pelvic floor and perineum, forcing the latter backwards and then passing over its edge. Directly this has happened and the posterior pole of the head is free, it moves

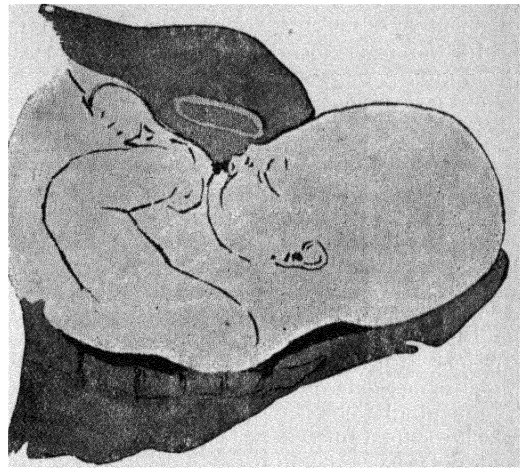


FIG. 28.—Persistent occipito-posterior. (Moulding of head exaggerated.)

backwards still farther over the perinæum till the nape of the neck presses on the edge of the perineum. The head is now born by extension, and the forehead, face, and chin glide under the pubic arch. The shoulders come down with the shoulder which was originally forward still to the front.

*Modified Mechanism.*—There is a modification of this mechanism which is occasionally seen. In it the forehead does not descend so low, and, in fact, hitches on the upper edge of the symphysis. Flexion takes place as before, but at a higher level. The head often remains fixed in these cases, and labour is arrested. The vertex is found distending the perineum to some extent, and the cause of arrest is not readily obvious. When attempts at delivery are made by the forceps, the blades are put on with a little more difficulty than usual owing to the full diameter of the head not being able to be grasped by them; and when traction is made the forceps invariably slips off. The writer's experience, which is doubtless shared

FIG. 29.—Rarer form of mechanism in persistent occipito-posterior cases.

by others, is that this condition of affairs is a very common cause of a consultation being necessary. The remedy for it is to carry the handles of the forceps very far back while the blades are being locked, and at the same time to push the head bodily backwards by pressure applied to the forehead above the pubes.

*Moulding of the Head.*—In the first described and commoner mechanism the diameters compressed are the occipito-frontal and the biparietal. The head is thus made rather dome-shaped, and the suboccipito-frontal and suboccipito-bregmatic diameters are rather increased than diminished.

In the rarer form the head resembles the head after a very severe occipito-anterior labour.

*General Character of Labour.*—The head is longer in passing through the pelvis than in the last group where the occiput turns forwards (p. 177), as it offers greater resistance to moulding along the lines in which moulding is required; and also there may be, as above mentioned, slight pelvic contraction. The fronto-occipital diameter (4½ inches) distends the perinæum, and makes rupture probable in multiparæ, and certain in primiparæ. There is an increased risk of septic absorption, partly because of the laceration, and partly because of the manipulations necessary.

The child is little, if at all affected, unless there is long detention of the head. (For Management, see p. 198.)

### Mechanism in Face Presentations

The principles involved in the mechanism of labour when the head presents by the face are identical with those already described as governing the mechanism in vertex cases, the only difference being that different diameters of the head are in relation with those of the pelvis.

Fig. 30.—Relation of head to trunk in face presentation.

The head is extended instead of flexed.

The general effect of this is that a somewhat less favourable relation exists on the part of the head both to the forces expelling and to the passage. The blunter face takes the place of the occiput, and therefore a less effective wedge-action is brought about at the time when the head has become the dilating agent — that is, after the membranes have ruptured; and further, the fœtal-axis-pressure is not applied so nearly at right angles to the base of the skull as in the presentation of the vertex, but impinges on the skull at a tangent.

*Frequency.*—The head presents by the face in about 1 in 300 of all cases.

*Mode of Production.*—The head becomes extended for several reasons, which may act separately or in combination.

1. *Uterine Obliquity.*—This has been shown in the case of vertex presentations where the head lay in the commonest position of the vertex, namely, with the occiput to the left, and where the uterus had the usual obliquity, namely, to the right, to distinctly favour flexion. Where, however, one of these conditions is reversed—for instance, where the occiput lies to the right—the arrangement does undoubtedly favour extension, and the head will be brought first into the attitude of a brow presentation, and then into that of a face (Fig. 22, p. 175).

The explanation already given in reference to the causation of flexion in vertex cases need not be made again at full length.

2. *Flat Pelvis.*—If the brim in this case is of the elliptical variety, the biparietal diameter will have to lie in a diameter of the brim, which will to a greater or less degree retard its advance. This is a diameter to one side of the conjugate, and roughly parallel to it, since in this class of pelvis the head enters the brim in the transverse diameter.

The biparietal diameter lies nearer to the occipital end of the head than to the frontal, and in consequence the occipital end will be retarded while the frontal end is allowed to advance. The result of this is to extend the head (see also p. 179).

3. *Dead Child.*—Dead children present by the face in a larger proportion than living ones do. The reason of this is that the normal muscular tone is wanting, and the head may reach the brim in any attitude; and then if there are other forces (obliquity of the uterus in a suitable direction, for instance) which will tend to extend the head, a face presentation is readily produced.

4. Other causes of far less importance sometimes bring about a face presentation. A goitre may sometimes be large enough to cause extension of the head by its bulk. It has been said that an unusually long head (dolichocephaly) is very liable to present by the face; this is quite uncertain, and the type would have to be extraordinarily well marked to cause this result.

Before entering on the detailed description of the mechanism in each kind of face presentation, it will be well to point out the features wherein face cases differ from those of the vertex.

The chin takes the place of the occiput in being the most advanced part of the head, and the occiput comes last.

The submento-vertical diameter (4½ inches) takes the place of the suboccipito-frontal (4 inches) in relation to the walls of the birth-canal. A larger diameter has therefore to pass, and there is proportionate delay.

The chin does not project so far in advance of the general mass of the head as the occiput

does, and so does not so soon come under the influence of the pelvic floor. In the case of the chin being behind, rotation forwards takes place later in face presentations than rotation forwards of the occiput in occipito-posterior vertex cases.

Moulding takes place with more difficulty than in vertex presentations, for the whole hind-head has to be depressed on to the back of the neck. This means more delay in the case of a closely-fitting head and pelvis.

*The positions* of face cases are four, and are named according to the direction of the chin. They are :—

1st, or right mento-posterior. The long diameter of the face is in the right oblique diameter of the brim. This is called the first position, since it is derived from the first vertex position by extension.

2nd, or left mento-posterior, in a similar way from the second vertex.

3rd, or left mento-anterior, from the third vertex.

4th, or right mento-anterior, from the fourth vertex.

In each of these cases the forehead in the face presentation lies in the place occupied by the occiput in a vertex presentation.

It will be easy to remember the relations of the corresponding positions of the vertex and face if it be kept in mind that the back of the child looks in the same direction whether the case be one of face or vertex.

It may again be stated for the sake of clearness, that although in *naming* the positions the forehead takes the place of the occiput, the chin represents the occiput in the mechanical relations of the process.

### Diagnosis and Mechanism

First Face Position.—*Right Mento-posterior.*—This, being derived from the commonest vertex presentation, is naturally the commonest position of the face.

*Diagnosis.*—On abdominal examination the back of the child is found lying to the mother's left; the limbs are rather prominent on the right side. There is a sharp angle between the back and the occiput. The fœtal heart, if it is heard, is most easily audible on the same side as the limbs, namely, the right. This is, of course, due to the extension of the neck and the upper part of the thorax, which makes them lie near to the uterine wall, since the convexity of the fœtal trunk is now on the anterior surface.

On vaginal examination, if the os is sufficiently dilated, some part of the face is usually felt, most commonly the orbital and glabellar region. The bridge of the nose is traced backwards and upwards (in the ordinary obstetric position) to the mouth, where the alveolar ridges are felt, and beyond this the chin. The right cheek is anterior.

*Mechanism.*—The head passes through the brim in the right oblique diameter, becoming slightly more extended, and the chin impinges on the pelvic floor. The child can now descend farther only by rotation of the chin forwards, for its neck is as far extended as possible, and the tension of its curved axis is very considerable. It thus comes to have the relation to the curve of the axis of the parturient canal that an elastic rod, whose natural curve was one with the concavity backwards, would have to a tube

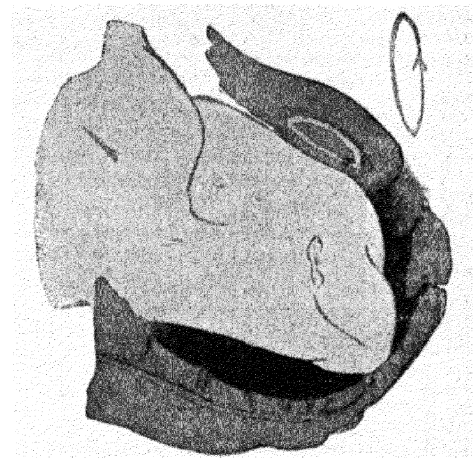


Fig. 31.—Mechanism in first face presentation. The curved arrow shows the direction of the chin-rotation.

containing it whose curve was a forwardly concave one. In this case, if the tube allows of a certain amount of movement on its axis on the part of the rod, and there is little friction, the rod will rotate on its axis until it lies with its curve coinciding with that of the tube; that is, with both their concave sides facing the same way. It will readily be seen that in the case of the fœtus the tendency will be for it to come to lie with its chin forwards, so as to relax the tension of the over-extended head and thorax. The chin will be guided by the slope of the right half of the pelvic floor forwards and downwards till it appears at, and escapes from, the under surface of the pubic arch. This tendency of the chin to come forwards is less opposed by the shape of the pelvis than in the case in those positions of the vertex where the occiput lies behind; for the length of the face is less than the occipito-frontal diameter, and it can turn through the transverse diameter of the cavity without difficulty. The chin then rotates through three-eighths of a circle, and the face comes to lie in the antero-posterior diameter of the pelvic outlet. The angle between the chin and the neck now "hitches" under the pubic arch—to use a convenient but rather incorrect expression—and the bulk of the head is pushed forwards by the pelvic floor, producing a movement of flexion. The face, forehead, vertex, and occiput successively clear the perineum, and the head is born, the chin

rising up in front of the symphysis in the same way as the occiput does in vertex presentations.

The head is now free to move on the shoulders. These have by this time rotated into the right oblique diameter, the right shoulder being to the front as at the beginning. The face therefore looks towards the mother's right thigh, and restitution takes place exactly as in vertex positions.

SECOND FACE POSITION.—*Left Mento-posterior.*—The face here lies in the left oblique diameter of the brim, with the forehead to the front.

*Diagnosis.*—The back of the child is to the right and the limbs to the left. The angle between the back and occiput, felt per abdomen, is on the right side, and the fœtal heart, if heard, is on the left.

On vaginal examination the left cheek is anterior; the bridge of the nose can be traced backwards and downwards to the mouth and chin.

*Mechanism.*—The head descends, passing the brim on the left oblique diameter; and after that it follows, *mutatis mutandis*, the same course as that just described for the first facial mechanism.

In the first and second facial positions it will be noticed that the chin has to make a long rotation of three-eighths of a circle to reach the space under the pubes, thus contrasting with the corresponding vertex positions, in which the rotation of the occiput is the shorter one of one-eighth of a circle. In the third and fourth facial mechanisms, on the contrary, the rotation of the chin is a short one, while it will be remembered that the third and fourth vertex cases undergo a long rotation.

THIRD FACE POSITION.—*Left Mento-anterior.*—The face lies in the right oblique diameter of the brim, with the forehead backwards.

*Diagnosis.*—The back of the fœtus is to the right, and the limbs to the left. The fœtal heart is heard on the left. The left cheek is to the front; the bridge of the nose can be traced forwards and downwards toward the mouth and chin.

*Mechanism.*—As the head descends the mechanism is simple. The chin is directed forwards, rotating through one-eighth of a circle, till it comes into the sub-pubic space. The head is then born by flexion, as has been described in the first facial position, when it has reached this stage.

The shoulders are in the left oblique diameter of the pelvis, the left shoulder being in front. As they rotate into the antero-posterior diameter at the outlet the face undergoes restitution, and looks directly to the left, just as in the third vertex position.

FOURTH FACE POSITION.—*Right Mento-anterior.*—The position and relations in this mechanism are the same as in the last described one, "left" being substituted for "right," and *vice versâ*. The mechanism corresponds.

If we now contrast the mechanism in vertex cases on the one hand with that of face cases on the other, it will be seen that the following are the most important points of difference:—

In vertex cases there is flexion at the beginning; the third position rotates into the second, and the fourth into the first. Delivery of the head is accomplished by extension.

In face cases there is extension at the beginning; the second position rotates into the third, and the first into the fourth. The head is delivered by flexion.

Also, in mento-posterior positions, reduction can, and usually does, occur later than in occipito-posterior ones.

*Moulding of the Head.*—The head in face presentations does not lie with its long axis so nearly parallel to that of the parturient canal as is the case in vertex presentations; and so, although the longest axis of the ovoid is the one which is, on the whole, lengthened, the diameters which are shortened are not exactly the same as in the vertex moulding. The lengthening takes place along the fronto-occipital and mento-occipital diameters; the shortening along the cervico-vertical diameter, or one close to it. The occipital region is compressed between the back of the neck and the wall of the birth-canal, and is therefore squeezed into a rather sharply-pointed wedge.

There is, in face cases, a peculiar and somewhat unaccountable prominence of the forehead, in spite of the fact that the face is compressed along its vertical diameter. This is due to the greater firmness of the frontal bone, which, in addition to the fact of its being of greater thickness at its lower part than the other bones forming the vault of the skull, is also really part of the base of the skull as far as its orbital portion is concerned, and is therefore not easily bent.

The caput succedaneum is usually formed when the face is at the vulva, or close to it, and lies near the angle of the mouth—on the right side in first and fourth positions, and on the left in second and third positions. If a caput is formed while the head is still within the os, or high up in the canal, it will appear in the mento-posterior positions somewhere near the left or right eye, according to which eye lies to the front. The tumour is sometimes very large, and is not seldom the seat of much ecchymosis.

*Persistent Mento-posterior Mechanism.*—The chin sometimes fails to rotate forwards, just as in the case of a posterior occiput. This is very rare, for the mento-frontal measurement, which is about 3¼ inches, does not prevent rotation by its length in regard to the transverse diameter of the cavity, as happens in the case of the fronto-occipital diameter in persistent occipito-posterior positions (p. 178). The chin comes forward under the influence of the pelvic floor

quite easily as a rule, even if the head has descended well on to the pelvic floor before rotation is begun.

In addition to this, the curve-tension of the fœtus has here a very powerful effect. If the head descends deeply into the pelvis with the chin still backwards, the extension of the head and neck is very considerable, and the tension in the direction of flexion is very great. We have therefore the curved rod (p. 181) bent by the shape of the tube in which it lies into a reversed curve. Given the comparatively unrestrained movement on its long axis secured by the short mento-frontal diameter, the trunk of the child will rotate on this axis as already described.

It is fortunate that reduction is so much the rule; for with an unreduced mento-posterior position, in the case of a normal pelvis and head, *delivery cannot take place.*

The reason of non-reduction is analogous to that of vertex cases. It is due to insufficient extension, whereby the most favourable diameters are not brought into relation with the pelvis. In other words, the presentation is one very nearly that of the brow (see below), and a diameter near the mento-vertical is thrown across the pelvis. Such a diameter will measure about 5 inches, and will prevent forward rotation; the chin will therefore move backwards into the hollow of the sacrum.

There is a great difference between the state of things now present and those which obtain in the case of occipito-posterior mechanisms. In the latter the head flexes a little more; the occiput clears the perinæum, and frees the head. Here, however, the anterior fontanelle is jammed against the back of the pubes, and to enable the chin to clear the perinæum, an amount of additional extension, of which the head is not capable, is required. For, as the base of the skull comes deeper into the pelvis, to enable this extension to take place, it brings with it the neck, and after a little more descent the upper part of the thorax. A wedge is thus endeavouring to enter the pelvic brim which the latter is unable to accommodate, and impaction results. To enable delivery to take place, the head has to be reduced in size by perforation. In some cases of small or dead children the head has been able to extend sufficiently to permit the chin to escape over the perinæum, and allow of flexion of the head, and the gliding of the face and forehead from behind the pubes.

*Moulding of the Head and Caput.*—These changes are pretty much the same as those found in normal face presentations. The caput will be found over the eye and adjacent parts, on that side of the face which lies anterior in the pelvis.

*General Character of Labour.*—The prognosis is not so good in these cases as in vertex presentations. Labour is prolonged as already mentioned, and manipulations are often necessary. In the unreduced mento-posterior cases the mother runs all the risks of arrested labour.

There is danger to the fœtus from over-extension of the neck, especially when the chin is backwards; and the cord may prolapse. (For Management, see p. 199.)

### Brow Presentations

The head in these cases, which are very rare, is in an attitude midway between flexion and extension; and the longest diameter, the mento-vertical (5½ inches), endeavours to engage in the brim. The head is, in consequence, in a state of unstable equilibrium, and no doubt practically all face cases pass through this condition at one time or another in their progress; so that until it engages in the brim, or at all events persists in endeavouring to engage, this attitude has no special importance.

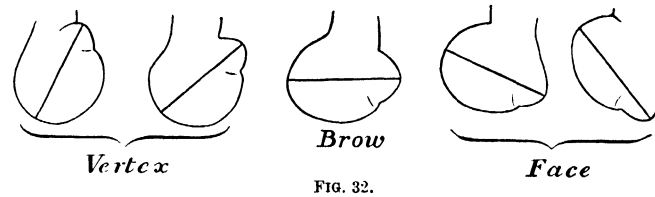



FIG. 32.

Engagement at all, in the case of a normally sized head and normal pelvis, is impossible until a very great amount of moulding has taken place.

*Mode of Production.*—When the head lies on the brim in this attitude the downward pressure must have a vertical direction; or if there is a tendency towards flexion or extension, the obliquity of the uterus must be exactly enough to counteract such tendency and preserve the unstable equilibrium. Thus the cause of brow presentations is the same as that of face presentations, but acting less completely.

On each side of brow presentations may be placed a series of presentations of the vertex and of the face. Nearest to it on the one side, that of flexion, is the imperfectly flexed head that leads to persistence of the occipito-posterior position, and nearest on the side of extension is the incompletely extended face presentation which leads to persistent mento-posterior mechanisms.

*Diagnosis.*—On abdominal examination, if the woman be a favourable subject, the projection of the occiput and the chin can be made out, one on each aspect of the child. The head will in practically all cases be lying high, since it cannot enter the brim.

Per vaginam, if the head can be reached, the bregma is found at one end of the presenting part and the glabella at the other. The orbital arches will be recognised, and will indicate the anterior surface of the child.

*Mechanism.*—After the head has been reduced in its longest diameter by moulding at the brim it descends, probably by slight advances of the chin and occiput alternately; but the amount of advance in neither case is enough to convert the presentation into a face or a vertex. Rotation is controlled entirely by the shape of the pelvis, for the fit is a very tight one indeed; so that whichever end of the head lies most to the front at the beginning comes round under the pubic arch eventually—it is usually the chin.

The forehead, now elevated into a marked projection, descends to the vulva, and presents there. The head then flexes, rotating round some part of the face, usually about the glabella, which lies under the pubic arch. The vertex and occiput then glide over the perineum, and the head is freed by slight

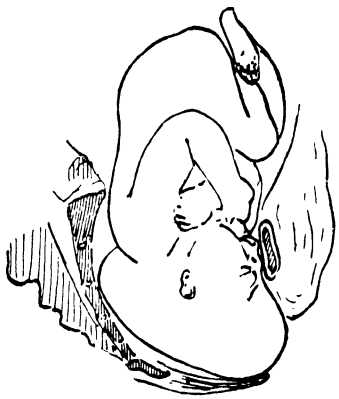


FIG. 33.—Relations of head and pelvis in brow mechanisms

extension and the passage of the face and chin under the pubic arch.

If the chin rotates backwards, delivery in ordinary cases is impossible; but if the head is very small and easily moulded, the chin will descend by extension and be born over the perinæum, the vertex afterwards passing under the pubic arch by flexion.

Even in the more favourable mechanism the chances of delivery without perforation of the head are extremely small.

Restitution will take place according to the rules already laid down.

*Moulding and Caput.*—The head is distorted to a remarkable degree. The frontal bone is elevated, as already mentioned, and the head slopes down from this to the occiput very sharply. The diameters lengthened are the occipito-frontal and the suboccipito-frontal; those shortened are the cervico-bregmatic and the mento-vertical.

*General Character of Labour.*—The mother runs great risks in these cases from the prolonged labour and the necessary manipulations and use of instruments. The perineum is sure to be much lacerated.

The nervous centres of the child may be greatly damaged by the considerable compression undergone by the head. (For Management, see p. 200.)

### PODALIC LIES

These lies include presentation of the full breech; of the incomplete breech; of the knees, or a knee; and of the feet, or a foot.

*Full Breech.*—This is the commonest presentation of the podalic end of the child, because the natural attitude of the child *in utero* is that with the thighs and knees flexed. This attitude makes the presentation consist of the buttocks and the feet, the legs being crossed and closely applied to the front of the body.

*Incomplete Breech (siège décomplété, mode des fesses*, of French authors).—The legs are extended on the thighs, so that the feet lie by the side of the child's head. It is pretty certain that this attitude is the one, in many cases, which has prevailed during pregnancy, for after the delivery of such children the limbs frequently fly back to the same place, even when they have been brought down into their natural attitude. In other instances the legs, after delivery, will remain as they are placed, and the extraordinary attitude must have been produced during labour by the hitching of the heels at the brim.

*Knee presentations* are very rare, and probably produced by manipulations in most cases.

*Footling presentations* may be of one or of both feet. If only one present, the half breech (one buttock, and possibly one foot) remains to dilate the maternal passages. But if both feet come down the passages are very imperfectly dilated before the head, and this is a matter of great importance. The half breech attitude is, in all cases of podalic version, the one intentionally produced.

*Causes of the Podalic Lie.*—The proportion in which it occurs is about 1 in 40 of all cases. It is due to the absence or to the inversion of the causes which bring about the cephalic lie in such a very large proportion of labours.

It occurs thus in cases of *hydrocephalus*, where the head-end of the child is larger than the breech, and is better accommodated in the fundal end of the uterus, and in cases where for some cause the specific gravity of the child is not decidedly near the anterior end of the child, as in *premature children*. In these last, too, the size of the child is not sufficient to make its lie in the uterus a matter of importance, and it is able to occupy either end of the uterus by its head indifferently. It occurs where there is *excess of liquor amnii*, or where the *uterine walls are unduly lax*, as they are sometimes in multiparæ. *Twin pregnancy* causes the adaptation of the fœtuses to the cavity of the uterus to be different from that present in single cases. *Contracted pelves* prevent the head from resting on the cup formed by the normal brim, and thus

cause it to be more readily displaced; and in these cases the whole uterus lies higher than usual in the abdomen, and it and its contents are more readily displaced. A *placenta prævia* fills up the brim and renders the seat of the head less secure.

The presentations of the foot and knee are brought about by a want of close fitting between the breech and the brim, whereby a part of the presentation is allowed to prolapse in front of the rest.

*Positions.*—The positions are named according to the direction in which the sacrum looks. They correspond, therefore, to the positions of the vertex.

The diameter, however, which governs the rotations of the pelvis of the child in the parturient canal is the bitrochanteric, which is at right angles to the sacro-pubic, and may be in either oblique diameter of the pelvis, with the sacrum facing either backwards or forwards. The positions with the sacrum forwards are commoner that sacro-posterior ones, owing to the lumbar convexity of the mother fitting more easily into the ventral concavity of the child.

### Diagnosis and Mechanism

*First Breech.*—Left sacro-anterior.

The bitrochanteric diameter lies in the left oblique diameter.

*Diagnosis.*—On abdominal examination the back of the child is found to lie on the left side of the uterus, and to be continued upwards into the easily recognised head which occupies the fundus. The fœtal heart is heard to the left of the navel, a little above it.

Per vaginam, the cleft between the buttocks lies in the right oblique diameter. Usually about the middle of the furrow the anus may be made out, and in front and to the left of this the coccyx and sacrum with the sacral spines, near the foramen ovale. It is not easy to distinguish the sex of the child by its genital organs at this stage.

*Mechanism.*—The breech descends with a rotation of the anterior trochanter, the left, to the front, obeying the shape of the pelvis and of the gutter of the pelvic floor. The left trochanter appears at the vulva, and the right trochanter forms the outer extremity of the curve produced by the pelvis revolving round the left trochanter as a centre. This revolution is the same as that of the head round the suboccipital region in vertex mechanisms.

The revolution causes a lateral flexion of the trunk, for the shoulders lie pretty well in the plane of the brim. Rotation is combined with slight extension of the trunk, for the child does not rotate so completely as to bring its bitrochanteric diameter into the exact antero-posterior diameter of the outlet. This, again, is quite comparable to the slight lateriflexion of the head found in vertex cases at this stage (p. 176). Both buttocks are born together.

An external rotation takes place immediately the hips are free, for the shoulders are now lying in an oblique diameter, and the trunk has acquired a twist. The hips, in consequence, rotate a little way back so as to occupy their former position, the sacrum looking slightly forwards. As the shoulders now come down they rotate into the antero-posterior diameter of the pelvis; the left shoulder forms the centre of a revolution in which the right sweeps down the posterior wall of the canal, and both shoulders are born together.

The head comes into the pelvis somewhat flexed. The occipito-frontal diameter lies in the left oblique with the occiput forwards. The occiput, as the head descends, rotates to the front, obeying the shape of the outlet. The longest diameter, the mento-vertical, remains as far as it is allowed by the connection of the head with the neck, in the axis of the canal, and it is on this coincidence of axes that the proper delivery of the head greatly depends.

The nape of the neck is now lying against the lower edge of the pubic arch, and the whole head revolves round this point, the chin soon appearing at the vulva. The vertex is the last part of the head to be born.

The delivery of the head is the critical part of breech presentation, for not only is the progress at this stage slow, owing to the fact that the uterus has contracted down to its smallest useful size, and thus leaves the expulsion to be completed by the vaginal and abdominal muscles, but the child is in a precarious condition. For the placental area is contracted, and the blood-supply to the placenta is much diminished; the cord is suffering pressure between the head and the pelvic wall; and the body-surface of the child is very liable to be so stimulated by the cold air in which it now finds itself as to bring about an inspiratory effort by reflex action, and to possibly fill the lungs of the child with liquor amnii, meconium, and blood contained in the vagina. The methods of assisting the delivery of the after-coming head will be considered elsewhere.

As the head rotates into the antero-posterior diameter of the outlet it brings the trunk round in the same direction, and causes it to face towards the mother's back, the left thigh and shoulder lying close to the mother's right thigh.

*Second Breech.*—Right sacro-anterior.

The bitrochanteric diameter lies in the right oblique.

*Diagnosis.*—Per Abdomen.—The back is to the right, and the limbs to the left. The head is felt at the fundus. The fœtal heart is heard to the right of, and above, the navel.

Per vaginam, the sacrum is found near the right foramen ovale, and the cleft between the buttocks runs in the left oblique diameter.

If left and right are interchanged, the *mechanism* of this position is described in the same way as that of the first breech.

*Third Breech.*—Right sacro-posterior.

*Diagnosis.*—The bitrochanteric diameter lies in the left oblique. The back looks to the right and rather backwards, and the limbs to the left and slightly forwards. The heart may not be heard; if it is, it will be found on the right side above the level of the navel.

*Mechanism.*—As the breech descends, the right hip rotates towards the pubic arch. The trunk is lateriflexed, and also bent slightly forwards. The hips are then born in the same way as in the mechanisms just described, and the abdomen of the child is turned to the mother's left thigh.

After this there are two ways in which the rest of the child may follow:—

1. The trunk may continue to rotate in the same direction as that in which it has already moved, and the shoulders will then descend in the right oblique, the right shoulder still being in front. The occiput is thus brought forwards, and the rest of the mechanism is that of a second breech.

2. The shoulders descend in the same oblique diameter as the hips, and the head will then lie in the right oblique with the occiput slightly backwards.

The head then comes down and the occiput is rotated forwards. This is mainly on account of the tension in the direction of flexion which the upper part of the child is now experiencing. If further descent were to take place with the occiput backwards, the chin would have to be still further flexed on the thorax to allow the neck and upper part of the chest to follow the curve forwards of the maternal canal at this level; whereas if the occiput comes forwards the tension is at once relieved, and the head, by an easy extension, is born as in the preceding mechanisms. Also, if the head came down with the occiput not forwards a long diameter of the head would be thrown transversely across the outlet—one, in fact, near the mento-vertical, or at least the mento-occipital—and this would cause the walls of the canal to be stretched in a way that would bring about a rotation of the head into another position, this position being one with the occiput forwards (see p. 176).

In the first kind of mechanism the rotation forwards of the back of the child occurs between the passage of the hips through the brim, and that of the shoulders through the same ring; and in the second, after the passage of the shoulders.

*Fourth Breech.*—Left sacro-posterior.

The bitrochanteric diameter is in the right oblique, and substituting left for right throughout, the mechanism is the same as that occurring in the last described position.

*Abnormal Mechanisms in the Sacro-posterior Positions.*—These variations occur when the head descends into the pelvis with the occiput backwards and the head insufficiently flexed. Then, just as in presentations of the head-end of the child, the longer diameters of the head lie across the pelvis, and prevent the rotation forwards which normally takes place. The occiput is found, therefore, in the hollow of the sacrum, unable to rotate forwards as it has done in the hitherto described sacro-posterior positions.

The usual thing to happen now is that the occiput shall hitch on the edge of the perinæum, which fits into the nape of the neck. The head then flexes farther on to the chest; and the chin and the rest of the face glide under the pubic arch.

A less common way of delivery of the head in such a case is for the chin to hitch behind the symphysis, and for the head to revolve round this as a centre. The longest (mento-vertical) diameter of the head is thus thrown across the outlet, and the head is delivered as an inverted face, plus the length of the chin.

*Footling Presentations.*—The *diagnosis* of this presentation can be made from the vagina only. One or both feet may present, and one or two limbs may be found in the vagina. The only other presentation with which a foot can be confused is that of the hand and arm. The characteristic part of the foot is the heel, to which no counterpart exists in the hand. The toes all lie close together, and there is no thumb to be separated from the rest of the digits. The heel is distinguished from the olecranon, with which it might be momentarily confounded, by tracing the sole of the foot forwards and finding the toes. The direction of the heel shows the direction of the occiput.

*Mechanism.*—If both feet are down, labour is easier in its early stages than in the case of a breech. But when the shoulders, and later the head, come down, these parts have to do the dilatation which should have been performed by the breech, and there is corresponding delay; otherwise, the mechanism is that of a breech. If, however, only one foot present, the other being doubled up in its normal position, the state of affairs is much more favourable. The half breech is able to dilate the passage pretty well, and this presentation is the one always artificially produced after version.

It is important to remember how the mechanism is modified by the presentation of one foot. This foot is the lowest part of the child, and is therefore first influenced by the trend forwards of the pelvic floor; it is in consequence rotated to the front, wherever it may be to start with. The bearing of this is, that when version happens to be required in a pelvis of which one side is more roomy than the other, and in which it is desirable to make the occiput pass through the larger half, the

operator has it in his hands to place the occiput in which side he prefers. For instance, if the right side of the pelvis is the larger, he will bring down the right leg of the child, which, coming to the front, causes the occiput to come down into the right side of the brim.

*Knee Presentations.*—The knee is recognised by its size and by the movable patella. It is liable to be confused with the shoulder only, and an abdominal examination will prevent this mistake being made.

*Moulding of the Head in the Podalic Lies.*—The head passes through the pelvis flexed, that is, as has been already explained, with its longest diameter as nearly as possible in coincidence with the axis of the parturient canal. In consequence, the diameters shortened are almost the same as those in vertex presentations with the occiput forwards. There is the difference, however, that the vault of the skull is not pressed in as it is in the head-first cases, where it has to overcome the resistance of the pelvic floor, and so the suboccipito-frontal and suboccipito-bregmatic diameters are not so much reduced. There is also, perhaps, slightly less complete flexion in breech cases. The fronto-occipital diameter is shortened considerably, and the head is thus rendered slightly dome-shaped. If the child is born alive, however, there can be but little moulding; for the head is not long in the pelvis, and remains for a still shorter time on the perineum.

*General Character of Labour.*—The mother's safety is not endangered in these cases unless manipulations are necessary. The child, however, is in some danger if delivery of the head does not take place speedily after the trunk is born. For the placental site is contracted, and the supply of oxygenated blood to the child cut off; there is much risk of compression of the cord between the head and the mother's pelvis; and the stimulus of cold air on the surface of the child's trunk is very liable to cause inspiratory efforts while the mouth and nose are lying in the vagina, and mucus, liquor amnii, and blood will in that case be sucked into the lungs.

In unreduced sacro-posterior positions the delay and the risk are greater.

The child's sterno-mastoid muscle is occasionally torn, and a hæmatoma produced. This is sometimes followed by wry-neck. (For Management, see p. 200.)

### Transverse Lies

In the case of a transverse lie the long axis of the child is at nearly right angles with that of its mother. In practically all examples of this abnormal lie the shoulder is the presenting part, and the head lies at a considerably lower level than the breech.

*Causation.*—A transverse lie has been shown, in discussing the mode of production of the normal lies, to be, under ordinary circumstances, a condition of unstable equilibrium for the child; and there must, therefore, be either a cause continually in action to keep the fœtus in this relation to the long axis of the mother's uterus, or an absence of most or all of those forces which tend to place the child in the axis of the uterus.

Such conditions are found to be--

Contracted Pelvis.—This cause acts through the increased uterine obliquity usual in contracted pelvis, and by the head being prevented from entering the brim.

Prematurity.—There is disproportion between the child and the uterus, and the lie is indifferent (see p. 174).

Death (with, possibly, decomposition) of the Fœtus.—There is no muscular tone, and the compact ovoid shape of the fœtus *in utero* is not preserved.

Twin Pregnancy.—The shape of the combined ovoid is irregular.

Placenta Prævia.—The lower uterine segment is filled up and the long diameter of the uterus shortened.

Hydramnios.—The same reason holds here as in prematurity.

Tumours in the pelvis, or fibroids in the uterine wall, may displace one of the poles of the fœtus.

*Positions.*—The child may have its back anterior or posterior; and in either case its head may be to the right or left.

Owing to the dextro-rotation of the uterus the child will not lie in the transverse diameter of the brim, but rather parallel to one of the oblique diameters. The back is most commonly directed forwards for the same reason as in all lies; and owing to the dextro-rotation of the uterus the head is usually in the left iliac fossa, since this is the lowest part of the uterine cavity.

*Diagnosis.*—In all cases where there is no tumour complicating the case the diagnosis can be made *by the abdomen*. The shape of the uterus is characteristically altered, for its long diameter is transverse instead of vertical.

The head is felt in one iliac fossa. The breech is higher than the head, and is about half-way up to the fundus. The lie of the child can usually be quite well made out whether the uterus is contracted or relaxed.

Per vaginam, there will early in labour probably be no presenting part to be made out. Later on the arm frequently prolapses, and in any case the shoulder may be reached when it has been forced down on the brim. If there is any doubt at first, after an abdominal examination has been made an anæsthetic should be given, and the pelvis and abdomen thoroughly explored.

If the membranes are unruptured when the examination is made, the characteristic finger-like shape of the bag will be recognised. Care must be taken not to rupture them during an examination.

If the shoulder is felt it has to be distinguished from a knee, from the breech, and from the side of the face. Its characteristic points are the clavicle, acromion process, and spine of the scapula. If the examining finger is able to pass the point of the shoulder and to reach the ribs in the axilla, there can no longer be any doubt.

If the elbow is at the os, it might be confused with the heel owing to the projection of the olecranon. The finger should be passed along the surface continuous with the projection, and the absence or presence of the sole of the foot, ending in the toes, will serve to identify the part. But such confusion cannot possibly arise if a careful abdominal examination has been made. The direction of the head and of the back of the child can be ascertained by abdominal and by combined examination. The axilla felt per vaginam shows which way the head lies, and the spine of the scapula shows the back. If the arm has come down, the thumb after supination of the hand will point to the head, and the palm will show which is the ventral surface of the child. If the prolapsed hand is the right one, the right hand of the physician will be able to grasp it as in shaking hands; whereas if it is the left, this cannot be done.

*Natural Course of Labour if unassisted.*—The prognosis is very unfavourable in these cases, both for the mother and the child. Speaking generally, the usual course of things is that the os dilates very slowly, owing to the projection of the bag of membranes. This projection in the form of the finger of a glove, already mentioned, is due to the fact that there is no ball-valve in this case, such as is provided by the head in normal cephalic lies, to prevent the whole intra-uterine pressure coming on the bag of membranes. The membranes are therefore thrust forward unduly, and they rupture long before they have done their work.

The cord often presents, and when the membranes rupture, prolapses, for the presenting part does not fit the lower uterine segment, and the cord slips past. This is made more easy by the child's belly being close to the os.

When the membranes have ruptured, the liquor amnii drains rapidly away, and the uterus retracts on the fœtus. The fœtus is driven down into the lower uterine segment, which soon thins owing to the tension caused by the retracting upper segment and to the transverse stretching caused by the bulk of the child. The uterus may now become *tetanic*, and the woman may die in that way soon from exhaustion; or the uterus may become *exhausted*, and then on a renewal of its efforts may become tetanic.

*Rupture of the vagina or lower uterine segment* may occur.

*Spontaneous delivery* of one of the kinds to be mentioned may take place.

In all these cases the child soon dies after the membranes have ruptured, owing to the retraction of the uterus. This arrests the placental circulation by pressure, and in the same way kills the nerve-centres. (For Management, see p. 203.)

### Spontaneous Delivery

This is possible only when the child is dead or small.

If it is alive, and possesses its muscular tone, delivery may happen by (1) spontaneous rectification; (2) spontaneous version; or very rarely by (3) spontaneous evolution.

If it is dead it may be delivered by (1) spontaneous evolution, or by (2) spontaneous expulsion (corpore conduplicato).

*Spontaneous Rectification.*—This occurs above the brim and with unruptured membranes. By this movement the lie is converted into a cephalic one. It is brought about by the tendency of the uterus to resume its normal shape during contraction. The projecting head and breech are pushed in towards the middle line, and the child is caused to lie in the axis of the uterus. This movement would be represented artificially by cephalic version.

*Spontaneous Version.*—This is a podalic version of the child taking place spontaneously. As the uterus contracts the breech is forced down, and the trunk of the child is pushed across the brim in the direction of the head. This must take place soon after the membranes have ruptured and when not too much liquor amnii has escaped. Then, even if the arm has prolapsed, which it often does, the elastic spinal column is able to transmit the pressure in such a way that at the cephalic end of it the direction of the force is converted into an upward one, the lowest point of the curve of the spine dipping a little way into the brim. The head rises and the breech is forced into the brim; the shoulder rises out of the brim, and the lower part of the trunk turns into the hollow of the sacrum, thus bringing the head to the front well above the symphysis. The curve of the fœtal spine, which has its convexity downwards to start with, comes now, on account of the descent of its caudal end, into the pelvis, to have its convexity upwards. The lower end of the child being in the axis of the cavity, the head is able to be pushed by the inward pressure of the uterine walls into the middle line, and the case is thus converted in an ordinary podalic lie.

The important feature of this mechanism is that the descent of the caudal end of the fœtus into the pelvic cavity takes place with the shoulders above the brim. This will be appreciated when the next paragraph is read.

*Spontaneous Evolution.*—The events in this method of delivery occur below the brim. The child is practically always dead or very premature, though delivery of a child at term by this movement has been recorded.

The difference in mechanism between this and the last described case begins when the child lies with its shoulder in the brim. The spinal column, being devoid of tone, does not form an elastic rod as in spontaneous version; so that when the uterus forces the breech down the shoulder is thrust deeper into the pelvis, and does not glide across the brim. The shoulder is driven down into the pelvic cavity; this is followed by the side of the thorax, the abdomen, and finally the breech, which finds itself in the hollow of the sacrum. The side of the neck is now jammed against the back of the symphysis, and the shoulder comes down under the pubic arch and presents at the valve. The thorax and then the breech are forced down past the head and neck and are born, the legs being the first part to make a complete escape from the canal. The rest of the trunk and the head then follow, as in a breech case.

*Spontaneous Expulsion* (corpore conduplicato). —The child here is always dead and small. The body is born doubled up, flexion taking place about the lower dorsal region, which is born first. The chest is squeezed against the belly, and the head and pelvis are born together last.

## Management of Labour


| | |
|---|---|
| Preparations for | 189 |
| Antiseptic in | 190 |
| Anæsthetics | 192 |
| Management of 1st Stage | 193 |
| ,, ,, 2nd Stage | 194 |
| ,, ,, 3rd Stage | 196 |
| ,, after Delivery | 197 |
| Management of Special Presentations | 198 |
| *Occipito-posterior* | 198 |
| *Face* | 199 |
| *Brow* | 200 |
| *Breech* | 200 |
| *Transverse* | 203 |


In the general management of labour there are three essential important indications:—

1. To prevent any septic infection from being introduced from without.

2. Be ready to assist when necessary during labour, and thus recognise early and possible dangers. Assistance may be required to prevent undue length of labour from any cause, retention of any parts of the placenta or membranes; also help will be required to arrest hæmorrhage, to avoid lacerations to the genital tract or accidents to the child.

3. Reduce suffering to a minimum by the administration of an anæsthetic.

Preparations for Labour.—If, as is usually the case, the doctor has seen the patient at least once before labour commences, special instruction should be given as to the management of the last fortnight of pregnancy, in regard to the care of the nipples, the systematic clearing out of the rectum, and the use of hot baths; vaginal douching, if any reason to believe that there is a venereal discharge (see "Pregnancy, Management of"). In most cases it is necessary to give the patient a list of the appliances that must be in the house, and to advise as to the choice of the room and position of the bed.

*Choice of a Room.*—The room in which the labour is to take place should preferably be large and airy, with a south or western exposure if possible. A patient always gets on better if the room is bright, and gets a certain amount of sunshine. There should be an open fire-place (not a gas stove), and a good window to ensure proper ventilation. A fixed basin in the room is never to be commended, as there is always the possible danger of sewer-gas entering by it. If the room can have a dressing-room opening off it so much the better, as the bathing of the child, and the nurse's preparation of food, douches, etc., can be carried on without disturbing the mother. It is also better for the nurse to sleep in the second room at night. The temperature of the room should be kept at 60° to 65° F.

*The bed* should be fairly hard, so as not to form a pit where the patient lies. A feather bed is out of the question. A narrow bed, standing out into the middle of the room, is the most convenient during labour, as the patient can be more easily got at, and the administration of the anæsthetic is easier. If the bed is not narrow, and cannot be placed so that there is access to both sides, it must be arranged so that the doctor has access to the patient's right side; if this is not done, the examination of the patient when on her left side has to be made with the doctor's left hand, or otherwise entails a great deal of moving. The bed is made up in the ordinary way, then covered with a mackintosh sheet, well tucked over the edge, and covered with a draw-sheet. It is of great advantage to have over this a thick square of absorbent wool, which is burned after it is soiled by discharges. This should be changed once or twice during labour.

*List of Special Articles required*

Douche can, 2 quart size.

Glass vaginal nozzle.

Higginson's enema syringe.

Gum-elastic catheter or rubber catheter, size 8.

Bed-slipper.

Mackintosh sheet (size 1 yd. sq. at least).

4 absorbent wood wool sheets: 2 large size, 32 in. × 32 in.; 2 medium size, 26 in. × 20 in.

2 dozen large-sized wood wool towellettes. These are better in every way than ordinary linen diapers.

4 binders of strong towelling (linen or huckaback), 1 yd. wide × 1¼ yds. long. The binder is folded lengthways.

Box of assorted safety-pins, some specially large for the binder.

Small bottle of best olive oil. (For removing vernix from the child.)

Creolin.

Chloroform, 4 oz.

Linen thread, 1 hank, boiled and put into a bottle.

Some pieces of fine clean linen. A pair of scissors (blunt-pointed).

A dusting powder of equal parts of boracic acid, prepared chalk, and starch; this can be scented with rose or violet.

Boracic acid solution.

Brandy or whisky.

A new nail-brush.

A packet of absorbent wool.

Fluid extract of ergot, 4 oz.

Castor oil, 3 oz.

Complete set of clothing for child (see "Newborn Child").

The medical man should attend as soon as possible on being summoned. Not only is it a great mental relief to the patient, but it also is an opportunity to diagnose the sort of labour that is likely to take place. If the presentation is abnormal, it allows of a chance of rectifying it by postural treatment during an early stage.

The practitioner should take with him:—

A lubricant for the fingers; a tube of 1 in 1000 corrosive sublimate and glycerine is useful, it does not get infected as a pot of vaseline would.

Tabloids of corrosive sublimate.

A pair of forceps.

Needle-holder, pair of scissors.

Needles, and strong silk or catgut.

Hypodermic needle, with hypodermic tabloids of ergot, morphia, strychnine, and digitalis.

### Asepsis and Antiseptic Measures

It is not too much to affirm that the most essential point in the successful management of a midwifery practice is a thorough knowledge of the theory and practice of asepsis. The extraordinary reduction in the mortality shown by the records from the various lying-in institutions since the adoption of antiseptic methods, proves that if the principles of aseptic midwifery could be perfectly carried out there would be no deaths from septicæmia, and that there would be also a great reduction in the diseases directly due to childbirth. With regard to this point, some interesting figures are given by Dakin from a study of a large series of hospital statistics; he shows that the unavoidable deaths from childbed which are caused by diseases other than septicæmia may be taken as about ·2 per cent, which figure represents the present ideal death-rate. With care, equally good results can be obtained even under apparently adverse circumstances. Thus, out of a series of 1549 consecutive cases under the writer's care in out-patient dispensary practice, the total mortality was 4, or 1 in 397, or ·2 per cent; the causes of death were rupture of the uteris, pulmonary embolism (2), and pneumothorax (phthisis pulmonalis). These results were simply due to *strict cleanliness* in all the details of the confinement.

Before studying in detail the methods of preventing infection from the hands and clothes of the doctor or nurse, and from dirty instruments, etc., a brief reference may be made to nature's method of keeping the vagina *sterile*.

Krönig has shown that the normal vaginal secretion in pregnant women has a germicidal reaction. It contains non-pathogenic bacteria. The vagina has been found to be aseptic within forty-eight hours after the introduction of septic bacteria. This bactericidal power is not apparent; it is not due to a simple process, but it is quite possibly the result of a joint chemical, mechanical, bacterial, and leucocytic action. In addition to the germicidal action of the vaginal discharge, the operculum or plug of mucus blocking the cervix completely prevents the entrance of bacteria into the uterus. When the membranes rupture, the liquor amnii washes out the greater part of the vagina and carries off the bacteria present. After the birth of the child, when the walls have been stretched to their utmost, the liquor amnii that comes after the child is able to wash out any remaining bacteria. In this way the uterus is kept free by nature from bacteria after delivery.

As in the course of labour vaginal examination and operations must be performed, it is necessary to do all that is possible to avoid the introduction of germs. The keynote to this is *absolute cleanliness* in every detail.

In arranging the method to be adopted it is well to have the details as simple as possible, so that they can be constantly carried out. It must also be remembered that many antiseptics are incompatible when mixed together.

Soap decomposes corrosive sublimate, iodine, and permanganate of potash; carbolic acid and permanganate of potash are incompatible. Carbolic acid and soap can be used together.

*Cleansing of the Hands.* — The thorough cleanliness of the hands is as important for the nurse as for the doctor, and neither should touch the genitals of the patient without having washed the hands in some such way as the following:—First, scrub the hands, and especially the nails, thoroughly with soap and hot water (the variety of soap is not of importance provided there is a good lather); then rinse the hands in plain hot water to remove all the soap, and thereafter soak them in a 1 in 500 solution of corrosive sublimate for one minute. Never place the hands direct from the soapy water into the antiseptic, as soap decomposes corrosive sublimate. It is not necessary to use a lubricant for the examining finger; but if it is preferred, avoid the use of oily or fatty materials, as it is almost impossible to render them aseptic.

Pots or boxes of so-called antiseptic vaseline give a false sense of security; they are usually far from being aseptic, having been contaminated by discharge and blood-stained fingers.

A collapsible tube of carbolic acid (1 in 40) and glycerine, or a 1 in 500 mixture of corrosive sublimate and glycerine, are perfectly safe. The benefit of having the lubricant in the tube lies in the fact that it cannot get soiled by dirty fingers. Carbolic acid is the more useful, as it can be used for forceps; if the sublimate is used the mercury is deposited on the instrument.

*Cleansing of Appliances and Instruments.*—*Vaginal nozzles* are best made of glass, and should be boiled for at least five minutes before use. The *catheter* to be used should be new. Before and after using, both these instruments should be washed and kept in an antiseptic solution — 1 in 40 carbolic, or 1 in 1000 sublimate.

*Mackintosh sheets* must be washed over with carbolic before being put on to the bed, and bed-pans should be carefully washed and disinfected before and after use.

All the *instruments* to be used should be of metal, so that they can stand being boiled; and this should be done for five minutes, and after boiling they are immersed in an antiseptic solution.

*Cleansing of the Patient.*—This must be thoroughly carried out to ensure that the douche, or clean fingers of the doctor or nurse, or the nozzle of the douche, will not get contaminated from bacteria round the external genitals, and thus carry infection into the vagina. At the commencement of labour, and before any examination is made, the nurse should thoroughly wash with soap and water, and then rub over with a 1 in 40 carbolic lotion, or wash off the soap, and then scrub with a 1 in 1000 corrosive sublimate. This washing of the external parts is most important, and must be repeated from time to time if the labour is long, and is also necessary at least once daily during the puerperium. An old sponge or loofah should not be used for this, but rather a new piece of flannel, or a bit of absorbent wool or tow. If flannel be used, it must be washed out and kept in solution until required; the wool and tow can be burnt.

If any operation is to be done which entails the hand or instruments passing into the uterus, the vagina must be disinfected as well. This would not be necessary if we could be certain that the vaginal discharge is normal, but it so often is swarming with bacteria that are capable of becoming pathogenic when introduced into the uterus. To do this douche the vagina (see *infra*), then scrub it all round with the fingers and a small piece of soap, and then repeat the douche.

*Douching.*—Krönig, when making experiments on the normal vaginal secretion, found that after douching with plain water the germicidal action was lessened, and after a corrosive sublimate douche the action was destroyed, probably by precipitating the albumin. Thus it seems that both ante-partum and post-partum douching, apart from being quite unnecessary as routine work in a normal case, may actually do harm. Careful douching is, however, indicated under the following conditions:—

*Ante-partum.*—1. When there is an offensive or *purulent discharge* from the vagina, *e.g.* of venereal cancer, etc.

2. In cases where any *operation* or *manipulation* is to be performed in the uterus.

3. If the liquor amnii has lain long in the vagina during a *prolonged labour* it may begin to decompose, therefore douching is advisable.

*Post-partum.* — 1. Where any *operation* or *manipulation* has taken place inside the os uteri during labour. This category includes cases where forceps have been applied to a head above the brim, internal version, induction of premature labour, removal of retained or adherent placenta or membranes.

2. In some cases where the *membranes have ruptured early*, and the labour is unduly prolonged. Owing to the loss of liquor amnii during the long period there will be none left to wash out the vagina after the child's birth.

3. If the *fœtus* has been *putrid*.

4. In all cases where there has been a *purulent discharge* either before or during labour.

5. Any time during the puerperium if the *lochia become fœtid*.

6. Cases of *post-partum hæmorrhage*. Here, however, the object of the douche is not so much its antiseptic or cleansing property as the promotion of uterine contractions so as to arrest hæmorrhage.

*The Composition of the Douche.*—Its antiseptic properties are only of use by keeping the water in the douche antiseptic. The antiseptic substance does not remain long enough in the vagina to destroy the bacteria. The water used should be boiled, allowed to cool, and strained through muslin. Corrosive sublimate is not of much use for douching purposes: if used before labour, it renders the tissues rough and more rigid; after delivery, if used too strong, or if any is left behind, it may cause symptoms of mercurial poisoning (spongy gums, foul breath, diarrhœa, and abdominal pain). The strength of this substance should be, if used, from 1 in 5000 to 1 in 8000; if stronger, the symptoms of poisoning are very apt to appear. Members of the phenol groups of substances are more satisfactory: carbolic acid 1 in 60, or *creolin* 1 *tea-spoonful to the quart of water*. This latter is an exceedingly safe and useful substance, and, being non-poisonous, can be used always.

The douche should be given by means of a

*douche can* rather than a Higginson's syringe, the advantage being that a constant stream can be applied, and there is thus much less chance of introducing air. It can be given in almost any position; there is, further, no chance of introducing any pieces of debris and clot that are apt to get drawn into the tube, as is the case where a Higginson's syringe is employed. The can should be capable of holding two quarts of solution. The nozzle is best made of glass, with the perforations in the sides of the nozzle and not one central one at the top, in case of injecting fluid into the uterus. This might quite easily occur immediately after labour when the os is patent.

To administer the douche, if a bed-pan is available, the patient must lie on her back, and with the shoulders raised and the bed-slipper arranged beneath her. If not, she should lie in the left lateral position, with the hips drawn well over to the edge of the bed; this is less likely to soil the bedding. For a cleansing douche, the antiseptic solution should be at a temperature of 100° F. to 110° F. If required to check hæmorrhage, the temperature must be from 115° F. to 120° F. When the patient complains of the heat a little soap or carbolised glycerine smeared over the labia, perineum, and buttocks enables the patient to bear the heat better A small quantity of the fluid should first be run off, so that the tube may contain no air when it is introduced into the vagina. The nozzle is passed for 2 inches into the vagina, and the tap turned on. The left hand of the nurse must be laid over the uterus to prevent the fluid finding its way into the uterus, or from the uterus into the tubes, and then out through the ostium abdominale. This is very unlikely to happen with a vaginal douche and with a nozzle not having a central perforation. Retention of the fluid in the vagina is prevented by pressing firmly down into the fundus in the axis of brim at the end of the douche, and at the same time depressing the perineum by the nozzle.

*Sanitary Condition of the House.* — This should be ascertained to be free from defective drainage; any sewer-gas finding its way into the lying-in room from water-closets, fixed-in basin, ventilating pipes, is a source of illness and danger during the puerperium. It is, however, doubtful if sewer-gas is ever the actual cause of septicæmia; probably it is not.

### Anæsthetics

Anæsthesia is as justifiable in all obstetric cases as it is in surgical operations, and in the more difficult cases it is as indispensable. In a normal labour it not only prevents the acute suffering which accompanies the second stage of labour, but is frequently of actual assistance in the progress. If the anæsthesia is carried to a deeper degree than is required to alleviate the pain, the spastic state of the uterine and cervical muscle becomes relaxed, and the voluntary action of the abdominal and pelvic muscle is abolished, thus enabling any operation, *e.g.* turning, to be carried out much more easily than it otherwise could be. Probably the best anæsthetic to use is *chloroform*. In spite of all that may be alleged against its use, it has the following advantages: chloroform is more manageable and more rapid in its action, and also more agreeable than *ether*. Further, when given in small quantities short of surgical anæsthesia, it exercises its effects in some degree, and it does not require the undivided attention of a skilled administrator.

If complete anæsthesia is necessary, *ether* can be used, as there will be almost invariably a skilled assistant present who can devote his whole attention to the anæsthetic.

The immunity from danger during anæsthesia possessed by parturient women is well known, and lasts until the birth of the child. A very few cases of death have been recorded, all occurring when the patient has been anæsthetised to the full surgical degree. The cause of the immunity is not known: one reason ascribed has been the physiological hypertrophy of the heart which tends to prevent syncope; another theory is that alterations in the vaso-motor system of the pregnant woman enable her to resist the toxic action of chloroform to a greater extent than usual.

The possibility of post-partum hæmorrhage should be borne in mind; the general relaxation of uterine tissues produced is supposed to increase the dangers of hæmorrhage. Hæmorrhage is very rare after the administration of chloroform if sufficient attention be paid to the uterus during the third stage of labour.

If a healthy woman in labour inhales a small quantity of chloroform she quickly passes into a semi-comatose state, perception is diminished, and the general sensibility is dulled, yet she is quite conscious when spoken to. During the intervals between the pains she lies quietly asleep, but at the commencement of a contraction she grows restless, groans, and, if the os uteri is fully dilated, she bears down; she appears to be conscious of the pains, but does not suffer from them.

The anæsthesia has not much effect on the contractions; the frequency at first is slightly diminished, but it soon regulates itself—each individual contraction becomes more energetic and effective than before, on account of the resistance from the rigidity of the canals being reduced.

An anæsthetic is of special value in nervous, excitable patients, who, on account of the fear of increasing their own sufferings, almost entirely abolish the assistance that is obtained from the voluntary efforts of the abdominal muscles. When deep anæsthesia is necessary, as in cases

of obstetric operation, an assistant is usually required in order to allow the physician to devote his whole attention to the operation.

THE RULES FOR THE ADMINISTRATION OF CHLOROFORM.—The anæsthetic should not be started until the end of the *first stage of labour*. Before this there is little need for it on the ground of suffering, but in some cases of *rigid cervix* it may be employed after simpler remedies have failed.

During the administration there should be perfect quietness in the room. Chloroform may be given by the open method, or preferably by one of the graduated methods, *e.g.* Krohne's apparatus, or one of its modifications, described vol i. p. 141. If the graduated method is not available, a few drops of chloroform are put on the end of a towel and should be given only when a pain is coming on, and then is withdrawn as soon as the pain is over. During the *second stage* the anæsthesia is most useful. The amount given is gradually increased as the head descends. This, by alleviating the suffering to a great extent, enables the patient to bear down more fully. As the head emerges at the vulva the patient should be fairly deeply under; this allows the doctor to have more control of the movements of the head, and thus there is less danger of a ruptured perineum. *After the birth of the child* the anæsthesia should stop, but there is no advantage gained by wakening the patient artificially. As the chloroform is supposed to predispose to post-partum hæmorrhage, the uterus must be more carefully guarded than usual. It is unnecessary to give chloroform in this stage even for the repair of slight tears of the perineum, as the parts are usually insensitive from the stretching they have undergone; but if the perineum is badly ruptured, or if the placenta is adherent, chloroform must be given.

No harmful effect of chloroform upon the child has been established. Occasionally there seems to be some slight delay in the establishment of respiration in the new-born infant, but with slightly more vigorous stimulation this is soon got over.

THE ADMINISTRATION OF AN ANÆSTHETIC UNDER SPECIAL CONDITIONS.—In cases of *heart disease* the administration of an anæsthetic is necessary, as the labour should in most cases be completed as soon as possible. The slight tendency to post-partum hæmorrhage is here rather an advantage than otherwise, as it will relieve the extra burden thrown on to the heart when the change in the circulation takes place after the birth of the placenta.

In cases of *anæmia*, after placenta prævia or accidental ante-partum hæmorrhage, it is better to give ether. If the labour is accompanied by troublesome pulmonary complications, chloroform is the best anæsthetic.

For the treatment of *convulsions and chorea*, and in the performance of almost all major obstetric operations, the use of the anæsthetic is necessary.

### MANAGEMENT OF FIRST STAGE, OR STAGE OF DILATATION

This stage begins with the commencement of true labour pains, and ends with the complete dilatation of the os. Its duration is about fifteen hours in primiparæ and eleven hours in multiparæ. The chief indications for treatment are: (1) to assist nature in every way; (2) to maintain the strength by means of suitable nourishment; (3) to avoid needless examination.

Labour progresses most satisfactorily when the pains are regular. A great deal can be done by keeping the patient quiet, interesting her, and not allowing her to be disturbed or annoyed by the presence of undesirable relatives.

The nurse's tact and consideration in this stage are most helpful. The right sort of woman will try to interest and cheer the patient in every way.

*The dress* of the patient should be a clean night-gown, doubled up on a level with the crest of the ilia, and pinned on the shoulders, and a petticoat of some flannel material loosely tied round the waist.

An excellent and most convenient form of petticoat will be found in taking three yards of flannel with a tape run into it lengthways. This is tied round the waist, and the fulness taken to the back, and the ends pinned together with safety-pins. This has the advantage of being most easily raised up during the second and third stages of labour, and also it can be very easily removed with the minimum of disturbance to the patient. The *patient* also wears a larger size of woollen stocking and loose slippers; also, during the first stage, she should have on a dressing-gown.

One has at this stage constantly to keep in mind the factors that may interfere with the progress of labour. These are: (1) the *position of the uterus* not being that best suited for the ovum to dilate the os; (2) a *loaded rectum*; (3) an *over-filled bladder*. At this time the patient usually assumes an erect attitude, and either walks about or sits on a chair. This should be encouraged, as the position helps the natural process of dilatation of the os, owing to the influence of gravity and the force of the pains driving the ovum downwards on the os. Voluntary "bearing down" is of no use, as it does not increase the force acting on the os, and it is, further, very exhausting. If the uterus be anteverted or obliquely inclined, the action of the pains presses the head on the brim of the pelvis rather than against the os uteri, and thus a serious delay to labour is caused. This can be rectified by applying a firm abdominal binder, and letting the patient lie on her back until the head fixes in the brim. A straight

piece of strong linen towelling is much superior to any of the varieties of shaped bands.

A loaded rectum may seriously interfere with the progress of labour by offering an obstruction to the descent of the head. The method I have found which ensures the rectum being in a satisfactory condition during labour, is for the patient to take for the last week of pregnancy a sufficient dose of some mild purgative (*e.g.* liquorice powder) every night. Then, on the onset of labour, if the bowels have been opened within five hours, do nothing until the first stage has lasted about eight hours, then *always* clear out thoroughly with a soap, water, and glycerine enema. If there is any reason to doubt that the bowels have had the thorough preliminary clearing, give an enema on the onset of labour, and another enema before the commencement of the second stage.

*The bladder* very rarely requires the use of the catheter during the first stage. But, if necessary, it must be used.

As this stage will probably last from eight to twelve hours, the second indication, that of maintaining the general strength, may call for consideration. At the beginning of labour there is no reason why she should not have a full meal of plain food. After this the patient will not be at all inclined for anything beyond some hot drink—a cup of warm milk, coffee, tea, a cup of soup or thin gruel. The stimulating effect of the hot drink acts often by increasing the strength of the pain. This, of course, can be repeated at intervals. If the labour has been going on a very long time, and the pains are growing feeble, the question of giving an opiate has to be considered.

On first seeing a patient at the commencement of labour a thorough careful abdominal examination should be made (as described under section Diagnosis, p. 169). This, however, requires to be supplemented by a vaginal examination at the commencement of labour; it is well to repeat the examination after rupture of membranes, in case the rush of liquor amnii has caused the prolapse of the cord or of an arm. Thorough antiseptic precaution must be taken regarding the cleanliness of the external genital organs and the operator's fingers, hand, and forearm. All instrumental and digital interference, such as early application of forceps, digital dilatation of the cervix, and unnecessary vaginal douching, are much to be deprecated.

### Management of Second Stage

This, the stage of expulsion of the fœtus, extends from the time of complete dilatation of the cervix to the delivery of the child. Its duration is variable; in primiparæ it may last for three to four hours, while in multiparæ the average time is from one to two hours. It occasionally occurs that the delivery is exceedingly rapid, and the child is born after a very few pains. Such a rapid second stage is, however, not desirable, as it predisposes to post-partum hæmorrhage, and is very apt to be accompanied by laceration of the vagina and perineum. It may also be the cause of imperfect involution of the uterus.

The chief phenomena are the *regular and intermittent uterine contractions* aided by the *voluntary contractions of the abdominal muscles.* The sequence of events is: (1) the rupture of the membranes, brought about by the removal of the support from the cervix; (2) the gradual expulsion of the child into the vagina; (3) the pressure of the head on the perineum, followed by its birth.

The lines of treatment are now as follows:— After complete dilatation of the os the membranes have served their function, and if they have not ruptured spontaneously, it is best to do so artificially, as their presence now retards the advance of the child's head. *Artificial rupture* is best performed during a pain by scratching through the thin membranes with the nail. If they are too tough for this, the stilette of a catheter may be used under the necessary antiseptic precautions. It is difficult to render a hair-pin aseptic, and therefore it should not be employed. After rupture and the partial escape of the waters, the head, being no longer kept back by the fluid, comes well down upon the cervix. The uterus, with the escape of the liquor amnii, is able to contract and retract on to the body of the fœtus, and this acts as a stimulus to the pains, which have now passed from dilating pains to expulsive pains. There is frequently a short cessation from pains immediately after the rupture, while the uterus is retracting on the child.

During the early part of the second stage the patient should lie down; she can assume any position that she cares to, except when an examination is made, when she should lie on the left side with the hips down well to the edge of the bed. Some arrangement must be made to help her in her bearing-down efforts. A board placed at the end of the bed against which the patient can place her feet flat, and a roller towel fastened to the foot-rail for her to pull upon, will be found of great advantage. She should be instructed that when the pain comes on to hold her breath, press with her feet, and while pulling on the towel to "bear down" with all her strength. By these means the pelvis and thorax are fixed, and thus the full action of the diaphragm and abdominal muscles is obtained. Chloroform may now be given just when a pain is commencing.

If the *anterior lip of the cervix* is pushed in front of the head it should be pushed up during a pain as soon as it is diagnosed, as its presence is a delay to labour; the bruised, congested part

may prove a ready starting-place for a septic process during the puerperium.

*The bladder* must not be allowed to become over-distended; the pressure of the head on the urethra often causes difficulty, and makes it necessary to use the catheter—a new gum-elastic catheter, or a flexible rubber one, size 10, will be found more easy to introduce than a metal one. Guide the point of the catheter by passing the index finger along the anterior vaginal wall until the point of contact between the head and the symphysis is reached. The pressure on these forms of catheter is rarely sufficient to obliterate the lumen.

*Delivery of the Head and the Preservation of the Perineum.*—As soon as the head begins to distend the perineum, more energetic treatment is called for. With regard to the position of the patient there are two methods in vogue, dorsal and left lateral. The *lateral position* is most usually adopted in this country; it certainly has the advantage of allowing the operator to see more clearly what is going on. If the patient lies on her left side, at right angles to the edge of the bed, the hips coming well up to the edge of the bed, and the legs flexed at the hip and running parallel with the side of the bed, the best attitude for guarding the perineum will be obtained.

To preserve the perineum intact is very important; but if, in spite of all precautions, a laceration occurs, it must be repaired at once. The methods of doing this are described in the section, Injuries of the Generative Organs. A certain class of cases is more apt to have a ruptured perineum than others, *e.g.* elderly primiparæ, peculiarly inelastic perineums even in young primiparæ, a previously repaired perineum, or a specially long perineum. Then certain malpresentations, as a persistent occipito-posterior, are more likely to cause rupture. The administration of an anæsthetic during this stage is often advisable, by giving the physician more control over the movement of the head, and by lessening the voluntary muscular power during the pains.

The *methods* adopted for the *preservation of the perineum* are modifications of the following principles, either to keep the head as much off the perineum as possible by pushing it forward, or to apply direct support to the perineum. The *direct method* is carried out by laying the palm of the left hand on the perineum, with the concavity between the first finger and the thumb lying over the posterior end of the vulva, and then pressing the perineum upwards against the advancing head. The disadvantage of this method is that it prevents the serous exudation passing into the tissues of the perineum, by compressing it between the hand of the operator and the child's head.

Of the various methods—*indirect methods*—which act by keeping the head pushed as much forwards as possible, I have found a modification of Hohl's method give the most satisfactory results.

This consists in applying support, not to the perineum, but to the presenting part. The two essentials to its success are: that the head should remain flexed until the lowest possible point of the occiput comes to lie under the symphysis—after this point is reached extension may begin; secondly, that delivery must take place between the pains, and not during one. It is carried out as follows:—

The operator sits in such a way that when the thumb of the right hand is applied to the presenting portion of the occiput, the elbow of the right arm can rest on the operator's right thigh.

The thumb is applied to the most anterior part of the occiput, and the index and middle fingers posteriorly upon that portion of the head lying nearest to the symphysis. Steady pressure is exerted during each pain on the most anterior visible portion of the head, this preventing any strain on the fourchette or perineum—at first during the intervals between the pains, the right hand grasping the presenting part of the head; the chin is made to flex as much as possible, while the forehead and face are pulled forward in such a manner as to keep the chin at the same time in contact with the chest. As soon as the lowest possible point of the occiput comes to lie under the symphysis, the extension movement may begin.

As the pains get stronger the power required to keep the head back off the edge of the perineum is considerable, and the right thumb has frequently to be supported by the left hand. The patient at the same time should be directed not to "bear down"; the voluntary efforts are prevented by making her cry out, taking away the pulley. When the supra-orbital ridges pass the tense border of the vulva, the perineum retracts rapidly over the face and the expulsion of the head is complete. This is the point when laceration is most apt to occur if it takes place during a pain; but if between the pains the patient bears down and the doctor pushes the head forward, it is easily delivered.

The method adopted at the Rotunda Hospital is also a modification of the indirect method. It is carried out by applying the hand behind the anus and pushing the head forward.

*Rectal expression* is carried out by some; it consists in passing two fingers into the rectum when the head is distending the perineum, hooking the fingers under the chin of the child through the then recto-vaginal septum, then by pressing forwards and upwards the head can be easily delivered between the pains.

*Local applications*, such as hot fomentation or the application of vaseline or other inunctions, are in use, but are not of much practical value. The same may probably be said of *digital*

*dilatation* of the perineum before the descent of the fœtal head. It is done by several times hooking a finger over the perineum during a pain, and drawing it back towards the sacrum. If a perineal tear seems inevitable, the perineum may be slit laterally; this small operation is called *episiotomy*; it is doubtful if it is of much service. It is claimed for the operation that it prevents deep lacerations through the sphincter ani, and that by reason of the arrangement of the muscular fibres the wounds heal spontaneously. However, as it is never certain that a laceration is going to occur—and if it does take place, even through the sphincter ani, the laceration heals well if stitched up at once—the special advantage of the operation is not very clear.

As soon as the head is born, pass the finger round the child's neck to see if there are any *coils of cord round it*. If one or more coils are found, a little more cord may be pulled down and the loop passed over the child's head. If this is impossible owing to the cord being too tight, the cord must be divided and tied. The dangers of allowing the child to be born with the cord round its neck are: (1) the child may be strangled by the cord; (2) the placenta may be detached by the tension on the cord; and (3) the delivery of the shoulders is delayed. While the cord is being set free, the nurse should carefully wipe the child's eyes to remove any discharge present. So as to give more room under the symphysis the right knee may now be held up by the nurse, or a rolled-up pillow may be placed between the thighs.

If the cord is pulsating, the delivery of the child can be left to nature. As the shoulders pass out through the perineum they must be watched to prevent a laceration, and the operator should keep the left hand carefully on the abdomen. If, then, there are any signs of the cord not pulsating, the child must be delivered at once. The best way is to press on the fundus, and as the shoulders come down, in order to assist the posterior shoulder getting over the perineum, lift the child up towards the mother's abdomen, then depress the child slightly to bring the anterior shoulder out from under the symphysis. After this the rest of the body readily follows, as the larger portion has come first.

### Management of Third Stage

This stage, lasting from the delivery of the child until the birth of the placenta, should receive most careful attention in every detail, as the health of the patient during the puerperium and afterwards depends mainly on its successful management.

The indication for treatment is to *promote contraction of the uterus*. From the moment of the birth of the child the uterus must be carefully looked after, a light steady pressure being maintained by the attendant keeping the fundus of the uterus in the hollow of the left hand. This pressure must be continued for a short time after the birth of the placenta and membranes. If the child requires any special attention from the doctor, the nurse must maintain the pressure on the uterus.

Care of the Child.—The infant normally cries out as soon as it is born, but if not, the mouth and fauces should be carefully freed from all mucus, and some slight stimulation applied. A few smart slaps with the hands or a dash of cold water are usually sufficient. If, however, the child is apparently not going to begin breathing, the methods of artificial respiration to be used in cases of asphyxia neonatorum must be adopted (see article "Asphyxia," vol. i.). If the chloroform administration has lasted for a long period, there is a greater probability of the child requiring more attention in this direction.

The question of late or immediate ligature of the umbilical cord was at one time the subject of much discussion, but now it is generally considered that the cord should not be tied until it has ceased to pulsate. Experimental research shows that with late ligature of the cord the child gains a considerable amount of blood. At the first inspiration the opening up of the pulmonary circulation creates a negative pressure in the great vessels near the heart, and thus the blood is sucked in from the placenta; the uterine retraction and contraction assists in compression of the placenta. By these means the child receives about three ounces of blood (equivalent to three pints in the adult). The children in whom the late ligation of the cord is adopted are stronger and healthier than those whose cords are ligatured at the moment of birth. There is insufficient evidence in favour of the view advanced by a few writers that late tying of the cord is more frequently followed by jaundice.

*Method of Tying.*—The cord should be tied when it has ceased to pulsate. The usual plan is to tie it in two places; first, about 2 inches from the umbilicus, the second an inch or so nearer the placenta. This site for the first ligature allows ample room for retying should the ligature slip. The cord is divided between the ligatures; the second ligature is not necessary if we wait until the pulsation in the cord has ceased. If there is a second child in the uterus this second ligature is advisable, as possibly the vessels of the two placentæ communicate.

After the division of the cord, the child is wrapped in some flannel material and taken away by the nurse.

The phenomena of labour during the third stage are the occurrence of intermittent contraction with permanent retraction of the muscular fibres. This causes the placenta to be expelled

from the uterus, while the bleeding is stopped by the closure of the mouths of the vessels. Thus it is the physician's duty to promote contraction of the uterus, in order to cause the expulsion of placenta and any blood-clots, also to arrest hæmorrhage and prevent air getting into the uterus.

To promote Contraction of the Uterus.—As soon as the child is separated the patient should be turned on to her back, and a small vessel, such as a saucer or soap-dish, placed under the vulva to catch any hæmorrhage.

In order to have the uterus completely under control and to promote contractions, the palm of the physician's *left* hand should be laid over the fundus. With the ulnar border pressed downwards towards the promontory of the sacrum and the thumb lying over the anterior surface, the whole body of the uterus is thus within the grasp of the hand, and it is impossible for the cavity to become distended with blood-clot during the intervals between the contractions. A uniform pressure exerted over the fundus is better than light touching on the surface, as this is apt to set up irregular contractions. The uterus is found lying midway between the symphysis and umbilicus.

The method of separation of the placenta, and the diagnostic points showing when it is in the vagina, have been fully described (p. 167).

The Removal of the Placenta.—The placenta may be got rid of in various ways:—

1. Nature's unaided efforts.
2. Créde or Dublin method of delivery.
3. Removal of the placenta by hand.
4. Traction upon the cord.

*Nature's unaided efforts* are somewhat tedious. When the placenta has been expelled into the vagina, which usually occurs within twenty minutes of the birth of the child, it lies there, and is slowly extruded by the action of the abdominal muscles. This process frequently takes several hours, and for this reason the third stage is invariably shortened artificially.

*Créde's or the Dublin method* of delivery is certainly the best artificial means we have of delivering the placenta. If practised as soon as the child is born, that is, before the separation of the placenta, post-partum hæmorrhage is very apt to occur as the mouths of the vessels will not have had time to close. There is also some danger of portions of the placenta being left behind. If we wait from fifteen to twenty minutes the placenta will have had time to be extruded into the vagina, when expression is perfectly safe. The physician grasps the uterus firmly during one of its contractions, and then first presses backwards and downwards, and then changes the pressure to a forward movement, by which means the placenta is expelled through the vulva. As the placenta appears it should be taken hold of by the nurse and turned rapidly round and round to form the membranes into a cord, thus diminishing the chance of their tearing. The twisted membranes usually slip out at once; if there is any difficulty, wait until the uterus relaxes and they will slip out easily.

*Removal of the Placenta by Hand.*—If the placenta is still in the uterus (retained), the methods of its removal are described (see Retention of Placenta). If it is lying in the vagina, there is no advantage to be gained over the method of delivery by expression.

*Traction on the cord* is the worst of all methods of delivery. If the placenta is not detached from the uterus before the traction is effected, the central portion is pulled off the uterine wall, thus creating a vacuum into which the blood is poured from the sinuses; and if the traction is at all excessive owing to the placenta being adherent, inversion of the uterus may quite likely result. If the placenta is in the vagina, the method is safe enough, but is more apt to cause retention of membranes than the other methods.

Examination of Placenta and Membranes.—The placenta on its removal should be placed in a basin of water and submitted to a routine examination. Firstly, the maternal surface. If the maternal side is entire, it will form a continuous surface when held upwards on the two hands; but if a lobe or part of a lobe is left behind, the surface will be correspondingly irregular. The continuous edge of the amnion round the placenta should next be examined in case it should be incomplete. If a pair of lacerated vessels are seen at the placental edge, this denotes a placenta succenturiata somewhere; if this is not found outside, it must still be in the uterus. Then inspect the membranes. The sac of the amnion and chorion can be separated from one another without much difficulty, and, if split up completely, it can easily be seen if the chorion is attached all round to the placenta.

Should it appear that everything has not come away, the uterus and vagina must be explored by the hand (see Retention of Placenta).

The perineum should now be carefully examined for any tears, and if there is any injury involving more than the fourchette, it should be sewn up at once (see Injuries to Generative Organs).

### Management after Delivery

For about half an hour after delivery the hand must be kept over the fundus to prevent it becoming filled with clots. If the uterus become flabby and lose its outline, the doctor must grasp and knead the uterus firmly until a contraction is set up. This manipulation of the uterus, besides preventing hæmorrhage somewhat, relieves the severity of after-pains in multiparæ.

*Ergot* may be given to further guard against

any chance of hæmorrhage.[1] A few words here on the action and use of ergot are not out of place. The physiological action of ergot is to cause tonic contraction of the entire uterus. When given by the mouth it acts in fifteen to twenty minutes, but in three to five minutes after a hypodermic injection. When given at the proper time ergot is often of the greatest service, but if given too soon its results are most dangerous.

The uterine contractions induced by ergot differ from the normal action in being tonic in place of intermittent. Therefore it is only safe to give ergot when the uterus is empty (*i.e.* after the birth of the placenta and membranes), as then a state of tonic contraction cannot possibly do any harm; and it is a good routine method to give a drachm of ergot to all multiparæ.

If ergot, however, is given during the first stage, the tonic uterine contractions kill the child and are even apt to loosen the placenta. It is only permissible during the second stage if there is absolutely no danger of any obstruction to labour being present; this can very rarely be made out definitely.

During the third stage the tonic contractions of the uterus may cause retention of the placenta. If this should be complicated with hæmorrhage, the physician is in a very anxious position, as nothing can be done until the placenta is removed.

As soon as it is apparent that the uterus is acting properly, attention must be directed to making the *patient clean and comfortable.* All the blood should be washed off the patient thoroughly. This is best done with creolin and water, soap, and a piece of new flannel; then the soiled clothing is slipped out from underneath the patient. No douching is required unless there has been some form of interference during the second or third stage. If it is indicated, see p. 191. Then the patient should be turned on to her left side again, and the knees drawn up in order to allow of a thorough visual inspection of the perineum, in order to be certain if there is any tear. Tears of the perineum and lacerations of the vagina should be at once repaired (see "Labour, Injuries to the Generative Organs"). Lacerations usually heal quickly and completely if sutured at once, and special attention paid to the cleanliness of the parts. This consists in washing the parts gently after micturition or defæcation with a creolin solution, and the application of a dressing of dry iodoform gauze; the dressing can be held in place by the diaper. If the wound fails to unite, it is probably due to syphilis or general lowered vitality.

[1] Schafer has shown that suprarenal extract is a powerful stimulant of the muscle fibres in blood-vessels, and more recently has demonstrated a similar action on the uterine muscle. His observations suggest that this extract may be a very useful remedy in cases of post-partum hæmorrhage and other uterine conditions, where stimulation of the uterine muscle is indicated.

The binder, although not absolutely essential, is a great comfort and support to the woman, enabling her to turn on her side. The best form of binder is a long strip of firm towelling, with no shaping. The practice of placing a pad over the uterus usually results only in pushing the uterus to one or other side, and does not serve any good purpose. The binder is fixed by inserting the pins from below up, at about 1½ inches apart. The toilet is finished by applying a warm pad to the vulva.

After the patient is tidy a drink of milk or beef tea may be given. As a rule, the patient can be left safely an hour after the birth of the child.

Directions should be left with the nurse to call the medical attendant in the event of any hæmorrhage, rigor, or syncopal attack occurring. This will be further referred to under "Puerperium."

Management of unusual presentations of the child, viz., persistent occipito-posterior, face, brow, breech, and transverse. In these sections it is assumed that everything is normal except the position of the child. For the etiology, mechanism, diagnosis, and prognosis of these special lies the reader is referred to section on Diagnosis and Mechanism, p. 168.

### Occipito-Posterior

If the occiput is posterior, labour is likely to be prolonged, as the long rotation of the occiput forward to the symphysis which takes place in the majority of cases occupies some time. In a certain number, however, the occiput rotates into the hollow of the sacrum, and becomes a persistent occipito-posterior.

The management depends on whether the case is seen early or late.

1. *Early: before Rupture of the Membranes.*—If, on arriving at a case, the position be diagnosed as a third or fourth vertex, the best treatment is to turn the child round so that the back of the child lies to the front. This is done as follows:—For the third vertex position—that is, the abdomen looking forward and to the left, and the left shoulder anterior and to the front—the operator places his hands thus—the left in front of the right shoulder, and the right behind the left shoulder; by a series of gentle pushes the child can easily be turned. If the rotation is fully effected the occiput now lies anteriorly, and quickly begins to descend and press on the os, and there is no chance of it slipping back.

2. *Late: when the Head is engaged in the Pelvis.*—As there is always the possibility that the head may rotate naturally, wait about three hours. If, during this time, it is neither rotating nor advancing, some help must be rendered. This should be in the direction of imitating nature as much as possible, and by *increasing flexion* in all cases where it is not

well marked. If we can *flex* the head the occiput will descend, and then the head will rotate forward. Flexion is attempted by pushing upwards and backwards on the frontal pole of the head during a pain. This is sometimes effectual, but if not soon followed by descent of the occiput, it will be of no use. An attempt can then be made to make the occiput descend by use of the vectis, which is passed over the occiput, and with it is pulled downwards and forwards. The vectis is not much used, but the single blade of a forceps, if it has an extra sharp curve at the top, can be used. (For vectis, see "Labour, Operations.")

If the attempts to flex the head have failed, and the physician has not a forceps with a suitable curve to act as a vectis, *rotation of the head by the hand* can be tried, the attempt being made between the pains.

Pass the left hand into the vagina. Grasp the occiput and try and bring it round to the front; at the same time try and move the shoulders round. If the rotation of the shoulders is not complete, the head will slip back into its old position. This method is of special value if the accoucheur have small hands. It is a useful plan to rotate the head, hold it in its new position, and then apply forceps. This requires much less effort in traction than in trying to deliver a persistent occipito-position with forceps. The danger of over-twisting the child's neck is more theoretical than real.

*Forceps.*—This is sometimes quite successful, as after the head has descended by pulling, it rotates naturally. When this takes place the forceps must be taken off and reapplied. If, however, the occiput does not turn, a great deal of force and time is often required for the tugging, and when delivery is at last accomplished there is great danger of having a very badly lacerated perineum.

### Face Presentations

(For Mechanism, see p. 180.)

When this presentation is diagnosed sufficiently early an effort should be made to transform the face into a vertex presentation. This is only possible under the following circumstances:—

1. When the face is not fixed in the brim. If it is fixed, the movement is impossible owing to the relationship of the diameters.
2. While the membranes are unruptured.
3. When the abdominal walls are lax.
4. When there is no cause present, such as a tumour of the neck, to prevent the child's head flexing.

Before making any manipulative attempts of this kind an exact diagnosis of the presentation and position must be made. There are two methods that may be tried: first, by pressing on the face and occiput; secondly, Schatz method of pressing on the shoulders and breech. Both manœuvres are harmless and both may fail.

*Manipulative Measures.* — The first method described by Herman is to place two fingers in the vagina, and the other hand on the abdomen over the occiput. Then with the internal hand press the face upwards by pressure on the jaws and then on the forehead; while with the external hand push the occiput down into the pelvis. When the forehead is raised above the pelvic brim use both hands on the abdomen, the left hand still pressing the occiput downwards, and the right hand pressing the face upwards and forwards. The objection to this method is that it will probably rupture the membranes.

*Schatz Method.*—This requires very lax abdominal and uterine walls, so it is well to anæsthetise the patient fully.

With both hands raise the head up from the pelvis by pushing upwards the anterior shoulder

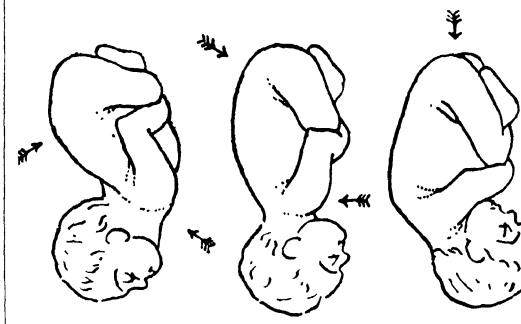


Fig. 34.—"Schatz's method" of converting face presentations into vertex presentations.

and chest of the child through the abdominal wall, then with one hand on the chest push in the direction of the child's back, while the other hand pushes the breech in the opposite direction; lastly, when the breech is directly above the pelvis push it downwards and apply a tight binder. Occasionally the flexion is not complete, and the face is transformed into a brow.

If the case is seen too late to transform it into a vertex, it must remain a face and be treated as such. The patient's friends should be warned that the labour in all probability will be long and tedious, and that the risk to the child is greater than usual.

The chief cause of delay is that the face is a bad dilator, and for want of proper support the membranes tend to rupture early. Therefore do everything to *prevent early rupture*, viz., keep the patient in bed, prevent her straining, and do not make an examination during a pain.

If the membranes rupture early and the face presses on to the os at each pain, leave the case to nature; also, if the os is fully dilated and the head is in the pelvic cavity and the chin to the front, there is seldom any need for interference. However, if the membranes are

ruptured, and the head is not coming into the os to dilate it, there must be some further complication present, probably a small pelvis or an extra large head. If the condition of the patient does not call for any active treatment, wait until the os is large enough to admit of internal version being performed. Should the patient be showing signs of exhaustion, frequent pains, rapid pulse, etc., and the dilatation being evidently delayed by absence of a dilator, put in a Champetier de Ribes' dilating bag, and, when the os is sufficiently large to allow of version, turn and bring down a leg.

In a few cases where the face is originally lying in the *mento-posterior position*, the chin rotates into the hollow of the sacrum. It is, therefore, necessary in a mento-posterior case to do everything to make extension as complete as possible, so that the chin may be inclined to rotate forwards. *Postural treatment* is most valuable in causing extension. Place the woman on the same side as that to which the fœtal back is lying; this produces an obliquity of the uterus which brings the direct intra-uterine pressure into a line impinging in front of the centre of the head. When there is a fully dilated os and the chin behind, try the postural treatment and wait two hours in the hope that the chin will rotate. If at the end of that time it has not done so, it is best to *turn the chin to the front, put on forceps, and deliver.* This is done by putting the left hand into the vagina and the right on the abdomen; grasp the face and turn it the shortest way to bring the chin to the front; at the same time press the anterior shoulder in the same direction.

If the chin cannot be rotated forward *perforation* is the only resource.

### Brow

An average-sized head cannot be delivered alive in this position. It is therefore necessary always to convert a brow presentation either into a vertex or a face. This can be done by completing flexion and producing a vertex, or by completing extension, and then dealing with the resultant face presentation as in the last section. (For mode of production and mechanism, see p. 183.)

*Before rupture of the membranes;* by the same methods as recommended for transforming a face case into a vertex. If these fail, push up the occiput so as to try and get the chin down; if these manœuvres fail, the method of procedure varies.

(*a*) *When the head is above the brim* and the os uteri partially dilated. If the pains are infrequent, and the patient not exhausted, the best method is to perform internal version and bring down a foot. If the pains, however, are frequent, and the uterus seems in a state of tonic contraction, version implies risk of rupturing the uterus, and forceps should be tried. Should these be unsuccessful, perforation must be resorted to.

(*b*) *When the head is in the pelvic cavity, but is not advancing.* As version would be dangerous to the mother, forceps must be tried if the head is small. A strong pull will most probably extract the child. If the pains are very strong, and labour has lasted some time, the head is in all probability large, and if this is the case the forceps will fail to deliver the head. Try first with the forceps, and if the head does not advance, perforate.

### Podalic Presentations

The management of pelvic presentations, whether *breech*, *knee*, or *foot*, is practically the same. (For cause and mechanism, see p. 184.) In these cases the prognosis is usually good for the mother, but an increased risk to the child. The duration of labour *averages* the same as in vertex presentation, but with a full-timed child the process occupies a longer time. The low average is accounted for by so many breech presentations occurring in premature infants. It must also be borne in mind that the predisposing factors for the malpresentation are present, and sometimes require special treatment, and imply additional risks.

The diagnosis of breech presentations should be made out entirely from abdominal palpation, and thus the possibility of rupturing the membranes during a pain is avoided. It is well to tell the patient's friends that the child is not presenting in the usual way, and that this circumstance may possibly delay labour and entail additional risk to the child.

If the case is seen early enough, *i.e.* before rupture of the membrane, and while the breech is movable, the question arises whether it would not be an advantage to *transform* the podalic into a cephalic presentation.

This point must only be decided after a full appreciation of the fact that in some conditions a podalic lie is preferable to a cephalic one. Thus in *placenta prævia* to turn and bring down a foot is the recognised treatment, and in slight cases of *pelvic contraction* delivery of the after-coming head is more easily accomplished than the head coming first.

If these indications are not present, there is no reason why the child should not be turned by external version. The best time to do this is when labour has commenced. After doing so, apply a tight binder to keep the child in its new position.

If the breech presentation is to be allowed to remain, the treatment varies in the various stages.

First Stage.—The only treatment here, as in all cases of abnormal presentation, is to *preserve the membranes* as long as possible. Keep the patient lying down to prevent undue straining,

and avoid unnecessary vaginal examinations. If the membranes rupture before complete dilatation of the cervix the labour will be prolonged, and the risk to the child's life is increased, as the breech is a very inferior dilator to the head. Further, as the breech requires less room, delay is caused in delivery of the head from want of previous complete dilatation.

SECOND STAGE.—Do not be tempted to pull down a foot or leg in the hope that it will hasten matters. Too early traction may result in the head becoming extended, or the body may come down and leave the arms extended by the side of the head, thus considerably increasing the difficulty of delivery. Leave the case to nature until the trunk is born as high up as the umbilicus. The only treatment required to this stage is to lift out the feet as the breech slips out from the perineum, so as to prevent them catching.

When the *umbilicus is born* pull down a *loop of the cord.* If this is pulsating regularly the child is all right. Pulling down the cord also prevents tension being put on the cord between the umbilicus and the part caught at the brim.

If the cord is pulsating normally, wrap the body and legs of the child in a warm cloth, so as to avoid the risk of the cold air stimulating the skin and inducing the child to respire. Usually the next pain drives the child out with the exception of the head. The important point in waiting for the pains is that the uterine contractions acting from behind keep the arms flexed upon the chest, thus making delivery more easy; if the child is pulled upon, the arms will very likely extend. However, if the cord is not pulsating, it is evident that in order to save the child the delivery must be hastened as much as possible, the life of the child now depending upon the skill and quickness of the practitioner. If the child has to be pulled upon, get the nurse to keep up firm and steady pressure over the fundus during the manipulations; this helps to keep the arms on the chest and the head flexed. Seize hold of the child round the pelvis, the thumbs lying parallel to each other over the sacrum (this avoids injuring the viscera from pressure by the finger), then pull downwards and forwards until the scapulæ are reached.

*Delivery of the Arms.*—Let the nurse draw the body of the child forward over the mother's abdomen. Then pass up the entire hand into the vagina, and along the front of the child's chest, to feel for the arms. If they are still flexed, pull them down by putting a finger first into one elbow and then into the other; this is quite easily done. If the arms are extended, turn the child so that one arm lies to the front and back. It is best to bring down the posterior arm first, as there is most room in the hollow of the sacrum. Now, with the body of the child held well forward over the mother's abdomen, pass the hand into the vagina, so that its palmar surface rests on the back of the child; then place the first and second fingers on the humerus, and slip them up until the elbow is reached, then with the tips of the fingers press the elbow across the child's face. The anterior arm can then be delivered as an anterior arm, or the body of the child can be rotated so that the anterior arm comes to lie in the hollow of the sacrum, and is delivered in the same way as the posterior arm.

Occasionally the arm is extended and the elbow bent, and the forearm lies behind the child's back. This is known as *dorsal displacement of the arm.* This displacement in a full-time fœtus and a normally-sized child arrests the advance of the head, the displaced arm becoming caught on the brim of the pelvis.

The position of the arm will be discovered when the hand is passed in to deliver the arms. The arm can be set free by turning the body and pressing the vertex towards the free arm; if this fails, the arm may have to be fractured before it can be brought down.

*Delivery of the Head.*—Unless the head is expelled by the same pain as trunk and

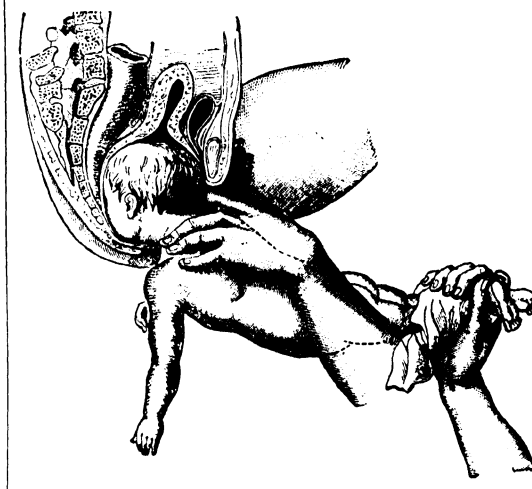


FIG. 35.—The "Prague method" of extracting the head.

shoulders, assistance is called for. This must always be effected with the greatest rapidity. As a rule, the head must be delivered within five minutes of the birth of the arms, or the child will be asphyxiated. The danger to the child from any delay is due to the following causes:—

1. The pressure on the cord between the head and the bony pelvis will stop the circulation.

2. The cold air stimulating the body of the child, causes premature respiration, and mucus or meconium is sucked into the lungs.

3. The placenta is very probably being detached.

The method of delivery depends upon whether

the head is delayed in the pelvic cavity or arrested above the brim. When *the head is arrested in the pelvic cavity* the "Prague method" is the simplest and quickest method. With the patient lying preferably on her back, the left hand is passed into the vagina and passed up over the back of the child, and the first and second fingers are hooked over the clavicles (Fig. 35). The limbs of the child are wrapped in a cloth and grasped by the right hand. Carry the legs forward over the mother's abdomen as far as possible, and by the joint pulling of the two hands pull the neck and shoulders forward. The head is thus made to roll out flexed from behind the perineum. The left hand pulls the shoulders towards the anterior parts, and the pressure of the symphysis at the same time presses on the occiput. Thus the head is flexed and is in the best position for delivery.

If the *head is arrested above the brim*, delivery can be effected by a manœuvre which combines jaw traction and pulling on the shoulder, or by forceps.

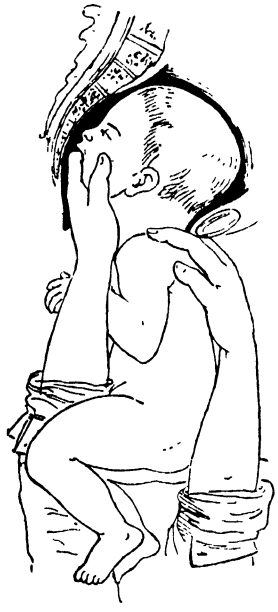


FIG. 36. — Method of jaw and shoulder traction for delivery of the head. (After Chailly Honoré.)

The former method is the best, as there need be no unnecessary delay. *Jaw traction* is especially suited for cases of arrest of the head above the brim, but it can also be used if the delay is in the pelvic cavity; it is the most powerful as well as the quickest method of delivery. The practitioner, standing on the patient's right side, passes the left hand into the vagina in such a way that the child rides upon the arm (Fig. 36). The two fingers are passed into the child's mouth as far back as possible (this is to lessen the risk of fracture of the jaw).

The right hand is placed over the shoulders. The fingers on the jaw prevent the head extending, whilst delivery is accomplished by traction on the shoulders and jaw. If the resulting flexion of the head is not sufficient, it can be increased by placing the first and third fingers of the left hand over the clavicles, and the second finger pressing the occiput forwards. Pressure on the abdomen is of assistance in hurrying up labour.

When the head is above the brim the traction should be first made backwards and downwards, and when in the pelvic cavity the direction is changed to forwards, at the same time carrying the body of the child well over the mother's abdomen.

*Forceps to the after-coming head* are certainly able to deliver the child, but they are slow. If the combined jaw and shoulder traction does not succeed, do not hesitate to at once apply forceps. Forceps should always be ready for use in cases where there is any possibility of difficulty in the birth of the head.

If forceps are used they must be locked under the body of the child, and traction applied in the axis of the pelvis.

If the pulsations in the cord have ceased, and there is difficulty in extracting the head, it is best to use the perforator (for method, see "Labour, Operations"), as it is less apt to damage the maternal parts than prolonged pulling.

DIFFICULT BREECH CASES. — In a certain number of cases accidental complications arise which make it necessary to assist the delivery of the breech.

In cases with a very large child, or a very small pelvis, or if the pains are so feeble there is great delay in the labour, and assistance is necessary both for the sake of the mother, to save her from exhaustion due to want of food, prolonged pain, and anxiety, and for the child, whose life may be endangered from pressure on the cord. Also, if there is prolapse of a loop of cord during the second stage of labour, the cord will have lost the protection the bent-up legs would have given it, and, therefore, it is as well to bring down a leg so that delivery can be quickly accomplished if the pulsations are evidently becoming arrested. It should never be forgotten that no interference is justifiable before dilatation of the os.

*Digital Pulling.*—In cases where delay is due simply to weakness of the pains, steady pulling will accomplish delivery. Pass up the right forefinger over the anterior groin between the abdomen and thigh, and during a pain pull on the anterior groin. Whenever the breech is low enough, pass in the left hand and put two fingers into the posterior groin; then whenever a pain comes, pull as strongly as possible. As the breech emerges, pull most on the posterior hip, as it has the farthest way to come. This movement is very useful, but is very tiring to the operator's fingers (often setting up cramp).

If the pelvis is small or the child too big, there are several methods recommended to assist delivery, *e.g.* bringing down a leg, or to deliver the breech by traction with the fillet, blunt hook, or forceps.

*Method of Bringing down a Leg.*—It is best to anæsthetise the patient. Then pass into the vagina the hand that will lie most easily flat on

the child's abdomen, placing the other hand over the fundus of the uterus. The anterior leg will be found the most convenient to bring down. When the fingers have reached the knee partial flexion is induced by pressing it outwards and backwards, and pass the hand up and seize the ankle with the first finger and thumb. By pulling the ankle downwards the knee is completely flexed; then by further pulling on the ankle the thigh is extended, and thus brought out of the uterus. Be careful only to pull on the ankle. If this plan is to be adopted, it should be done early before the uterus has contracted tightly on the child. If the second stage has lasted some time and the uterus is tightly contracted over its contents, it is better to bring down the breech.

Traction on the breech *by the fillet* is carried out by passing over one or both groins a silk pocket handkerchief or a bit of strong banding, which has previously been boiled and then put into an antiseptic solution for a short time. The end of the loop is seized and pulled upon; the traction thus supplied is, as a rule, very successful.

*The blunt hook* is the easiest way of delivery in a really difficult case; but unless used with very great care, it is apt to injure the child's genitals or lacerate the femoral vessels. If the child is dead the blunt hook can be used without fear, and will quickly deliver (see "Labour, Operations").

Forceps may be applied to the breech, but the objection is that the ordinary shape of forceps is not suited to shape, and is very apt to injure the child. Special forceps have been made, but they are required so seldom that it is better to be prepared to deliver by simpler means.

*Delivery of the Head in Cases with the Face Anterior.*—The mechanism of the delivery is described on p. 186. It is often impossible to deliver the child without extensive lacerations. *If the head is above the brim*, pass the hand up into the hollow of the sacrum behind the head, and then move the hand round until it reaches the mouth. This turns the head to the side of the pelvis, which is the position in which the head can best pass the brim. Should this fail, forceps must then be tried; and if then unsuccessful, resort must be made to perforation. If the *head is in the pelvic cavity*, the delivery can best be assisted by helping the head to flex further on the chest, and the chin and the rest of the face to glide under the arch. This is done by drawing the woman to the edge of the bed, depressing the body of the child, and carrying it well backwards; this draws the chin from behind the symphysis. If the face sticks put the fingers far back into the child's mouth, so that the face comes gradually out from behind the pubis, followed by the forehead and occiput.

Injuries to the Child in Breech Deliveries.—In breech deliveries where it has been necessary to assist nature in expelling the child, various injuries to almost all the different tissues and organs of the body have been described.

*Laceration and bruising* may occur in the muscles of the neck and back; the best known one is hæmatoma of the *sterno-mastoid*, caused by hæmorrhage into the sheath of the muscle. The tumour formed is usually about the size of a pigeon's egg; it disappears gradually in about six months, but is sometimes followed by permanent shortening, which is one of the causes of torticollis.

*Hæmorrhage* may also occur in the abdomen and cranium. The former is due to injury to the liver and suprarenals, which can best be avoided by only pulling on the trunk when grasping the pelvic girdle.

*Meningeal hæmorrhage* and also hæmorrhage into the brain substance itself are fairly common, and may occur quite independently of fracture of the skull bones. The late results of this injury are:—

*The genital organs* are very liable to injury if the blunt hook is used. Spiegelberg records a case where the penis and scrotum of a child were completely destroyed.

*Paralysis of the brachial plexus* has followed hard pulling on the shoulders in order to deliver the head quickly. This occurs without an accompanying fracture of the clavicle, and lasts from a few days to weeks; recovery invariably follows.

*The spinal cord* can be torn across in the cervical regions; this is especially apt to occur in delivery of the head by the Prague method, the whole force being transmitted through the neck.

Almost all the *bones can be fractured.* In the skull we meet with the basilar portion of the occipital being separated from the squamous. The parietal bones may be fractured. The vertebral column can be torn across; this, as in cases when the spinal cord in the cervical region is injured, occurs in cases that have been delivered by Prague's method.

Fractures of the clavicle and humerus occur, that of the clavicle being fairly common. Fracture of the lower limbs sometimes is met with, but is not usual. (For methods of treatment, see "New-born Child, Injuries of.")

By putting the finger in the *mouth* various injuries have occurred; *e.g.* dislocation of the jaw and detachment of the condylar epiphyses may occur, separation of the two halves of the lower jaw at the symphysis, or the jaw may be dislocated, or the condylar epiphyses may be detached. The soft tissues in the floor of the mouth may also be torn.

### Transverse Presentation

A transverse presentation so rarely rectifies itself, and the results of allowing such a pre-

sensation to persist are so disastrous, that early treatment is a necessity. (For causes, etc., see p. 187.)

The various means at our disposal are:—

1. Postural treatment.
2. External cephalic version.
3. Internal or bipolar podalic version.
4. Embryotomy.

1. Postural Treatment.—In cases of slight obliquity of the uterus with unruptured membranes this method is sometimes successful. The rationale of the method lies in the fact that the breech and lower limbs of the fœtus are heavier than the head, and therefore the breech tends to gravitate towards the lowest point at the same time that the head rises. Thus, with a head lying over the *left* iliac fossa the patient is placed on her left side, and the breech tends to fall towards the left side—the head then rising comes to lie over the brim. This plan of treatment is only possible in a very small proportion of cases in which the practitioner sees the case sufficiently early.

2. External Cephalic Version.—This operation also requires unruptured membranes and labour in an early stage. The fœtus is turned by external version until the head comes over the brim, when the membranes are ruptured. The head should be held with the hand, or by a tight abdominal binder, over the brim until it fixes. After version has been accomplished the child is very apt to slip back into the former position. It is, however, quite worth the trial when possible, as it gives the child the best chance of life.

3. Internal Podalic Version is indicated when external cephalic version has failed, or cannot be performed. The version should be performed as soon as possible and a leg drawn down; the case can then usually be allowed to finish as in a breech presentation.

There are, however, some contraindications to the performance of internal version, namely, if a considerable *portion of the child is driven out of the uterus*, and when the uterus is in a state of *tonic contraction* with Bandl's ring 2½ inches above the symphysis. (For methods of performing version, see article "Labour, Operations.")

4. Embryotomy.—It may be necessary to do this under the following conditions:—*If podalic version is contraindicated*, for the reasons mentioned above embryotomy must be performed. The main symptoms showing that the uterus is in a state of tonic contraction are—persistent pains, and the uterus continuously remaining hard instead of relaxing and contracting; the presence of Bandl's ring about 2 to 2½ inches above the symphysis; increased pulse-rate; drawn, anxious expression; and the vagina in a hot and dry state. Further, if the *podalic version is difficult, and if there is reason to believe that the fœtus is dead* (*i.e.* absence of fœtal heart sounds, and the cessation of fœtal movements; also, on feeling a loop of cord, the complete absence of pulsation), the embryotomy is the best chance to the mother; *or if podalic version is impossible* from too much of the fœtus having been driven out of the uterus.

The best method of embryotomy to adopt in these cases is to pull down the arm and decapitate. (The operation of decapitation is described in article "Labour, Operations.")

The choice of method to be adopted at the different stages is now briefly given.

(*a*) *The membranes unruptured* and the *os not sufficiently dilated to admit two fingers.*

At this stage do not interfere further than attempting to rectify the position by postural treatment, or by performing external cephalic version. Any further attempts would only rupture the membranes unduly early.

(*b*) *The membranes unruptured, the os sufficiently dilated to admit two fingers easily, but not fully dilated.*

Here, again, postural treatment and external cephalic version should be tried. If this fails, there are two courses open to the practitioner—either to remain beside the patient prepared to turn and artificially dilate the instant the membranes rupture, or, if it is impossible to stay beside the patient for hours, the best practice is to bring down a leg as soon as the size of the os admits of it. The former method affords the best chance for the child's life.

(*c*) *The membranes are ruptured, the os is not sufficiently dilated to allow of internal version* (*i.e.* it will not admit two fingers easily). In these cases dilate the cervix artificially by means of a Champetier de Ribes' dilating bag. When the os is sufficiently dilated to allow of delivery, perform internal podalic version.

(*d*) *The membranes are ruptured and the os fully dilated*, the uterus being moulded to the shape of the child. If the pains are not continuous, the uterus relaxing, and the child movable between the pains, listen for the fœtal heart sounds; if the child is found to be alive, bring down a foot and deliver by internal version. Should the uterus show symptoms of tonic contraction, or there be unmistakable signs of death of the fœtus, the best method is to pull down an arm and decapitate.

## Labour in Multiple Pregnancy

Introductory . . . . . 204
The Anomalous Features of—Maternal Risks . . . . . 205
Infantile Risks . . . . . 206
Management of . . . . . 206
Complicated Cases . . . . . 207

Introductory.—Labour in multiple pregnancy is, as a rule, comparatively easy, and yet the results to mothers and children are less favourable than in single births. It is difficult to

judge of the reason of this from the occasional occurrence of twins in general practice; it is only when grouped together in sufficiently large numbers and compared with ordinary labour that one can realise wherein labour with twins differs from single cases. Unfortunately, statistics have generally been compiled in a form which lessens their value for the purpose in view. Often the number of children stated as born dead includes, undistinguishably, the non-viable and decomposing, together with those lost in the birth; the first and second born are grouped together, instead of separately; and the length of the interval between the births, if given at all, is discussed apart from the results to the children.

The data that will be here quoted, when not otherwise stated, are taken from the Reports of the Dublin Lying-in Hospital, published by Collins, by Hardie and M'Clintock, and by Johnstone and Sinclair. The number of women confined of twins was 568. The value of the statistics lies in the fact that the sex and presentation of each child, the duration of the labour and of the interval, the number of the pregnancy and the results to mother and children, are given in the majority of the cases. Excluding all children reported as "putrid" and those born before a viable age, there were 538 cases available for analysis.

ANOMALIES.—The course of labour in multiple pregnancy is much the same as in single births, but there are certain anomalous features which have important bearing regarding management. Owing to a greater tendency to pathological conditions, the labour is liable to be *more or less premature*. The ratio of premature births has been estimated at as much as 26·5 per cent (Reuss).

*The relative frequency of the various presentations* is different from that found in single births; breech and transverse presentation are more frequent, yet this, it will appear, is favourable rather than otherwise.

Both fœtuses present by the head in about 50 per cent; one head and the other breech in 30 per cent; both by the breech in 9 per cent. More rarely a head or a breech presentation may be associated with a transverse lie, the latter generally in the second child; both fœtuses lying transverse is very rare (Spiegelberg).

*Duration of Labour.* — Multiple pregnancy rarely gives rise to difficulty in labour, and in the vast majority of cases the natural powers are sufficient to complete delivery. Conditions that retard the progress of labour are, however, more frequently present. The delay occurs before the birth of the first child, and chiefly, though not entirely, in the first stage.

According to the Dublin data, in SINGLE pregnancies 90 per cent of the cases were completed within twelve hours, and 2 per cent only were protracted beyond twenty-four hours; whilst with TWINS, 90 per cent of deliveries were not reached till fully twenty hours, and nearly 8 per cent were protracted over twenty-four hours.

The cause of the delay is usually ascribed to "inertia due to over-distension"—an expression often used inaccurately. The over-distension which causes delay in the first stage is not so much due to the increased bulk of the fœtal bodies as to the relatively larger amount of liquor amnii commonly met with in twin pregnancies; and the delay is not owing to any inherent weakness of the uterine muscle, but to imperfect transmission of the force by the uterine contents. Owing to the relative excess of liquor amnii the action of the bag of waters in dilating the os is liable to be defective,—a condition which is not peculiar to twin cases, but is frequently met with therein. The prolonged ineffectual action so caused is liable to produce secondary "inertia" of the uterus after the birth of the first child, leading to delay in the expulsion of the second child and difficulty in the third stage of labour.

*The risks to both mothers and children are increased in multiple pregnancy.* The material death-rate has been in some cases fully double, and the infant mortality two and a half times greater than in single pregnancies.

MATERNAL RISKS.—There is one cause that has a marked influence on the unfavourable results, which must be mentioned although it is apart from labour—it is, that in multiple pregnancy there is an *increased tendency to eclampsia.*

In 568 twin pregnancies there were 7 cases of convulsions, or 1 in 81 cases, whereas in ordinary pregnancies the ratio was 1 in 363 cases. The difference is not due to a relative greater number of primiparæ in one series more than the other, for the proportion, 30 per cent, was the same in both.

The great risk to the mother undoubtedly is *increased liability to hæmorrhage* after the birth of the children—dangerous in itself, and predisposing to complications in the puerperium.

The statistics of twin as compared with single births show that (1) post-partum hæmorrhage was five times more frequent; (2) the placentæ were adherent twice as frequently; (3) retention of the placenta from all causes, necessitating manual interference, occurred six times more frequently.

Various conditions here combine to increase the liability to hæmorrhage; there are the larger area of the placental site and an increased difficulty in the separation and expulsion of the secundines, due to the larger placental bulk to be expelled, and an apparent greater tendency to adhesion. To these must be added the increased risk of uterine fatigue when the labour has been retarded, causing slow and weak contraction and imperfect retraction.

INFANTILE RISKS.—It has long been recognised that labour in multiple pregnancy is specially unfavourable to the children, but more detailed information on certain points than is generally supplied by writers is necessary to decide the question of the proper rules of treatment. Here the Dublin data are of great value.

1. *Length of Interval between the Births.*—In 262 cases the interval was stated. Of these the second child was born within fifteen minutes in 46·5 per cent; in the second quarter of an hour, 30·2 per cent—giving 76·7 per cent in the first half hour. In the second half hour 9·9 per cent, and from one to twenty hours in 13·3 per cent.

2. *The Mortality in Relation to the Interval.*—Of those born within the first half hour, 1 in 20 was still-born; of those in the second half hour, 1 in 5; over one hour, 1 in 3·5; thus bringing out the important fact that *the mortality of the second half hour was four times greater than that of the first half hour.*

3. *Influence of the Presentation on the Mortality.*—In the FIRST BORN of twins the mortality of *head* presentations was higher, that of *breech and footling* distinctly less than in the same presentations in single births.

In the SECOND BORN, *head* presentations were nearly twice as fatal as in the first child—11 per cent, as compared with 6 per cent. In *breech* presentations 2·5 per cent only were lost. Of the children that lay *transverse*, and were, consequently, turned, and of those that originally presented by the feet (132 in number), *all were born alive.* The result may be stated in another form. Of the still births, 90·5 per cent presented by the *head*, 9·5 by the *breech*; whilst among the *footling and those that were turned*, there was not a child lost that was alive when the treatment began.

4. *Total Infant Mortality.*—Exclusive of non-viable and macerated children, the infant mortality in *twin cases* was 7·3 per cent, as compared with 2·7 per cent in *single births.* Of the *first* children 6·8 per cent were still-born, of the *second* 7·8 per cent.

Prematurity and feebler development may account for part of this high mortality, but it does not explain the higher rate of the second born as compared with the first; nor the anomalous results regarding presentation, how *the more frequent occurrence of "abnormal presentations" tends to diminish instead of increase the mortality.*

MANAGEMENT OF THE LABOUR.—The above-noted facts show the increased need of supervision, and the direction in which skilled assistance may with advantage be extended. The presence of a second child is, in general, unknown till after the birth of the first. Up to this point the management is the same as in ordinary labour; but, thereafter, so anomalous are the conditions and imminent the dangers, that one can no more rely implicitly on the natural powers for the safe delivery of the child, than in ordinary labour we trust to nature alone to expel the placenta. There is the same need of supervision, the like dangers in the one case as in the other. Instead of waiting half an hour, as text-books still recommend, before rupturing the membranes, the delivery of the child should be completed within that time.

After the first child is born, the unusual size of the uterus gives indication of the presence of a second. The necessary attention having been paid to the first born, and without any intimation to the patient of the state of affairs, a vaginal examination should be made to complete the diagnosis and ascertain the relationship of the fœtus to the uterus. The amniotic sac of the second child may be found ruptured, but usually it is still entire. Dilatation being already complete, the function of the bag of waters is gone, and nothing is to be gained by waiting for spontaneous rupture. Without withdrawing the examining hand, or waiting for a pain, the membranes should be broken, and, still holding back the waters, the necessary steps taken to secure command of the delivery. If the child lies transverse, it must be turned; if the breech presents, it is an advantage to bring down one leg; if it is a head presentation, two courses are open—either to leave it to the natural powers, and trust to the forceps should delay occur, which is very apt to happen, or the child may be turned and brought by the feet. In view of the unfavourable results given above, where delivery was left to nature because of the presumed safety of head presentations, and, on the other hand, the wholly favourable termination when brought by the feet, there can be no doubt that version is the better course. Under the conditions it is easy to perform, and safer to both mother and child.

Having got command of the delivery there is now no need of precipitate action. With the left hand steadily on guard over the fundus, intermitting traction can be made and the child cautiously withdrawn, even in the absence of uterine contractions, and this without fear of predisposing to hæmorrhage, for retraction is still active though contraction be absent.

From what has been said above regarding the *third stage* of labour in twin cases and the increased risk of hæmorrhage, special attention at this time is necessary, but the principles of treatment are the same as in ordinary confinements. Greater difficulty and, consequently, longer time may be required to complete the process of separation and expulsion. In the absence of hæmorrhage this may, within normal limits, be safely allowed. There is more danger in precipitating this stage than in facilitating the delivery of the second child. At the same

time undue delay may increase the difficulty; for in a partially emptied uterus delay tends to irregular action, and an unequal degree of retraction of the uterine walls, thus increasing the risk of post-partum hæmorrhage. The placentæ are usually expelled together, but when developed separately and occupying distant sites, the placenta of the first born may come away before the birth of the second child.

The same thing may occur where each fœtus occupies the separate halves of a double uterus; and so also may be explained the rare occurrence of one fœtus being retained *in utero*, it may be for weeks, after the birth of the other.

After the birth of a second child, the possibility of a third or more must be kept in mind.

Complicated Twin Cases.—Under various conditions, fortunately rare, real difficulties may be experienced. These arise where both fœtuses occupy a single amniotic sac, or where the membranes of the farther child rupture before the birth of the nearer.

1. *Different parts of each child may simultaneously engage in the brim*, as the head or breech of one and the feet of the other, or a foot of each child. Care in ascertaining the relationship of the several parts is essential; with accurate diagnosis difficulty can be avoided, by operating on one child at a time; with a head unable to enter the brim, turning should be tried.

2. Interlocked Twins.—Two or more varieties have been described.

Where *both fœtuses present by the head*, the difficulty arises when the second head engages in the brim or has passed into the pelvic cavity along with the neck and thorax of the first child. In the other variety, *the first is breech, the second head.* When the trunk of the first child presenting by the breech or feet is born, the progress of the after-coming head is obstructed by the head of the second child having been pressed in before it. Similar interlocking may occur where the second child lies transverse. The difficulty in delivery under such circumstances will, in all probability, be experienced before the operator is aware of the presence of a second child. When recognised, the actual relations of the two bodies should always be carefully examined by the external as well as internal methods; and it should always be remembered that in attempts at rectification, external pressure in a proper direction by an assistant will facilitate the process. The patient should be deeply chloroformed to check, as far as possible, uterine action; for contractions but aggravate the conditions, and waiting to see what nature may accomplish will increase the difficulty by allowing an increasing amount of uterine retraction.

An effort should first be made by combined internal and external pressure to raise the second head or other obstructing part above the brim, and, if successful, it must be kept there by steady external pressure, whilst traction by forceps or otherwise is made on the first child. Failing this, the two heads in succession may be extracted by the forceps; the small size of the heads usually admits of this being done. If too large for the size of the pelvis, decapitation may be necessary. Under such circumstances, as the child that first presents is the most likely to be lost, it is better to sacrifice it, in the hope of being able to save the second child, which should then be extracted by forceps without waiting.

3. United Twins (for varieties, see "Teratology").—It is surprising how frequently the delivery of conjoined twins has been accomplished by the natural powers—85 times in 150 cases (Hohl and Playfair). The presentation is always the same in the two fœtuses. By the feet is the more favourable; therefore, where possible, it is well to turn in head cases, and if breech, to bring down all four feet. The diagnosis, however, can rarely be made till the labour has considerably advanced. When, however, the cause of the difficulty is investigated and two heads are discovered, turning should, if possible, be adopted. In delivery by the natural powers a process of spontaneous evolution has been observed—one head and shoulders are born, then the corresponding trunk and limbs; the lower portion of the other follows, and, lastly, its shoulders and head. It is well, therefore, carefully to observe the mechanism that is taking place, and aid as far as possible. In some cases evisceration may be necessary.

## B. PATHOLOGICAL SECTION

### Precipitate and Prolonged Labour

General View of Labour . . . 208
*Precipitate Labour* . . . . 208
Prolonged Labour . . . . 208
A. *Primary Uterine Inertia* . . 209
B. *Secondary Uterine Inertia* . . 209
C. *Premature Uterine Retraction* . 210
D. *Obstructed Labour* . . . 210
Pelvic Deformities—
*How the Shape of the Pelvis is produced* . . . . . 212
The Common Kinds of Contracted Pelvis . . . . . 213
*The Flat Pelvis* . . . . . 214
*The Small Round Pelvis* . . . 214
*The Small Flat Pelvis* . . . 214
*Rickety Deformities of the Pelvis: the Flat Rickety Pelvis, the Small Flat Rickety Pelvis, the Scoliorachitic Pelvis* . . . . 215
The Mechanism of Labour with Contracted Pelvis . . . . . 217
Labour with the Flat Pelvis . . 217
Labour with the Small Round Pelvis. 219

THE RARE FORMS OF CONTRACTED PELVIS 220
*The Funnel-shaped Pelvis* . . 221
*The Oblique Pelvis of Naegelé* . . 221
*The Transversely-contracted Pelvis of Robert* . . . . . 222
*The Kyphotic Pelvis* . . . 222
*The Kypho-scolio-rachitic Pelvis* . 224
*The Osteomalacic Pelvis* . . . 225
*The Pseudo-osteomalacic Rickety Pelvis* 225
*Spondylolisthesis* . . . . 226
*Spondylizema* . . . . . 227
*Effect of Fracture and of Hip-Disease on the Pelvis* . . . . 228
THE DIAGNOSIS OF CONTRACTED PELVIS . 228
PELVIMETRY . . . . . 228
I. *External* . . . . . 228
II. *Internal* . . . . . 229
THE TREATMENT OF LABOUR WITH CONTRACTED PELVIS . . . . . 232
*Abortion* . . . . . 232
*Prevention of Pregnancy* . . . 232
*Cæsarean Section* . . . . 233
*Cephalotripsy* . . . . . 233
*Premature Labour* . . . . 233
*Symphysiotomy* . . . . 233
*Forceps* . . . . . 234
*Turning* . . . . . 234
SLOW DILATATION OF THE OS UTERI . 235
*Cicatricial Contraction of Os* . . 236
*Congenital Smallness of Os* . . 236
*Labour with Cancer of Cervix* . . 236
„ *Ovarian Tumour* . . 237
„ *Uterine Fibroids* . . 237
„ *Tumours of Pelvic Bones* 238

LABOUR is a mechanical process, which consists in the forcing open of the genital canal to a size large enough to let the child pass. The genital canal is in the bony pelvis; but with a child of not more, and a pelvis of not less than average size, the child can pass without resistance from the pelvic bones, the only obstacle to its birth being the muscular and fibrous tissues of the pelvic floor.

The average duration of first labours is about eighteen hours. The first stage consists in the dilatation of the os uteri. This opening is bounded by thick fibro-muscular tissue, and has to be enlarged from about the size of a quill to a diameter of about four inches. Its expansion takes about eighteen hours. The second stage consists in the dilatation of the vagina and vulva. This part of the genital canal is larger and more distensible than the os uteri; its dilatation, therefore, only takes about two hours. Its narrowest part is the vaginal orifice, and here the dilatation is always completed by tearing. When the canal has been opened up in former labours, it dilates more quickly; in labours not the first the dilatation of the os uteri takes about eleven hours, and of the vagina and vulva about half an hour.

The foregoing figures are averages. The time occupied in any individual case depends upon three factors, which Alex. Simpson has happily named the "Powers, the Passenger, and the Passage": first, the strength of the pains, and the down-bearing efforts with which the mother accompanies them; second, the size of the child, which conditions the amount of dilatation required; third, the dilatability of the parts.

If the child is very large, or if the pelvis is small, there may be resistance not only from the soft parts but from the bones, so that the child can only pass by altering its position in respect to the pelvic bones, and by alteration of the shape and size of its head by moulding.

From alterations in the factors which have been specified, labour may be either very quick or very prolonged. Very quick labour is called "precipitate" labour.

### PRECIPITATE LABOUR

Precipitate labour implies that the child is not large in relation to the pelvis. The labour may be quick (*a*) because the soft parts easily dilate, the powers being either normal or unusually vigorous. The only harm that comes from this kind of precipitate labour is such as follows from the child being expelled before the mother expects it. It may be driven out while the mother is at the water-closet, or in a vehicle, or standing. In the latter case, the sudden pull upon the umbilical cord as the child drops often tears it through close to the umbilicus. The muscular fibres of the torn arteries usually close them, so that they bleed not. If the cord is too tough to tear, and not long enough to let the child lie on the floor, the pull upon the placenta may, after uterine contraction has passed off, invert the uterus. This is the sole danger arising from this kind of precipitate labour. Labour may be precipitate (*b*) because the powers are excessive; either the uterine action is exceptionally strong, or the mother's down-bearing efforts are excessive. In either case the effect may be that the child is forced through the genital passage without time being given for this passage to dilate. The result is, that the parts are torn instead of stretched open, and bad lacerations of the cervix uteri, vagina, and perineum are the result; the last named being the most important. Cases have been recorded in which rupture of the sternum, subcutaneous emphysema, and cyanosis have been the result of the mother's excessive straining.

The *treatment of precipitate labour* consists in two things: first, to keep the mother recumbent; second, to abolish excessive down-bearing effort by the administration of chloroform. In some cases, as, for instance, when valvular disease of the heart is present, the latter measure is of high importance.

### PROLONGED LABOUR

Prolonged labour may result (1) from weakness of the powers; (2) from large size of the

passenger; (3) from anomalies of the passage—(*a*) of the bones, (*b*) of the soft parts.

(1) Weakness of the Powers.—We know hardly anything of the conditions which determine the strength and rapidity of uterine action during labour. We have no data from which, when consulted by a pregnant woman, we can predict that, other conditions being normal, her labour will be quick or slow. Uterine action depends not upon the general health: women in the last stage of phthisis have been known to have quick deliveries; and lingering labour has been observed in women of robust health and powerful build.

### Uterine Inertia

There are three forms of weakness of pains: A. *Primary Uterine Inertia.*—This means that the uterine contractions are infrequent, short, and weak. As Dakin puts it, "The process is leisurely." We know almost nothing of the causes of primary uterine inertia. It is annoying to the accoucheur, because it wastes his time; wearisome to the patient's friends; and tiresome to the patient herself, because she has to wait so long for her baby; but it is attended with no danger and no additional suffering. All the treatment wanted is time. The chief danger is lest the accoucheur's impatience should overmaster his judgment, and make him set about premature forcible delivery. There are some conditions which help to produce it. *Too much liquor amnii*, by overstretching the uterus, will weaken it, and thus cause weakness of pains. *Too close adhesion of the membranes* to the uterus sometimes prevents the bag of membranes from moving on as it should do, pressing into, and dilating the circle of the os. This will cause labour to be slow, for the stimulus of the pressure upon the os uteri which should provoke reflex uterine contractions is absent. In such a case, if the finger is inserted, and swept round the lower segment of the uterus, so as to separate the membranes as far as possible, the bag of membranes will be enabled to move on, and to press into the os uteri; and more frequent uterine contractions will follow as a reflex effect. To this cause is due the weakness of pains so often present in cases of placenta prævia: the placenta being attached round the os uteri is separated with more difficulty than the membranes, and hence more slowly protrudes into the os, and less effectively stimulates uterine contraction. The artificial separation of as much as possible of the placenta when it is prævia, has long been recognised as good practice, and it acts in the same way as the artificial separation of the membranes. The strength or frequency of uterine contractions cannot be influenced by *the will*, although the uterine action may be helped during the second stage of labour by the abdominal muscles. The action of the uterus is influenced by *emotion*; the entrance of the accoucheur often, to use women's phrase, "frightens away the pains." *Fulness of bladder and rectum* are commonly assigned as causes of uterine inertia, though it is difficult to explain how. If either viscus is full it should be emptied, by catheter or enema if the patient cannot relieve herself. The bladder is usually drawn up out of the pelvis into the abdomen during the second stage of labour; but if prolapse is present, the bladder may so sink that when full it obstructs the progress of the head, and the head may then prevent the bladder from being drawn up. If the rectum is allowed to continue full, the descending head will have to squeeze out the fæces before it—a process which delays delivery, and is annoying to the accoucheur.

B. *Secondary Uterine Inertia:* also called "*uterine exhaustion*" or "*temporary passiveness.*"—This means, that after uterine contractions have for a time recurred with average frequency and been of average strength, they get less and less frequent and usually also less and less vigorous. The patient may go for hours without a pain. The progress of the labour is during this time almost suspended: but some uterine retraction may go on, though pain is absent. If nothing is done, the patient will go to sleep: and by sleep nervous energy will be recuperated, and then uterine action will recommence with frequency and vigour. This condition is free from danger, excepting such as may result from the impatience of the accoucheur. If he will not wait for the return of uterine action, but drags the child out while the uterus is passive, post-partum hæmorrhage is likely to follow. This is the explanation of the well-known fact that there are some medical men in whose practice post-partum hæmorrhage is common, while others hardly ever meet with it. Those who get hæmorrhage are those who drag the child away while the uterus is not acting. It is true that delivery in the absence of a pain is not invariably followed by hæmorrhage; this is because uterine action is essentially intermittent, so that a long interval without a pain does not always mean that uterine exhaustion is present. Towards the end of such an interval uterine contractility may have returned, and if then the patient is artificially delivered, the stimulus of the accoucheur's manipulations may provoke uterine contraction. But forced delivery, while the uterus is exhausted, will certainly be followed by dangerous hæmorrhage in the third stage. The men who get no post-partum hæmorrhage are those who act on the rule never to deliver in the absence of uterine action: to pull in order to *help* uterine contractions, *not* to *replace* them.

*Diagnosis.*—It is most important to distinguish between secondary uterine inertia and tonic contraction of the uterus. The distinction is, as Braxton Hicks used to put it, the very

"keystone" of sound practice in midwifery. There are superficial resemblances. In both, rhythmical pains have ceased; and in both the patient and her friends may be alarmed at the delay, and clamour for speedy delivery. In tonic contraction of the uterus from obstructed labour, the patient's expression is one of anxiety; her pulse is quick, 120 or more, and gets quicker and quicker the longer the condition lasts; the uterus felt by the abdomen is of unchanging hardness—by the vagina, the presenting part is felt pressed down and fixed in the pelvic brim. In uterine inertia, on the contrary, the patient's expression is placid, her pulse is usually under 100, and of normal volume. By the abdomen, the outline of the child's body can be felt with unusual ease, and easily moved about. By the vagina, the presenting part of the child can easily be pressed back. In labour obstructed from excessive size of the child there is a large caput succedaneum, so that suture and fontanelles cannot easily be felt, and there is swelling of the vagina below the head: but not so in uterine inertia.

The treatment of secondary uterine inertia is to imitate and help nature by letting the patient sleep, or if sleep come not, to procure it by chloral or opium. Give a grain of opium, or fifteen minims of laudanum, or half a drachm of chloral; and if in half an hour the patient is not asleep, repeat the dose. I place the opium first, because opium can be conveniently carried in the form of 1-grain pills, which neither evaporate nor stain the bag or pocket. When the patient awakes, uterine action will return with increased force and frequency, and the labour will usually be quickly ended.

C. *Premature Uterine Retraction.*—This is a rare condition, first described by Litzmann, and made known to the profession in England by Matthews Duncan. In it, when the liquor amnii has escaped, pains follow one another rapidly, and the patient's manifestations of suffering make it seem that they are strong; the uterus becomes contracted round the child, and the retraction ring is drawn up. The condition of the uterus is like that in obstructed labour—but *there is no obstruction.* I have seen one case. I was sent for by Mr. T. R. Fendick to help him with a lingering breech labour. I found a patient, very nervous, and intolerant of interference, and who thought herself seven months pregnant; a uterus reaching to one-third of the distance between the umbilicus and the ensiform cartilage; a capacious pelvis; the os uteri well dilated. I told Mr. Fendick that a little time was all that was wanted; that the child was small, the pelvis roomy, the os uteri open, and that there was therefore nothing to hinder delivery. Several hours afterwards I was again sent for. I was told that the patient had been having strong pains ever since my visit, but without advance. I found the state of things on vaginal examination unaltered. I passed my hand into the uterus with ease, and felt a ring of contraction high up, several inches above the pelvic brim, encircling the child's feet and shoulders. I seized a foot and brought it down, and delivered the child without difficulty in a few minutes.

This condition resembles obstructed labour, except in the fact that there is no obstruction. The contractions of the upper part of the uterus instead of driving down the child, have stretched the lower segment of the uterus. Why or how this condition comes about, we know not. I have seen early in the first stage of labour very frequent and painful uterine contractions which, although recurring for a long time, produced very little effect in opening up the cervix; and I have seen these contractions made less frequent, more effective, and less painful by antipyrin—a drug which acts in like manner in painful uterine contractions during pregnancy, after delivery, and during menstruation. I conjecture that if these very frequent, painful, and ineffective uterine contractions were to go on, premature uterine retraction would be produced; but this is only a conjecture—I have not watched the one pass into the other. When premature uterine retraction has come about, the right and only treatment is to deliver, either by forceps, breech traction, or podalic version, according to the presentation. As there is no obstruction, delivery is easy.

The conditions just described are those in which uterine action is weak. I now have to describe the condition which comes about when the uterus is strong, but delivery is mechanically impeded.

D. *Obstructed Labour.*—This is the condition which comes about if the child cannot pass through the genital canal and labour is allowed to go on. The child may be unable to pass either because it is of excessive size, or because the pelvis is contracted, or the child is lying in a wrong position and is too big to pass in the faulty position, though it might have passed in a right position. It is not possible to define how large a child can pass through a normal pelvis, because its passage depends not only on its own size, but on the degree of ossification of its head, and on the size of the pelvis. With a pelvis of full average dimensions, and a child of not more than average size, there is abundance of room, so that if the pains are strong enough and the head soft enough to be moulded, the child can pass in almost any position. If it lies transversely, strong pains can expel it living by the process called the spontaneous evolution of Douglas. If the face or the brow present, a soft head of an average-sized child may be so moulded that it can get through if the pains are strong enough. But children lying in unfavourable positions are often too

large, and the uterine action is not often strong enough to drive a child in an unfavourable position through the pelvis.

When the child's progress through the pelvis is mechanically impossible, or possible only under exceptional conditions, a skilful accoucheur ought to find this out at the beginning of the labour, and apply proper treatment before

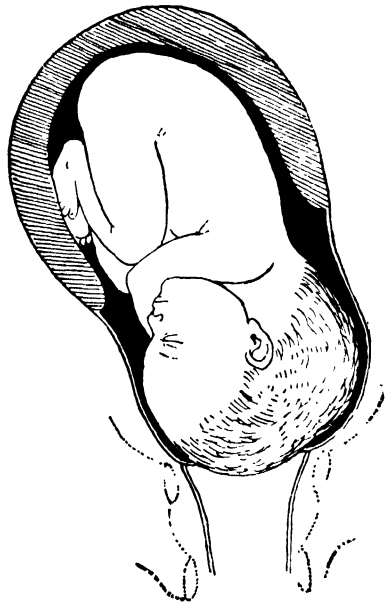


FIG. 1.—Diagram showing thickening of the upper part of the uterus, thinning and stretching of the lower uterine segment. Labour obstructed by hydrocephalus.

the mother's condition has suffered. If the accoucheur is not skilful, or is not sent for in time, the condition known as *obstructed labour* gradually develops. We owe our knowledge of this condition to Braxton Hicks, who was the first to carefully observe it. When labour is obstructed, the pains follow one another with increasing rapidity; the pauses between them get shorter and shorter until at length there is no pause, but the uterus is in continuous contraction. While this is going on the liquor amnii gradually drains away, and the uterus becomes more and more closely moulded to the body of the child. The child, hence, loses its mobility, and the pressure of the most salient and hard fœtal parts upon the parts of the uterus opposed to them becomes continuous. These parts suffer from the pressure; the uterus becomes tender, and the damage by pressure makes it prone to become inflamed after delivery. The continuous pressure hinders the circulation through the placental site upon which the supply of oxygen to the fœtus depends, and may thus kill the fœtus by asphyxia. If the cord be wound round a hard part of the fœtus, it is possible that the continuously contracting uterus may so compress it as to stop the circulation through it and thus kill the fœtus.

The uterine contractions expend much nerve force; and the faster they occur and the longer labour lasts, the greater the strain upon the nervous system. The pain also depresses nervous tone, and to these things is added want of sleep, for the patient cannot sleep while labour is actively going on. When a large head is impacted on the pelvic cavity there is a further source of pain in the pressure on the sacral nerves; but this is of less importance in the exhaustion of the patient than the causes mentioned before. From these causes it results that the continuance of obstructed labour is accompanied with progressively increasing exhaustion of the patient; this is marked by the pulse becoming quicker and smaller, the facial expression anxious, the patient restless, her lips parched and her tongue brown. If her condition continues unrelieved, she will die.

If the existence of disproportion is not recognised, and ergot is given, the symptoms of obstructed labour will develop more quickly; and if proper treatment is not applied, the fatal termination will come sooner.

The upper part of the uterus is that which contracts; the lower part, that below the equator of the fœtal head, has to dilate, and is pulled by the upper part up over the head. When obstructed labour has lasted long, the upper part of the uterus becomes thick, and

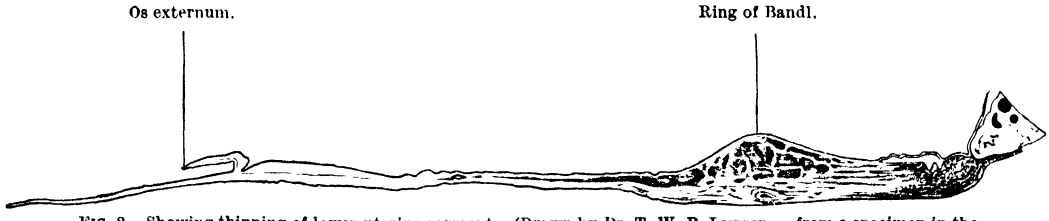



FIG. 2.—Showing thinning of lower uterine segment. (Drawn by Dr. T. W. P. Lawrence, from a specimen in the Museum of the University College, London, by permission of Sir T. Williams.)

the lower segment thin. The line where the thick part joins the thin can sometimes be felt through the abdominal wall, and is called the *ring of Bandl*, after the Austrian obstetrician who first described it.

When there is no hindrance to the pulling up of the cervix uteri, the lower segment of the uterus, the cervix, and the vagina are all equally stretched, and the junction between the

contracted upper part and the stretched lower part is not always marked by an abrupt change in thickness. The protracted pressure of the fœtus upon the stretched part of the genital canal may cause it to give way, and thus *rupture* of the *uterus or vagina* occurs. When the lower part of the uterus and the vagina are equally tense, the one is as likely to give way as the other.

(2) In some cases of obstructed labour a different effect is produced. When the head presents and is too big to enter the brim, it cannot come down far enough to enter the os uteri. The bag of membranes enters the os uteri, and the part unsupported by the cervix uteri receives the full pressure of the uterus upon the liquor amnii; it therefore protrudes more than it does in normal labour, the liquor amnii contained in it and called the "fore-waters" not being, as in normal labour, cut off by the head from the bulk of the liquor amnii. This abnormal pressure upon the part of the membranes protruding through the os, ruptures them long before the os uteri is fully dilated. Then the head nips the cervix uteri between it and the symphysis pubis. As labour goes on, the upper part of the uterus contracts more and more, pulling up the lower segment. But the cervix cannot rise, being held down where it is nipped between the head and the pubic bones. The lower segment therefore becomes more and more stretched and thinned, and the boundary between the upper part of the uterus which contracts, and the lower part which dilates and thins, becomes more and more abrupt. This line of sudden alteration in the thickness of the uterine wall is the ring of Bandl. Much discussion has taken place as to the part of the uterus at which it is formed. Some have maintained that it is identical with the internal os; I think that it is above this, and that it cannot be more exactly defined than as the part of the uterus which corresponds to the equator of the fœtal head, is situated at the brim of the pelvis, and is nearly that of firm attachment of the peritoneum. If obstructed labour still goes on, and the patient dies not from exhaustion, *rupture* of the *uterus* will take place, the rent being in the stretched and thinned lower segment.

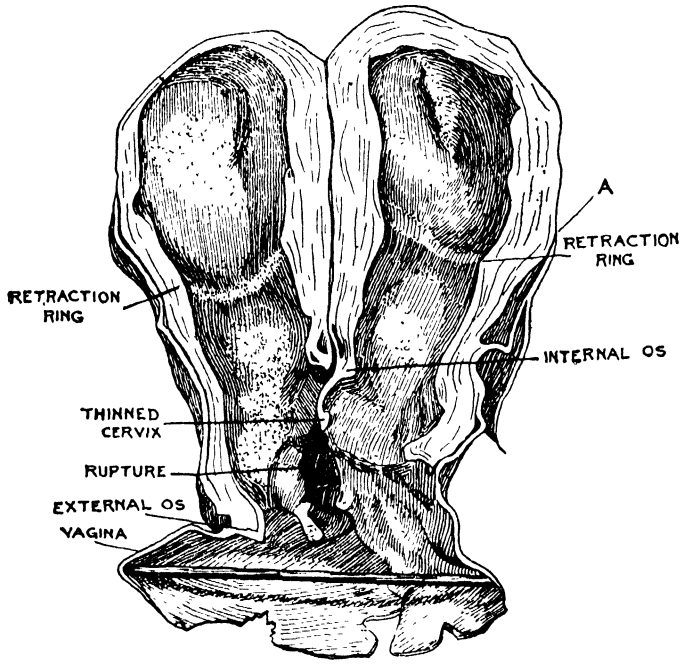



FIG. 3.—Ruptured uterus, showing retraction ring at level of firm attachment of peritoneum; thinning of cervix; gradual thinning of lower uterine segment from retraction ring down to os internum. A, firm attachment of peritoneum. (From a specimen in the London Hospital Museum.)

The symptoms, physical signs, and treatment of rupture of the uterus are described.

The prolonged pressure upon the soft parts nipped between the fœtal head and the symphysis pubis often produces sloughing of these soft tissues, and the formation of urinary fistulæ. These will be found described elsewhere.

In the preceding pages four kinds of abnormal uterine action have been described. In the three first, if they occur in labours otherwise natural, it may be correctly said that labour is prolonged through fault in the powers, and through that alone. In the fourth, the abnormal uterine action is secondary, and a result of the exceptional difficulties which the uterus has to overcome; but it is nevertheless a fault in the powers, and, therefore, I have here described it.

To make this account complete I must add, that labour may be slow because the auxiliary forces, the down-bearing efforts of the patient, are absent, as in paraplegia. But this is very rare; it never prevents delivery, though it may delay it. The only treatment is to supplement uterine action by pushing from above or pulling from below.

(3) I now have to describe the faults in the passage which make labour difficult. These are of two kinds: (*a*) in the bones; (*b*) in the soft parts. I take first obstruction by the bones; in other words, contraction of the pelvis. I shall describe the production, characteristics, effects, diagnosis, and treatment of those pelvic deformities which are common enough and great enough to be obstetrically important.

### PELVIC DEFORMITIES

The shape of the female pelvis is determined by three factors: (1) the innate tendency of

the bones to grow into their proper shape; (2) the pressure of the weight of the body through the vertebral column on the sacrum, which it presses downwards and forwards, and the reacting pressure of the femora upon the acetabula, which they press upwards; (3) the pull of muscles and ligaments upon the pelvic bones. Deformed pelves are produced by altered effects of these forces: (1) the bones may be stunted in growth either uniformly, or in special places; (2) they may be softened by disease, so that they yield unduly to pressure and pulling; (3) muscles and ligaments may be displaced by disease or accident so that they come to pull in an abnormal way. In many pelvic deformities these three agencies are combined, so that it is difficult to separate the action of each force; and there has been, and still is, difference of opinion as to the way in which certain deformities are produced.

At birth the sexual differences between the male and female pelvis are already evident, although they are not so marked as later in life. The differences between the infantile and the adult pelvis are more marked (Figs. 4, 5). In the fœtus, the sacrum is less curved from above downwards than in the adult; the sacral promontory is high above the plane of the brim; the transverse diameters of the pelvis are narrower in proportion to the others than in the adult. The vertebral column is nearly straight; the lumbar convexity and the dorsal concavity hardly exist. As the child grows up the curves of the spine are produced. The body weight presses the sacral promontory downwards and forwards; this increases the curve of the sacrum from above downwards, and presses down the promontory until it comes to be very little above the plane of the brim.

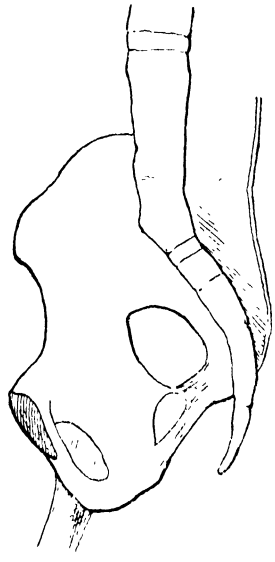


FIG. 4.—Pelvis of fœtus at term. (After Balandin.)

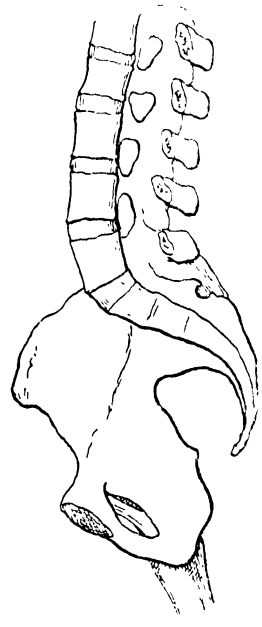


FIG. 5.—Pelvis of adult. (After Balandin.)

The pressure of the body weight upon the sacrum can be split up into two components,

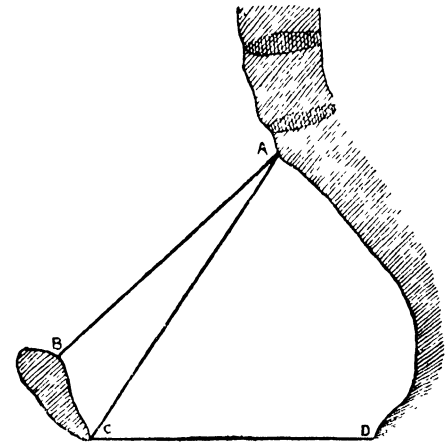



FIG. 6.—Sagittal section of normal pelvis. AB, true conjugate; AC, diagonal conjugate; CD, antero-posterior diameter of outlet. (After Pinard)

one acting downwards and backwards, the other downwards and forwards. The former tends to force the sacrum downwards between the two innominate bones; the latter tends to force the promontory forwards towards the symphysis. It is obvious that the relative magnitude of these two components will vary with the inclination of the pelvis. The less the pelvis is inclined to the horizon, the more the sacrum will be driven down and the less it will be driven forwards; the greater the inclination of the pelvis to the horizon, the more will the sacral promontory be driven forwards. This theoretical reasoning is unimpeachable; but it has never been shown that the projection of the sacral promontory does in fact vary with the pelvic inclination. The pelvic inclination, whatever it may be in the somewhat artificial conditions under which it has been measured, is continually varying in different postures; so that I think the pelvic inclination, although a factor in modifying the shape of the pelvis, is not a factor of the first importance.

The femora press directly upwards. As the acetabulum is outside the line along which the body weight is transmitted, viz., one from the sacrum to the feet, the pressure of the femora tends to force the acetabula outwards as well as upwards. This pressure is resisted by the ligaments of the pubic symphysis, which hold the pubic bones together. When these ligaments are divided, the pubic bones fly apart, and if the femora are pressed upwards the pubic bones diverge yet more. Hence the combined influence of the downward pressure of the sacrum and the up-

ward pressure of the femora is to widen the pelvis. The widening is also aided by the growth of the lateral masses of the sacrum, which is wider in proportion to its length in the adult than in the child, and by the growth of the ilia. In the child, the posterior half of the pelvic ring is formed almost entirely by the sacrum; but in the adult, the sacrum only forms a part of it, the rest being formed by the ilia.

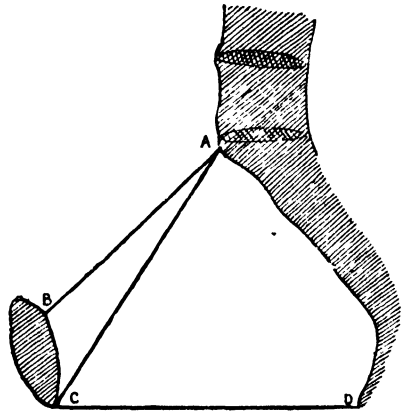



FIG. 7.—Sagittal section of flat pelvis. AB, true conjugate; AC, diagonal conjugate; CD, antero-posterior diameter of outlet. (After Pinard.)

These normal developmental changes vary in degree, from causes that we know not; just as some members of the same family grow tall, others short—we know not why. If the changes that have been described proceed to an excess, the promontory of the sacrum is lower down and farther forward, and the sacrum is more curved from above downwards than it should be. The

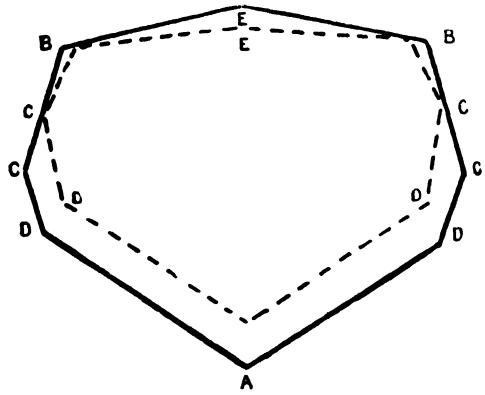



FIG. 8.—Diagram of the generally contracted flat non-rickety pelvis: black line normal pelvis; dotted line contracted pelvis. E, sacral concavity and brim; BB, sacrum; CC, transverse diameter; DD, ilio-pectineal eminence; A, symphysis.

conjugate diameter at the brim is then less than the average, but the other measurements of the pelvis are of average size. A pelvis of this shape is called a *flat pelvis*; it is one of the commonest pelvic deformities.

Sometimes the normal developmental changes are deficient in degree. The pelvis does not increase in breadth as it ought to do; the sacrum may be straighter than usual, and the promontory higher. Such a pelvis, contracted mainly in its transverse measurements, is called the *generally contracted pelvis*—the *pelvis æquali-biter justo minor*, or the *small round pelvis*. Sometimes the two conditions just described occur together: the growth of the ilia and lateral masses of the sacrum is defective, and so the pelvis does not attain its normal breadth, and at the same time the promontory of the sacrum is driven unduly forward and downwards. Then a pelvis is produced which is contracted in the conjugate diameter and in the transverse diameters also; this form of pelvis is called the *generally contracted and flattened pelvis*, or the *small flat pelvis*.

In the forms of contracted pelvis just described, there is no evidence of any disease of the bones, and there is no deformity elsewhere. The patients are often undersized; but they may be of average stature, or even above the average. We know

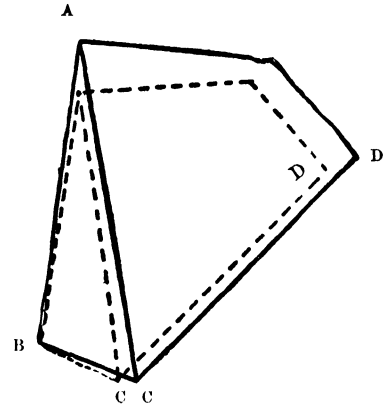



FIG. 9.—Diagram of pelvic cavity of generally contracted flat non-rickety pelvis. AB, true conjugate; AC, diagonal conjugate; CD, antero-posterior diameter of outlet.

nothing as to why these defects in growth occur. These minor degrees of pelvic contraction are not accompanied with any peculiarities of attitude or gait: they can only be detected by careful examination and measurement of the pelvis. The flat pelvis and the small round pelvis are the commonest kinds of pelvic deformity. I know of no trustworthy statistics showing how common they are in England. The practice of lying-in hospitals and of specialists gives not a true representation of their frequency, for patients go to such places and persons because they are known or suspected to have contracted pelves.

Two rare kinds of pelvis are often described as varieties of the small round pelvis. One is the *dwarf's pelvis*. This is a pelvis which is like the rest of the skeleton of a dwarf in being diminutive in size, but not deformed in shape. Females whose growth is so stunted that they may be properly spoken of as "dwarfs" are generally sterile. I know of no account of labour in a dwarf. The other is the *small round rickety*

*pelvis.* This is simply a small round pelvis without any rickety deformity, occurring in a patient

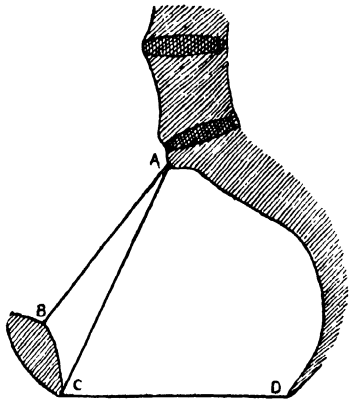



FIG. 10. — Sagittal section of small round pelvis. AB, true conjugate; AC, diagonal conjugate; CD, antero-posterior diameter of outlet.

who has signs of rickets, or what are taken to be such, in some other part of the body. I see

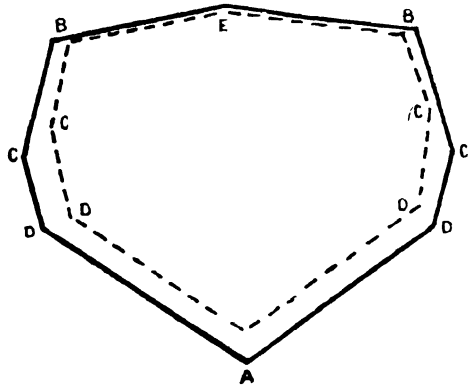



FIG 11.—Diagram of the brim of the same round pelvis · black line, normal pelvis; dotted line, small round pelvis. BB, sacrum; CC, end of transverse diameter; DD, ilio-pectineal eminences; E, centre of sacrum in plane of brim; A, symphysis.

no reason, in such a case, for labelling the pelvis with the adjective "rickety."

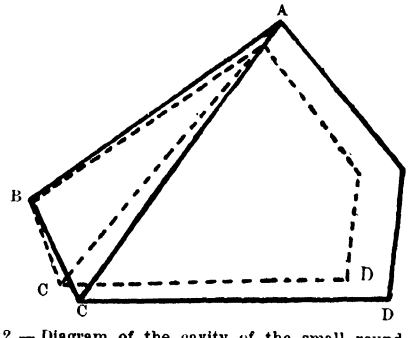



FIG. 12. — Diagram of the cavity of the small round pelvis. AB, true conjugate; AC, diagonal conjugate; CD, antero-posterior diameter at outlet; continuous line, normal pelvis; dotted line, contracted pelvis.

The commonest cause of *great* contraction of the pelvis is *rickets.* The features of this disease which are important obstetrically, are softening of the bones and enlargement of the epiphyses The bones being soft during part of their time of growth, yield excessively to the forces which mould the shape of the pelvis. The sacrum, yielding to the body weight, sinks farther downwards and forwards than it ought to do, and hence the conjugate diameter of the brim is shortened. The body weight falls on the upper part of the sacrum. The lower sacral vertebræ are not exposed to this pressure, but are held up by

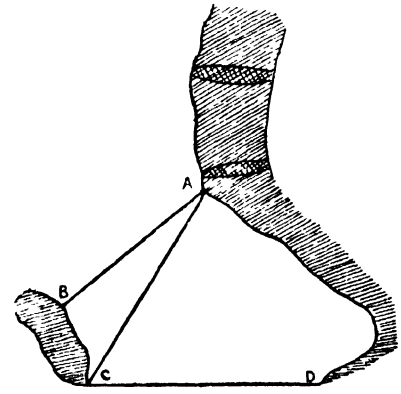



FIG. 13.—Sagittal section of flat rickety pelvis. AB, true conjugate; AC, diagonal conjugate; CD, antero-posterior diameter of outlet.

strong ligaments which connect them with the ilia; hence the sacral curve from above downwards is exaggerated, the upper part of the bone being abruptly curved forwards. The body weight falls upon the middle of the bone, the sides are held up by their ligamentous attachments to the ilia; hence the middle of

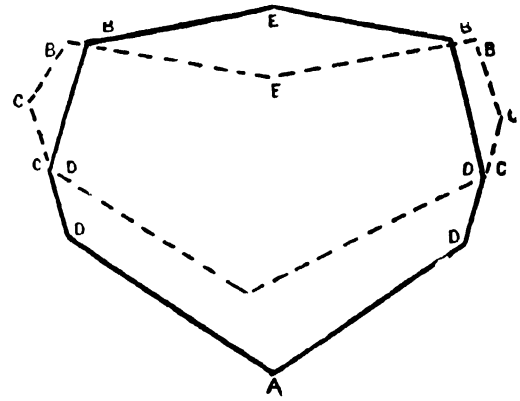



FIG. 14.—Diagram of rickety flat pelvis. BB, sacrum at level of brim; CC, transverse diameters; DD ilio-pectineal eminences; A, pubes; continuous line, normal pelvis; dotted line, contracted pelvis.

the sacrum is bulged down, and its anterior surface becomes convex from side to side, instead of concave, as in a normal sacrum. The downward pressure of the body weight is transmitted through the ilia to the femora, and by them to the legs and feet; its direction, therefore, is along a line from the sacrum to the feet. The acetabula are situated outside this line; the femora, therefore, press the acetabula upwards

and outwards, and so widen the pelvis. As the acetabula are pressed upwards and outwards, the ilia are pressed in the same direction, and

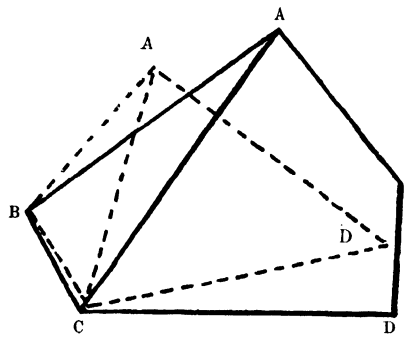



FIG. 15.—Rickety flat pelvis. AB, true conjugate; AC, diagonal conjugate; CD, antero-posterior diameter at outlet; black line, normal pelvis; dotted line, contracted pelvis.

the iliac fossæ come to look more forwards and less inwards than in the normal pelvis; so that the iliac crests, instead of at their anterior parts

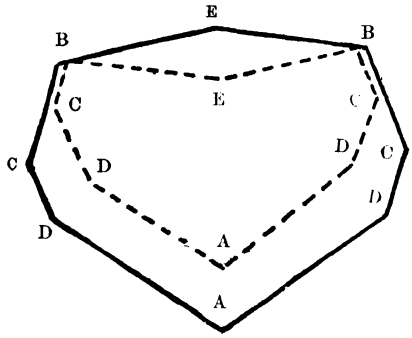



FIG. 16.—Diagram of brim of small flat rickety pelvis. BB, sacrum; E, centre of sacrum in plane of brim; CC, transverse diameter; DD, ilio-pectineal eminence; A, symphysis pubis; continuous line, normal pelvis; dotted line, contracted pelvis.

curving inwards, run directly forwards, or even forwards and outwards. The ilia are slightly rotated about an axis parallel with the sacro-

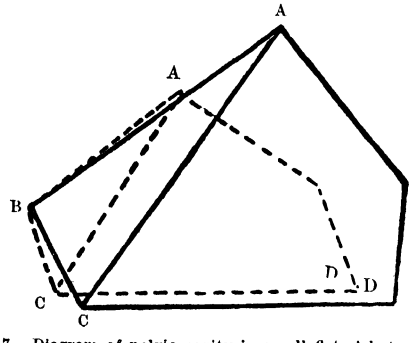



FIG. 17.—Diagram of pelvic cavity in small flat rickety pelvis: continuous line, normal pelvis; dotted line, contracted pelvis. AB, true conjugate; AC, diagonal conjugate; DC, antero-posterior diameter of outlet.

iliac synchondrosis, so that the posterior iliac spines are nearer together than in the normal pelvis. The trochanters are, with relation to the pelvis, farther upwards and outwards than normal; and this involves an extra pull, in an upward and outward direction, upon the muscles running from the ischia to the trochanters. Hence by slight eversion of the ischial tuberosities the outlet of the pelvis is a little widened. The epiphyses in rickets are enlarged; hence in the rickety pelvis the

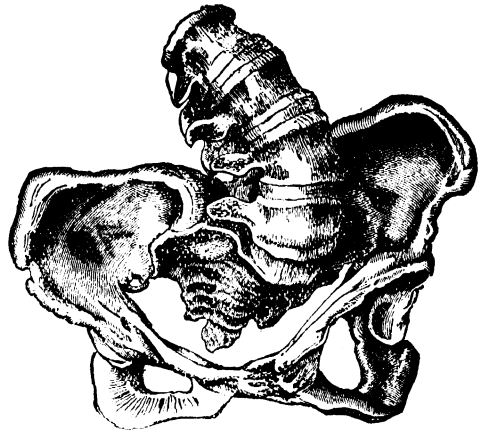


FIG. 18.—Scolio-rachitic pelvis.

epiphyses of the sacral vertebræ can be felt as ridges running across it, and the symphysis pubis is thickened.

Rickets is accompanied with stunting of growth; hence rickety subjects are generally undersized, and a rickety pelvis is often small. Therefore we have two kinds of rickety pelvis: the *flat rickety pelvis*, in which the conjugate is

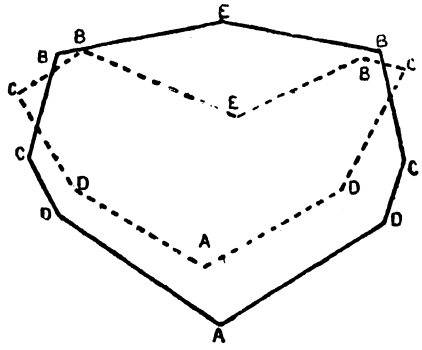



FIG. 19.—Diagram of brim of scolio-rachitic pelvis: continuous line, normal pelvis; dotted line, deformed pelvis. BB, sacro-iliac synchondroses; E, centre of sacrum in plane of brim; CC, transverse diameter; DD, pectineal eminences; A, symphysis pubis.

diminished but the transverse measurements either normal or increased; and the *small flat rickety pelvis*, in which all the diameters are small, but the conjugate is especially contracted. The cases of extreme pelvic contraction that are met with in England are almost all pelves either of this kind, or of the one next to be described.

With rickets there often goes lateral curvature of the spine. When this is so, the pelvis is unsymmetrically deformed. The body weight

falls unduly on the side to which the lumbar convexity looks, and presses the sacrum towards that side. The lateral mass of the sacrum and the ilium on the side of the lumbar convexity are compressed, the bony tissue being more compact than normal. The sacrum and the acetabulum are thus brought nearer together, and the ilio-pectineal line on that side is more

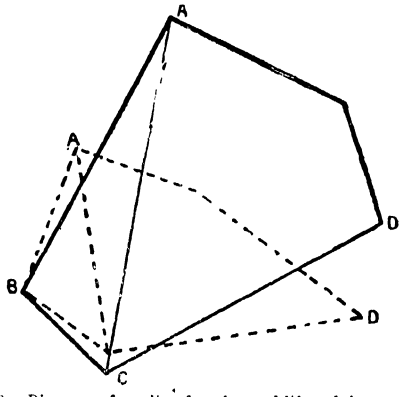



FIG. 20.—Diagram of cavity of scolio-rachitic pelvis; continuous line, normal pelvis; dotted line, deformed pelvis. AB, true conjugate; AC, diagonal conjugate; CD, antero-posterior diameter of outlet.

sharply curved. In short, the pelvis is unequally contracted; the side to which the lumbar convexity looks being the narrower, because it is the over-weighted side. The symphysis pubis is pulled over, away from the side to which the lumbar convexity looks, and the acetabulum on the underweighted side is higher up and farther out: because the outward pressure of the femur acts on this side to greater advantage. This pelvis is called the *scolio-rachitic pelvis.*

THE MECHANISM OF LABOUR WITH CONTRACTED PELVIS.—In describing the mechanism of labour with contracted pelvis, it must be premised that it is assumed that the child is of average size and normal conformation. If the child is too big, the effect on labour is the same as if the pelvis were generally contracted; if the child is below the average size, it may come through a contracted pelvis without difficulty or altered mechanism.

In considering the slighter kinds of pelvic contraction, the question arises—How, and where, is the line to be drawn between a normal and a contracted pelvis? The answer is, that a pelvis which will allow a well-formed child of average size to pass through it in the usual way, that is, with the normal mechanism, is a normal pelvis. If the pelvis is in any diameter so contracted that the child cannot pass in the usual way, but must, by a special mechanism, adapt itself to the altered diameters of the canal, that pelvis is contracted. Let us apply this principle. The true conjugate of a normal pelvis averages about $4\frac{1}{4}$ inches, its transverse and oblique diameters about 5 inches. The fœtal head as it usually presents, lies in the oblique diameter of the brim, partly flexed, so that the occipito-frontal diameter, which averages about $4\frac{3}{4}$ inches, lies in the oblique diameter. In the conjugate diameter of the pelvis lies a diameter of the head running from in front of one parietal eminence to behind the opposite one, and averaging about 4 inches. There is then, as has been pointed out in a former page, no hindrance offered by the bones to the passage of the fœtal head. If the size of the pelvis is so altered that the head cannot thus enter the pelvis, then the pelvis is contracted. If, for instance, the conjugate diameter measures only $3\frac{3}{4}$ inches, the diameter running from in front of one parietal eminence to behind the opposite one cannot possibly enter the conjugate, and the head must enter with its long diameter lying transversely and the bitemporal diameter occupying the conjugate. A pelvis with its conjugate diameter shortened to this extent is, therefore, contracted; and if its other diameters are normal, it is called a flat pelvis. Suppose now that the conjugate diameter of the pelvis only measures 4 inches, and the oblique and transverse measurements at the brim $4\frac{1}{2}$ inches. It will still be just possible for the head to enter the pelvis in the oblique diameter; but the occipito-frontal diameter will not enter the oblique diameter of the pelvis. To pass, the head must be much flexed, so that the suboccipito-frontal measurement may enter the brim. Thus a flat pelvis having a conjugate of $3\frac{3}{4}$ inches, and a small round pelvis having a conjugate of 4 inches or less, are called contracted; anything above these measurements may be considered as normal, only causing difficulty if the child is large.

The mechanism of labour with contracted pelvis is not merely theoretically interesting, but is important, because the delivery of a living child depends upon its entering the pelvis in the most advantageous way; and because from observation of the mechanism of the labour the existence and the kind of pelvic contraction can be inferred, the reason of prolongation of labour ascertained, and indications drawn as to the best treatment. This statement applies only to the slighter forms of pelvic contraction: in deformity so great as to prevent the delivery of a living child, the mechanism is less important.

I shall describe the mechanism of labour with the two common forms of slight pelvic contraction—the flat, and the small round pelvis. The mechanism of labour with the flat pelvis depends upon the degree of contraction, and not upon whether the pelvis is or is not rickety.

One feature of the mechanism of labour with the flat pelvis has already been alluded to, and the reason for it explained, viz., that the head enters the pelvis with its long diameter in the *transverse* diameter of the pelvis, so that its bitemporal diameter, or one a little behind it, is

engaged in the conjugate of the brim. It takes this position because there is not room in the conjugate for the oblique diameter, from in front of one parietal eminence to behind the opposite one, which in normal labour enters the conjugate. A second feature is that in labour with a flat pelvis the head enters the brim rather more *extended* than in a normal pelvis;

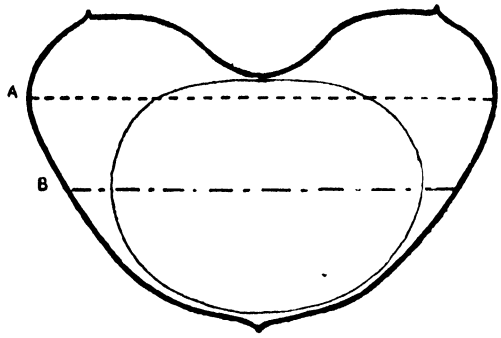



FIG. 21.—Position in which the head enters the brim of the flat pelvis. A, transverse diameter of pelvis; B, diameter in which long diameter of head lies.

so that the finger in the vagina, instead of feeling the smaller fontanelle low down and the anterior high up and behind, feels both fontanelles at about the same level. The reason is that the greatest transverse diameter of the head, the biparietal, is behind the centre of the head. Therefore the front of the head descends more easily than the back, and thus slight extension of the head is produced. This extension does not go beyond a certain degree, because for complete extension to occur the mento-vertical diameter would have to engage in the brim; and as the transverse measurement of the pelvis is 5 inches, and the mento-vertical diameter of the head $5\frac{1}{4}$ inches, this is not possible, unless either the child is very small, or the head greatly reduced in size by moulding. Under those conditions it does occasionally occur. The third feature of labour with the flat pelvis is the occurrence of what is called, from the obstetrician who described it, the *obliquity of Naegelé*. This means that the head is so inclined that its biparietal diameter is oblique in relation to the plane of the brim. The anterior-lying parietal eminence is lower down than the one which lies behind, and the sagittal suture is nearer the sacral promontory than the symphysis pubis. The production of this obliquity depends upon the fact that the axis of the uterus is not a continuation of that of the pelvic inlet, but lies behind such a line. If no pelvic deformity be present, the child is driven into the pelvis with exactly the opposite obliquity, viz., the posterior parietal bone sunk lower into the pelvis than the anterior, and the sagittal suture rather nearer the pubes than the sacral promontory. But if the sacral promontory jut forward abnormally, the descent of the posterior parietal bone is impeded, while that of the anterior parietal bone is not. Hence the anterior parietal bone is driven down, and the transverse diameters of the head rotate round the promontory, until the anterior-lying parietal bone can sink no farther into the pelvis, and thus the obliquity of Naegelé is produced. This obliquity is not only a characteristic feature of labour with a flat pelvis,

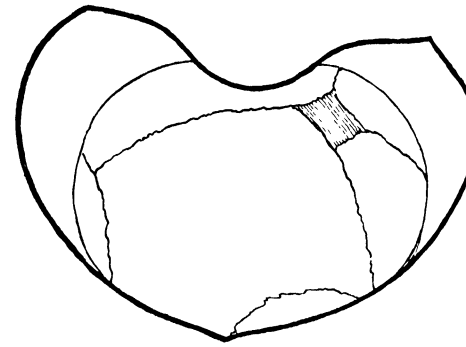


FIG. 22.—Obliquity of Naegelé; the sagittal suture near the sacral promontory.

but is a movement favourable to the passage of the head through the brim. When it has come about, the transverse diameter of the head which has to pass through the pelvis is a subparietal, superparietal diameter, which is about a quarter of an inch less, upon the average, than the biparietal diameter. Further, the existence of this obliquity implies, as a rule, that the head has been able to engage with nearly its greatest diameter in the brim; for if the pelvis is so contracted that the head cannot engage in it, the situation of the sagittal suture becomes, so to speak, a matter of accident. Litzmann regarded the distance of the sagittal suture from the promontory as a guide to the probable difficulty of delivery; he drew from his experience the practical rule that when the sagittal suture ran transversely, and was distant about three-quarters of an inch from the sacral promontory, forceps delivery was generally easy.

The three peculiarities just described—*transverse position* of the long axis of the head; increased, but incomplete, *extension* of the head; and the *obliquity of Naegelé*—are those which characterise the entry of the head into the brim of a flat pelvis. In a flat pelvis the only difficulty is that which attends the entry of the head into the brim, and its passage through it. When once the head has passed the brim, all difficulty is at an end. The head entering the brim with the Naegelé obliquity, the anterior parietal bone becomes fixed against the symphysis pubis, and then the posterior-lying parietal bone gradually scrapes past the sacral promontory; if it is pulled through with forceps, the operator will feel it suddenly slip past the promontory, and will find that then

it is easily pulled farther. The passage of the promontory is generally — always, in difficult cases — made possible by alteration in the shape of the head. The posterior-lying parietal bone becomes flattened, and driven under the anterior, and also under the parietal and occipital bones. The line along which the head was opposed to the most projecting point of the promontory is often traceable, either by redness and ecchymosis of the skin, or by a groove in the bone. The usual situation of such a groove is along the anterior border of the parietal bone. If the head is soft and has been allowed to remain long stationary at the brim, a deep spoon-shaped dint may be formed at the point where the head rested against the promontory. Lastly, it must be mentioned that the weakest part of the fœtal head is the anterior inferior angle of the parietal bone; and that where pressure upon, and overriding of bones, is great at this point, such force may lead to laceration of vessels, meningeal hæmorrhage, and death of the child. Cases are met with in which the child is known to be alive, and is delivered by short but strong pulling with forceps, but dead. In such cases, meningeal hæmorrhage is the usual cause of death.

When with a flat pelvis the child is delivered by turning, the after-coming head passes the brim by a mechanism exactly analogous to that which obtains when the head comes first. The head lies transversely, the biparietal diameter being at one side of the promontory. If there is much resistance to the passage of the head, it becomes partly extended. When the head is pulled upon, the anterior part of the head descends first. Then the projecting sacral promontory holds back the posterior-lying parietal bone, and the anterior side of the head descends first, rotating upon the sacral promontory as a centre; and then, lastly, the posterior-lying parietal bone slips down, often being grooved or marked in the same way as if the head had come first. In labour with a flat pelvis, a large caput succedaneum is unusual, nor is there often œdema of the vagina or vulva. Premature rupture of the membranes is common with flat pelves, from causes and with results which are described elsewhere. Abnormal presentations of all kinds are more frequent with flat pelves than with normal pelves; their treatment is described elsewhere. I would only here say that when a face presentation is met with in a flat pelvis, I think the best treatment is podalic version.

FIG. 23.—Showing mark made by promontory in delivery of the after-coming head. (After Kustner.)

The mechanism of labour with the small round pelvis is in one point in broad contrast with that of labour with the flat pelvis. In the flat pelvis all the difficulty is at the brim; in the small round pelvis there is difficulty throughout the whole pelvic canal. The difficulty is to get the head *into* the flat pelvis, to get it *through* the small round pelvis. In the small round pelvis there is not the liability to abnormal presentation, nor to premature rupture of the membranes, which the flat pelvis brings with it; for the head readily enters the pelvis, engages in it, and shuts off the forewaters from the general intra-uterine pressure.

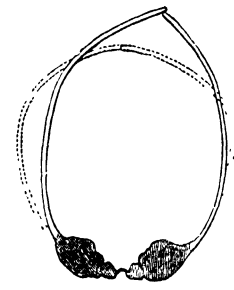


FIG. 24.—Showing change in shape of head produced by traction with base in advance. (See also Fig. 52, p. 235.)

In the small round pelvis the head can only get through it in a position of *extreme flexion*, so that the suboccipito-frontal diameter may be the largest which passes through the pelvic cavity. Hence the posterior fontanelle is lower down and

FIG. 25.—Pressure marks on head after a labour with flat pelvis.

nearer the middle of the pelvis than usual; and flexion occurs earlier than in normal labours, because it is produced as soon as the head sinks into the pelvis. The head enters the pelvis with its long diameter in the usual oblique diameter of the pelvis, because at the brim this diameter is the longest. At the pelvic outlet the antero-posterior diameter is the longest; and therefore the head, as soon as it gets into the pelvic outlet, turns so that its long diameter occupies the antero-posterior diameter of the outlet. In normal labour a similar turn takes place; but here the turn is caused by the soft parts, not by the bones, and it occurs not till the head is past the pelvic out-

let and is stretching the perineum. This *early rotation* is one of the features of labour with the small round pelvis. In labour with a normal pelvis the head advances during each pain, and recedes in the intervals between the pains; and after the soft parts have turned its long diameter till it lies antero-posteriorly, there is nothing to turn it into any other position. But if the pelvis is of the small round class, and the head is turned forwards by the bones, when it recedes between the pains its position is still governed by the bones; and it therefore turns back again, so that its long diameter may still lie in the longest diameter of the pelvis, which, after the recession of the head, is the oblique. This *variability of position* is very characteristic of the small round pelvis. When the passage of the head through the pelvis is difficult, either because the pelvis is a small round one, or because the child is very large, the difficulty is not, as in the flat pelvis, at the brim only, and there only in the conjugate diameter, bounded by the sacral promontory and the symphysis pubis, but it is throughout the whole pelvic canal, and the head is pressed upon all round by the pelvic canal. Hence where the head is so pressed upon — the girdle of contact—the return of blood from the part of the scalp which is presenting is impeded. Hence early and extensive œdema of the part—in other words, a *large caput succedaneum*. The return of blood is also impeded from the lower part of the vagina and vulva, and hence *œdema of the vagina and labia*. But as the blood from these latter parts can return to the heart by other channels than those pressed on by the advancing head, the œdema of the labia is not so great, and is later in forming than the caput succedaneum. The swelling of the labia is visible; the vagina is felt to be dry and swollen. With the small round pelvis the obliquity of Naegelé is absent; for the head is not hindered in descent by the sacral promontory—it enters the pelvis without difficulty, but meets with hindrance to its passage through it. The head often enters the small round pelvis with posterior obliquity; but I know not that the existence of this obliquity materially affects the course of labour. The passage of the head through the small round pelvis is helped by moulding of the head. The head being pressed upon all round, there is a general compression of the head mainly affecting the suboccipito-frontal measurement, and elongation of its long diameter, the vertico-mental. Grooves and dints in the bones are rare. There is sometimes a red stripe on the skin where it passed the promontory; this runs from the parietal bone downwards and forwards towards the jaw or eye. At birth the occipital and frontal bones are commonly pressed under the parietal bones, and the posterior-lying parietal bone underneath the anterior; but this overriding is effaced within a few days. Lateral asymmetry of the skull is a frequent result of prolonged labour, both with the small round pelvis and with the flat pelvis; but I think it more common and more marked with the small round pelvis. The reason is in the projection of the sacral promontory. As the head descends into the small round pelvis the half of the head that lies behind meets with more resistance, and hence gets pushed forwards (speaking with reference to the head). In a flat pelvis the resistance offered by the promontory to the descent of the biparietal diameter leads to the displacement backwards of the half of the head that lay behind. Rupture of the uterus occurs less frequently with the small round pelvis than with the flat pelvis; because in the former, if the head nips the cervix so tightly as to prevent it rising, such pressure is exerted all round the pelvis, and so quickly produces œdema of the vagina and vulva that the need for prompt treatment is soon apparent.

FIG. 26.—Pressure marks on head after a labour with small round pelvis.

In the foregoing pages I have described the common kinds of slight pelvic deformity and the mechanism of labour occurring with them. From the point of view of the practical obstetrician these are more important than the greatly deformed pelves, because (1) they are common, while great deformity is rare. (2) By recognising them early and managing labour properly, a living child can often be delivered, and the mother always saved injury from protracted labour; their accurate diagnosis requires full knowledge and careful examination; and the decision as to the best treatment is often difficult. Great pelvic deformity is, as it were, forced upon the notice of the doctor, and when discovered there is no doubt as to the proper treatment. (3) When the head cannot pass the pelvis, or cannot pass it without being first crushed, the mechanism of its passage is not important.

### RARER FORMS

I now describe the rarer forms of pelvic contraction, and I shall point out the important features special to labour with each.

The flat, the small round, and the different kinds of rickety pelvis are the common kinds of contracted pelvis—those which anyone who has a large midwifery experience is sure to meet. Some of the rare forms are interesting on account of the light they throw on the development of the pelvis.

¶ The *funnel-shaped pelvis* means a pelvis without disease of the bones, in which the transverse dimensions lessen in size from above downwards. Only two specimens of this deformity had, up to 1889, been described. The accounts of clinical observers would make one think that this pelvis must be commoner in practice than it is in

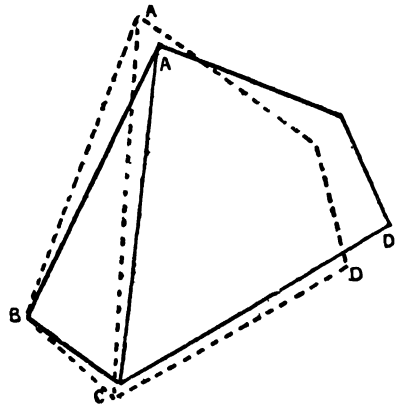



FIG. 27.—Diagram of cavity of funnel shaped pelvis in sagittal plane; continuous line, normal pelvis, dotted line, funnel-shaped pelvis AB, true conjugate; AC, diagonal conjugate; CD, antero-posterior diameter of outlet.

museums. But the difficulty in diagnosis is so great—for we have no means of accurately measuring during life the transverse diameters of the pelvis—that clinical accounts can only be accepted when it is evident that the reporter has been aware of the great probability of error. If labour is lingering, and the cause seems to be that the advance of the head is blocked by a narrow pelvic outlet, the treatment is to help

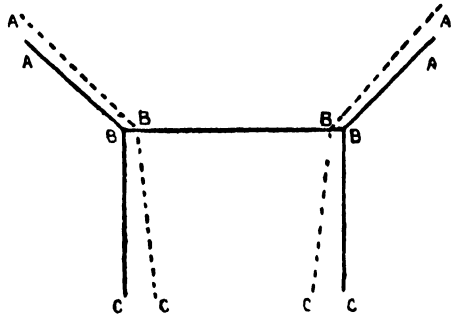



FIG. 28.—Diagram of cavity of funnel-shaped pelvis in coronal plane: continuous line, normal pelvis; dotted line, funnel-shaped pelvis. AA, iliac crests; BB, transverse diameter of brim; CC, inner surface of tubera ischii.

delivery by pulling with forceps. If this fail, cephalotripsy is the only resource. After delivery the pelvis should be measured; and the patient told to come for advice, should a subsequent pregnancy occur, not later than the seventh month.

The *oblique pelvis of Naegelé* is a rare pelvis, the shape of which is altered by a defect in ossification. The defect consists in imperfect development, on one side only, of the lateral part of the sacrum and the adjacent part of the ilium, and ossification of the sacro-iliac synchondrosis on that side. We know nothing as to the cause or date of the developmental defect. Its effect is to bring the acetabulum, on the affected side, nearer the middle line than in the normal pelvis, and nearer the middle line than the acetabulum on the opposite side. I have

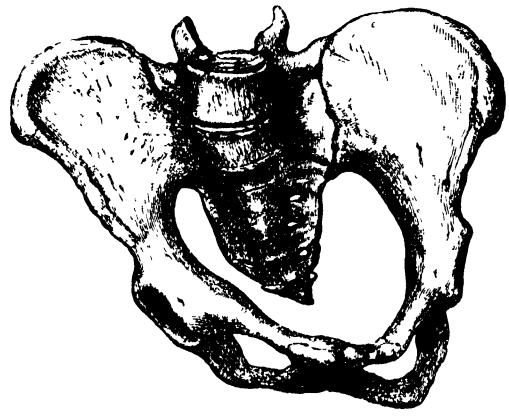


FIG. 29.—Obliquely contracted pelvis of Naegelé.

already pointed out that the femora press the acetabula upwards and outwards. The farther out are the acetabula, the more effective is the outward thrust; hence on the diseased side the thrust of the femur is mainly upwards, and but little outwards. On the sound side the outward pressure acts with greater advantage and the upward pressure with less; hence on the sound side the acetabulum is pushed farther outwards than usual, the wing of the ilium looks more forwards and less inwards, and the

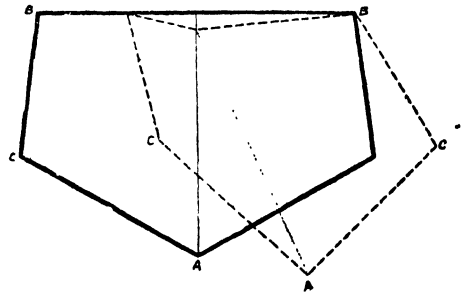



FIG. 30.—Diagram of the brim of Fig. 29: continuous line, normal pelvis; dotted line, oblique pelvis. BB, sacrum; CC, acetabulo; A, symphysis pubis.

symphysis pubis is pulled towards the sound side.

There is no deformity in any other part of the body. There is no history of injury, disease, or lameness, and the patient presents, when clothed, no peculiarity of aspect or gait. Hence the existence of this pelvis is not suspected until it is discovered on obstetrical examination during pregnancy or labour.

The diagnosis of the oblique pelvis of Naegelé is to be made (1) by feeling the outline of the iliac crests, perceiving their asymmetry and

the displacement of the symphysis pubis. (2) By measuring with callipers the distance between the posterior superior iliac spine of one side and the anterior superior iliac spine of the opposite side; these measurements on the two sides will be unequal, that which is taken from the diseased side behind being the greater. (3) By exploring the pelvic cavity with two fingers in the vagina, and noting its shape.

The important point obstetrically about the Naegelé pelvis is the diminution in the oblique diameter on the diseased side. If labour is to terminate naturally the head must be small, and must enter the pelvis with the occiput towards the obturator foramen on the sound side. As these conditions are not always complied with, the infantile mortality is about 25 per cent.

If consulted during pregnancy by a patient who is found to have an oblique Naegelé pelvis, the relative sizes of the head and the pelvis should be estimated by abdominal palpation, and labour induced before the head has got so large that it cannot be pressed into the brim. If consulted for the first time when labour is in progress, the question is—Can the head enter the brim or not? If its equator is engaged in the pelvis, or can be pressed down into it, there is no need for interference. If the head cannot enter the brim, the choice lies between craniotomy and Cæsarean section—the former being the safer for the mother, the latter preserving the child. Cæsarean section should not be chosen if the patient has been long in labour. Turning gives no advantage. Symphysiotomy, owing to the ankylosis of one sacro-iliac synchondrosis, will not enlarge a Naegelé pelvis as much as it does a normal pelvis.

The *transversely contracted pelvis of Robert* is that produced by want of development of the

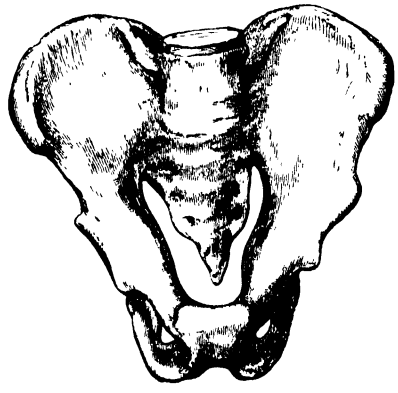


FIG. 31.—Transversely contracted pelvis of Robert.

lateral masses of the sacrum, and ankylosis of the sacro-iliac synchondrosis on both sides. The result is that the pelvis grows not in breadth as it should do. Hence the acetabula are nearer the middle line, and the outward pressure of the femora upon them is exerted to less advantage; the acetabula are driven more upwards and less outwards. The ossa innominata are less curved; the parts between the acetabula and the sacrum are compressed, shortened, and thickened. Hence the conjugate diameter is shortened as well as the transverse, though not to the same high degree.

The diagnosis of the transversely contracted pelvis of Robert is made by the transverse measurements of the pelvis: the intercristal, anterior and posterior interspinous, and bitrochanteric. With the finger in the vagina, the closeness of the ischial tuberosities and the narrowness of the pubic arch will be perceived. This pelvis is usually so small that the only way of delivering its owner of a living child is by Cæsarean section.

The *kyphotic pelvis* is that which is produced when angular curvature of the spine occurs low down. When such curvature is high up it is compensated for by lordosis of the lumbar spine. But when it is so low down that change in the curve of the spine below it cannot compensate its effect, then a change in the inclination of the pelvis takes place, and this change in inclination gradually produces change in shape.

In the diagram (Fig. 32), CG represents the upper limb of the kyphosis. The weight of the upper part of the body acts along the line CGI. GP represents the lower limb of the angle; P is the sacral promontory; PC the sacrum. The effect of the pressure acting along the line CGI is to drive the angle of the kyphosis downwards and backwards, and this movement, through the traction on the lower limb of the angle, pulls the sacral promontory upwards and backwards. The inclination of the pelvic brim is changed, so that if its shape were unaltered its plane would form a less angle with the horizon. But the continuous pull on the promontory in the course of years makes the curve of the sacrum from above downwards less, raises the promontory above the level of the pelvic brim, and lengthens the distance between the sacral promontory and the symphysis pubis. As in rickets the bodies of the sacral vertebræ are the parts most pushed down, so in the kyphotic pelvis the bodies are the parts most pulled up—the lateral parts of the sacrum being in each case bound to the iliac bones. Hence the concavity of the sacrum

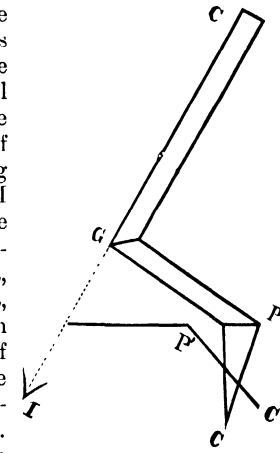



FIG. 32.—Diagram illustrating the production of kyphotic pelvis. G, angle of kyphosis; PP, promontory of sacrum; CC, tip of sacrum.

from side to side is increased in the kyphotic pelvis. The traction on the upper part of the sacrum leads to a rotation of the bone about a horizontal axis, so that while its base is displaced

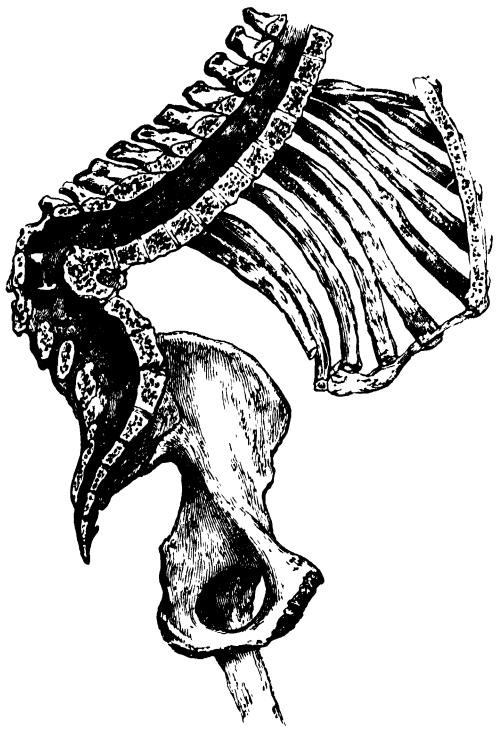


FIG. 33 —Kyphotic pelvis. (After Barbour.)

backwards its apex is moved forwards, thus lessening the antero-posterior diameter of the outlet; the movement upwards and backwards of the base of the sacrum pulls on the ilium,

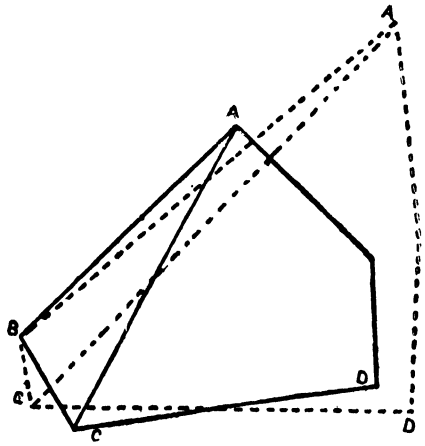



FIG. 34.—Diagram of cavity of kyphotic pelvis in sagittal plane: continuous line, normal pelvis; dotted line, deformed pelvis. AB, true conjugate; AC, diagonal conjugate; CD, antero-posterior diameter of outlet.

and makes the curve of the os innominatum not so sharp; the lessened inclination of the pelvic brim to the horizon causes increased strain on the ilio-femoral ligament. These pull the anterior inferior iliac spines down and out, and so rotate the ossa innominata about an axis running from before backwards. This rotation widens the space between the iliac crests and approximates the ischia. Hence the main changes from an obstetrical point of view are lengthening of the antero-posterior diameter at the brim, slight widening of the transverse measurements at the brim, considerable narrowing of the transverse measurements at the outlet. How marked these changes are depends upon how low down the kyphosis is.

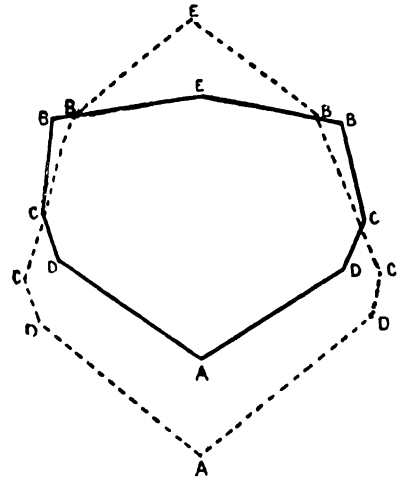



FIG. 35.—Diagram of brim of kyphotic pelvis: continuous line, normal pelvis; dotted line, kyphotic pelvis. A, symphysis; BB, sacro-iliac synchondroses; CC, transverse diameter; DD, ilio-pectineal eminences; middle of sacrum in plane of brim.

Kyphosis of the spine is a common disease; but a well-marked kyphotic pelvis is not common, because for its production it is necessary that the disease should begin early in life, and be situated low down.

The diagnosis is easy because the condition is suggested by the patient's short stature and crooked back. When the curvature of the spine has been discovered the pelvic outlet should be measured; those of the brim are difficult to take, and as if altered they are increased, it is not important to make them. The ease or difficulty of labour depends upon how much the outlet is contracted.

Alike in head, breech, and transverse presentations, dorso-posterior positions are more frequent with kyphotic than with normal pelves. The abdominal concavity of the child adapts itself to the lumbar convexity of the normal spine. The dorsal convexity of the child fits the concavity which kyphosis produces in the lumbar spine. If the deformity is not so great as to make delivery impossible, and the head presents with the occiput forwards, the only difference the kyphotic pelvis produces is that the increasing approximation of the sides of the pelvis as the head moves down makes the occiput turn forwards earlier than it does in normal labour.

When the occiput is behind and the deformity great, the narrowing of the front of the pelvis often prevents the occiput from turning forwards, and the head, if it be small enough, is born through the space bounded by the ischia in front and the coccyx behind, the sagittal suture lying in an oblique diameter.

The treatment of labour with a kyphotic pelvis should be guided by the same principles as in other forms of contracted pelvis. The index for treatment is the transverse measurement at the outlet. If this is so contracted that a living child cannot be drawn through it, early Cæsarean section should be done. Symphysiotomy is not here of much use, because the separation of the ischia which it allows is but slight. If the transverse at the outlet exceed three inches, and the child is of not more than average size, it can probably be born alive; and if the pains are so weak that help is needed, it should be given with forceps. Turning is no advantage.

*The Kypho-scolio-rachitic Pelvis.*—This is the pelvis produced when caries of the spine low

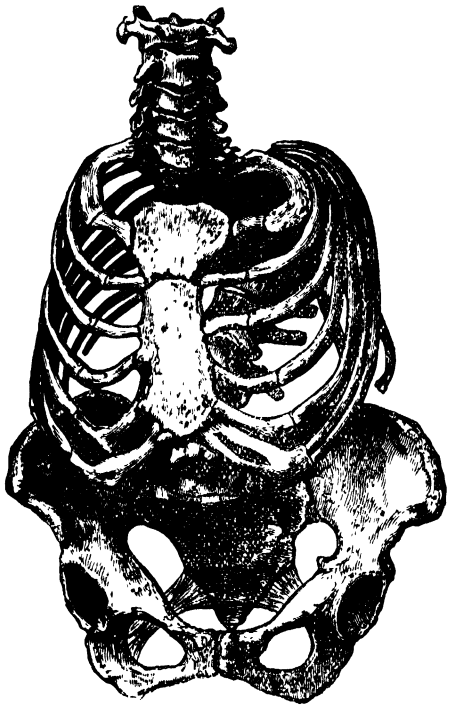


FIG. 36.—Kypho-scolio-rachitic pelvis. (After Leopold.)

down occurs in a rickety subject who has a lateral curvature of the spine. The rickety pelvis and the kyphotic pelvis are almost the exact opposite of each other; this pelvis is, as it were, a compromise between them. As in the kyphotic pelvis, the sacral promontory is drawn up and back, and the tip tilted forwards; but the sacrum presents the rickety convexity from side to side, and the thickening of the epiphyseal lines The general shape of the pelvis is funnel-shaped, like that of the kyphotic pelvis. The antero-posterior diameter of the brim is lengthened, and the transverse slightly diminished, and the ilio-pectineal line is longer and straighter. The transverse diameter at the outlet is diminished. The scoliosis leads to asymmetry of the pelvis. The sacrum is pushed

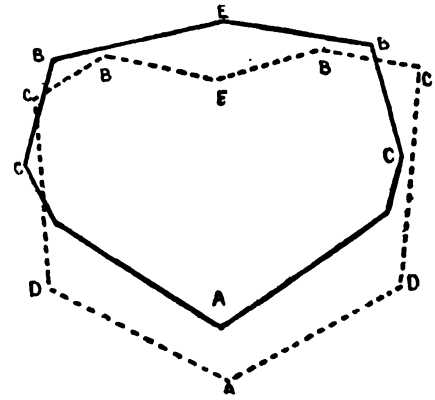



FIG. 37.—Diagram of brim of Fig. 36: continuous line, normal pelvis; dotted line, deformed pelvis. BB, sacro-iliac synchondrosis; E, centre of sacrum in plane of brim; CC, transverse diameter; DD, pectineal eminences; A, symphysis pubis.

towards the side of the lumbar convexity, and therefore the sacro-cotyloid diameter on that side is shortened. On the opposite side the upward and outward pressure of the femur acts to greater advantage, and therefore the symphysis pubis is pulled over to that side. The degree of these changes depends upon the

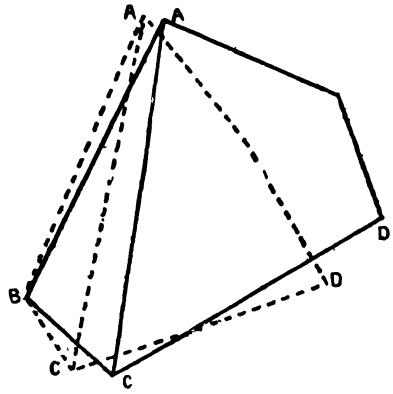



FIG. 38.—Diagram of cavity of Fig. 36: continuous line, normal pelvis; dotted line, deformed pelvis. AB, true conjugate; AC, diagonal conjugate; CD, antero-posterior diameter of outlet.

extent and situation of the spinal curvatures producing them. According to whether the kyphosis or the scoliosis is the more marked, and whether the kyphosis is low down or not, the pelvis will approximate to either the scoliotic or the kyphotic type. The diagnosis of this form of pelvis will be suggested by the spinal curvatures present, and will be completed by measurement of the pelvis. According to whether the pelvis approaches more nearly the

kyphotic or the rachitic type, so the treatment must be guided by the principles governing treatment in the kyphotic and the rachitic pelves respectively.

*The Osteomalacic Pelvis.*—In this pelvis, as in the rickety pelvis, the deformity is due to softening of the bones, so that they yield to pressure and pulling. The conditions of its production differ from those of the rickety

FIG. 39.—Osteomalacic pelvis.

pelvis, in that it occurs in adults, in whom the muscles are stronger and more used than in rickety children; and that the softening is greater than in rickets. The consequence is that muscular action affects the shape of the pelvis more with osteomalacia than it does with rickets. The muscles pull out the pubes and ischia, and pull in the head of the femur. As soon as the head of the femur is within the line passing from the sacrum to the feet, the femoral pressure reacting to the body weight becomes upwards and inwards, instead of upwards and outwards; and then it combines with the action of the muscles to crumple in the acetabula. Hence the pelvis becomes "rostrate," the two pubic bones running nearly parallel so as to project forwards like a beak. The acetabula are approximated to the sacro-iliac synchondrosis, so that the pelvic canal becomes somewhat the shape of a **Y**. The sacrum yields to the body weight, and is pressed down, as in rickets, but more; the promontory is often so sunken that the fifth or even the fourth lumbar vertebra may come to lie in the plane of the pelvic brim. As in rickets, and for the same reason, the sacrum becomes convex from side to side, but more so; the curve is so great as to appreciably narrow the bone. While the sacral promontory is pushed down, the tip of the sacrum is prevented from moving back by the sacro-sciatic ligaments; hence the sacrum becomes sharply curved from above downwards. The pull of the sacrum upon the ilium at the synchondrosis, combined with the upward and inward pressure of the femur upon the acetabulum, crumples up the ilium until the iliac fossa becomes like a gutter. The sacrum and ilium may get separated at the synchondrosis. When the disease is advanced the bones become so soft that the patient cannot stand or walk, but lies or crouches in various attitudes; hence different distributions of pressure in different patients, and corresponding minor differences in the shape of different osteomalacic pelves. Although all osteomalacic pelves conform to the same general type, yet they do not exactly resemble each the others, as do pelves of the Naegelé or the Robert type.

Osteomalacia begins during pregnancy or lactation. There is severe pain, especially on movement. The spine and ribs are soft as well as the pelvis; from this they become bent, and the capacity of the chest is diminished. Hence the lungs cannot properly expand, and the patient suffers from cough, shortness of breath, suffocative attacks, and muscular cramp. There is an excessive excretion of phosphates in the urine. When the disease has been cured, this ceases. The cure of this disease is by removal of the ovaries. We know not how this acts, but the fact is established. The disease is rare in England; endemic in certain parts of Europe.

The diagnostic points of osteomalacia while it is progressing are: (*a*) the tenderness; (*b*) the softness of the bones. The pelvic deformity is so extreme that measurement is not needed to detect it.

Obstetrically the osteomalacic pelvis is unique among contracted pelves in this, that it has been found possible, so soft may the bones be, to force the pelvis open with a hand to a degree sufficient to allow a living child to pass. But it is hardly worth while to do this, because the patient must, for her cure, have her abdomen opened. The best treatment is to perform Cæsarean section, and then remove the body of the uterus and the ovaries. This done, the patient's pains will cease, the phosphates in her urine diminish, and the bones will get hard. The deformity will never be removed.

There is a rare form of contracted pelvis known as the *pseudo-osteomalacic rickety pelvis.*

FIG. 40.—Pseudo-osteomalacic rickety pelvis. (After Naegelé.

The shape of this pelvis is like that of the osteomalacic pelvis (only not to the same extreme degree)—that is, the acetabula are crumpled in so as to make the pelvic cavity **Y**-shaped,

pushes the top of the sacrum backwards. This tends to narrow from before backwards the canal of the sacrum. The pushing of the sacrum back separates the posterior superior iliac spines (see Fig. 43). There is extreme lordosis of the lumbar spine, so that the front edge of the bodies of the vertebra are farther apart than they ought to be, while the neural arches are pressed together. This pressure may lead to bony outgrowths, ossification of the ligaments, and, finally, synostosis. The inclination of the pelvis to the horizon is diminished. This throws increased strain on the ilio-femoral ligaments; the pull of these ligaments rotates each os innominatum about an antero-posterior axis, so that the upper part of the bone is turned outwards, the lower inwards. Hence, as in the kyphotic pelvis, the transverse diameter at the brim is widened, that at the outlet narrowed.

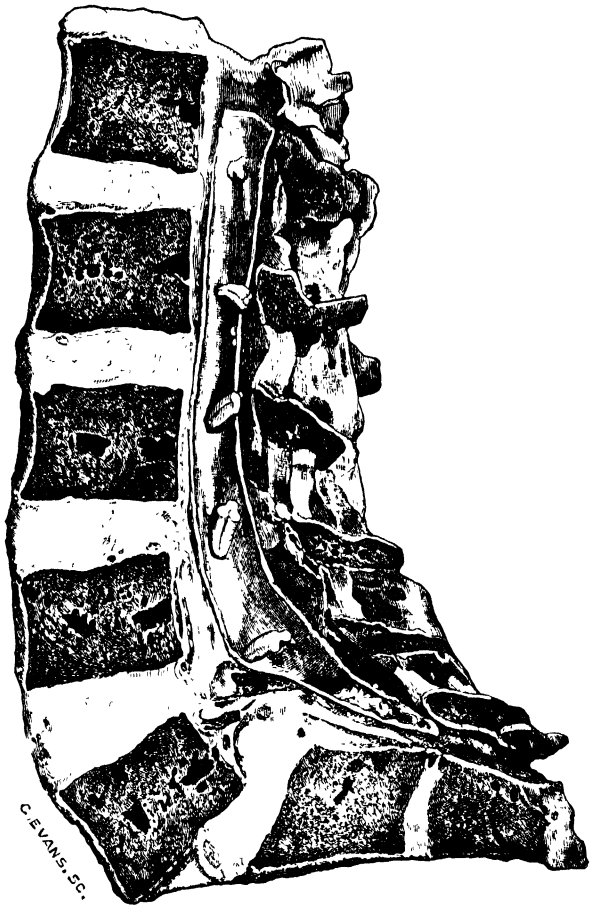



FIG. 43.—Early stage of spondylolisthesis (After Targett.)

The changes described above are seen in different degrees in different pelves. The ossific defect may be on one side only, and then the vertebra will slip down on that side more, and the deformity produced will be asymmetrical. But the disease is so rare that it is not worth while to dilate upon the differences in degree and in symmetry.

In the *diagnosis* of spondylolisthesis investigation has to be made along three lines. First, the history. This will be of some violence or strain, leading to a long illness, attended with pain in the lower part of the back, and severe enough to keep the patient in bed. The usual date of this illness is from the fifteenth to the eighteenth year. Second, the shape of the body. The patient is short, and this is seen to be due to shortening of the lumbar spine. The distance between the ribs and the pelvis is diminished; the ribs may even be sunk into the false pelvis. This makes conspicuous the distance between the wings of the ilia. The posterior superior iliac spines are farther apart than usual. The back of the sacrum is plainly felt. From the less inclination of the pelvis the external genitals look more forward and less downwards than usual. The patient walks with short steps, and with the feet slightly inverted, so that the marks made by the feet are wanting in breadth. Third, vaginal examination. The displaced lumbar vertebra is felt narrowing the brim. It is distinguished from the projecting promontory of a rickety pelvis by the fact that at its sides nothing like the lateral masses of the sacrum can be felt; and also that by external examination the sacrum can be felt not to be displaced. A distinct angle between the displaced vertebra and the sacrum cannot be felt, because this angle is filled up with new bone. As in the kyphotic pelvis, the distance between the ischial tuberosities is lessened, and the tip of the coccyx extends farther forwards than usual.

When caries of the last lumbar vertebra and top of the sacrum has been present, the angular curvature produced leads to the lumbar vertebræ overhanging the brim of the pelvis somewhat as the last lumbar vertebra does in spondylolisthesis. This deformity is called *spondylizema*. This and spondylolisthesis have been classed together under the common name of the *pelvis obtecta*.

The treatment of labour with spondylolisthesis depends upon the length of the obstetrical conjugate. It is possible that cases may be met with in which deformity is so slight that delivery can be effected by forceps or turning; but in most—in all which deserve the term pelvis obtecta—Cæsarean section is the proper treatment.

*The Split Pelvis.*—In this deformity the symphysis pubis is absent. The two halves of the pelvis not being bound together in front, the upward and outward pressure of the femora forces them widely apart, so that there is a wide

gap between the pubic bones, which are united only by some fibrous tissue. The ossa innominata are rotated about an axis parallel with the axis of the pelvic brim, so that the posterior iliac spines approach one another. This shortens the distance spanned by the ligaments which suspend the sacrum from the ossa innominata, and the sacrum is therefore allowed to slip forwards and downwards. This approach to one another of the posterior iliac spines, and sinking downwards of the sacrum,

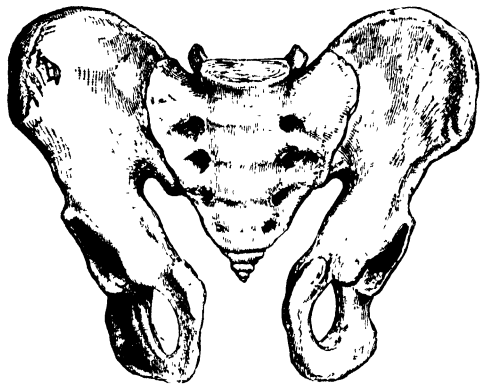


FIG 44.—Split pelvis.

reaches in extreme cases such a degree that it looks as if there was a canal behind the sacrum instead of in front of it; and this has caused the name "inverted pelvis" to be applied to it. The general shape of the pelvic canal is that of an extreme type of rickety pelvis, but with a large gap in front. It is almost always associated with extroversion of the bladder; and from the disgusting nature of this deformity pregnancy with this pelvis is rare—only seven cases have been recorded. In the management of labour with this deformity the choice is between turning and Cæsarean section, according to the size of the child and the antero-posterior measurements of the pelvis.

There are two causes which may deform the pelvis—*fracture* of the pelvic bones, and *hip disease*, in which the deformity cannot be said to conform to any type. In the former, the nature of the injury and the position of the fragments during union regulate the shape of the pelvis; in the latter, it depends upon the extent of the disease, the age of the patient, and the presence or absence of dislocation. All that can be said is, that old hip disease generally in some way modifies the shape of the pelvis.

There are other kinds of pelvic deformity, for instance, that due to congenital dislocation of the femora, which narrow not the pelvis, and, therefore, obstruct not labour. A pelvis called the fœtal or lying-down pelvis has been described; but no case of pregnancy with it has yet been known. These pelves are obstetrically not important, although from other points of view they may be very interesting.

I shall now describe more in detail the methods of measuring the pelvis, and the application of such measurements to the management of labour.

### PELVIMETRY

The existence and degree of pelvic contraction are found out during life by *pelvimetry*. There are two kinds of pelvimetry, external and internal.

1. *External pelvimetry* is done with callipers, the best for the purpose being those sold under the name of Matthews Duncan's. The essential features of the instrument are that the points should be blunt, so that they hurt not the patient; that the limbs be large enough and curved enough to embrace half the pelvis; and that a measuring scale be attached so that the distance between the points can be read off without trouble. The external measurements usually (and easily) taken are three:—(1) *The anterior interspinous*, which is the distance between the anterior superior iliac spines. It may be measured either by putting the points of the callipers outside each bony point, or by applying the thumbs to the inner side of the spines, and by then feeling that the points of the callipers are level with the inner borders of the iliac spines. I think the latter is the more accurate method. The method adopted makes a difference of an inch or more in the measurement obtained, the distance between the inner borders being less than that between the outer. It averages about 10 inches, but varies from 8 to 12 inches. (2) The *intercristal*, or the distance between the most distant points of the iliac crests. This is obtained by putting the points of the callipers on the outside of the crests and moving them about until the greatest separation between them is reached. This measurement averages about 11 inches, but varies from 10 to 14 inches. These measurements have but little practical importance, and would not be worth making if making them caused discomfort to the patient. They show roughly the width of the pelvis, but their relation to the internal transverse measurements varies so much that no inference can be drawn unless the measurements differ extremely from the normal. The due proportion between the two measurements shows a normal curve of the iliac crests. An altered relation, so that the interspinous is as great as the intercristal, shows that the ilia look more forward than they should do, and that the pelvis is flattened; but no inference can be drawn from an altered proportion so slight as to need measurement for its detection. (3) The *external conjugate*, which is measured from the depression below the last lumbar spine to the most distant point on the

front of the symphysis pubis. The last lumbar spine is usually to be found about an inch above the line joining the posterior superior iliac spines. This diameter averages in thin women about 7½ inches. It was at one time supposed that there was a constant relation between the external and the internal conjugate, that by deducting 3 inches from the former the length

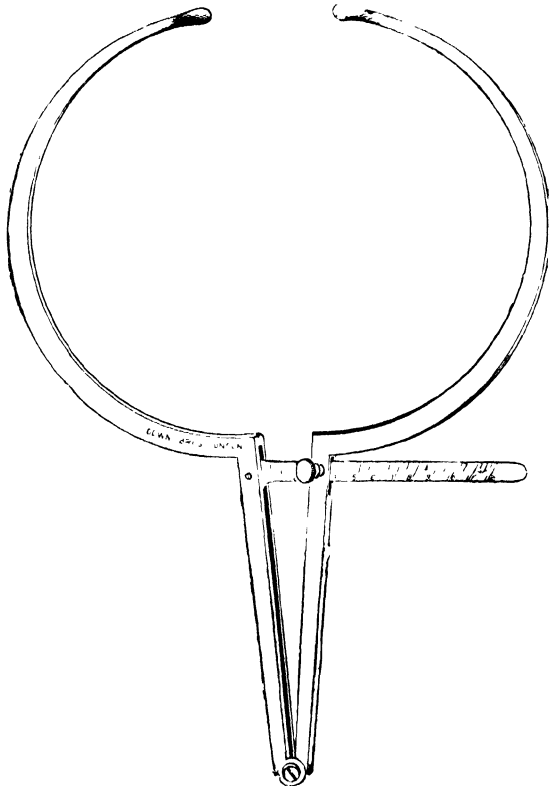


FIG. 45.—Duncan's callipers.

of the latter might be ascertained. This has been carefully tested and found not to hold good. The difference between the two conjugates varies from 3 to 4½ inches. Hence an external conjugate of 7½ inches is no guarantee that the pelvis is not contracted. On the other hand, when the internal measurements are normal, if the patient be thin and her bones slender, the external conjugate may be slightly less than 7½ inches; but if the external conjugate is less than 7 inches, it is certain that the internal conjugate is contracted.

Some other external measurements are not so easily made. When the patient is not pregnant, and if she is not too fat and will relax her abdominal walls, the true conjugate can be measured by "Hardie's method." This consists in depressing the anterior abdominal wall until the promontory is felt, and then measuring the distance from the promontory to the top of the pubes. This cannot be done accurately, for the posterior end of the measurement is not the promontory, but the promontory plus the thickness of the abdominal wall; and the anterior end of the measurement, the top of the symphysis, is not the nearest point to the promontory. It is thought that in women with abdominal walls of ordinary thickness these two inaccuracies about neutralise one another; this may be so, but the existence of these inaccuracies prevents this mode of measurement from being more than an approximation. Still, it is in some cases a useful approximation, and can be used as a "control experiment" to measurements otherwise obtained.

In some pelves, the kyphotic and the funnel-shaped pelvis, it is important to measure the *transverse diameter at the outlet*. This is difficult to do, because the bony points, the distance between which we want to know, viz., the tubera ischiorum, are covered with such a thickness of soft parts that it is impossible to get any measuring instrument directly on to them. The best way of doing it, in my opinion, is to make the patient kneel on her elbows and knees, and then, feeling the position of the ischia with the fingers, to mark their outline on the skin of the buttocks, and then measure the distance between the markings. It is more difficult than would be expected to mark out accurately the position of bones which lie so deep; but it is obvious that such error as may occur in deliberately marking out the outline of the bones is more likely to happen if the measurement is made, or attempted to be made, without first marking the skin.

In cases of *oblique deformity* of the pelvis measurements should be taken from the posterior superior iliac spine on each side to the anterior superior iliac spine of the opposite side, and from the hollow below the last lumbar spine to the anterior superior spine on each side. This will enable the amount of deformity to be estimated; but a degree of obliquity that cannot be detected without this measurement is not of great importance.

The distance between the *posterior superior iliac spines* may be measured; it is usually about one-third of the distance between the anterior superior iliac spines. In the flat pelvis the posterior spines are abnormally approximated and the anterior abnormally separated, and therefore the difference between the posterior and anterior interspinous measurement is increased. But in a pelvis so much flattened that this diminution in the proportion of the posterior interspinous to the anterior is marked, the deformity will be more easily and accurately ascertained in other ways. In fat women it is not easy to feel the posterior superior iliac spines.

II. *Internal pelvimetry* is that which is really important, but it is also more difficult and is very disagreeable to the patient. It is the measurement of the diameters of the pelvic canal. Instruments have been made for this purpose, consisting of variously shaped metal

rods with knobs at their ends, which are intended to be applied to different points in the pelvic canal, so that the distance between the points may be measured. These answer excellently in the dried pelvis; but when the neophyte tries to use them on the living patient he finds that the pelvis contains a bladder and rectum, besides a uterus and vagina and a good deal of fibrous and muscular tissue, and that these parts are resistant and sensitive, so that it is often difficult to feel with the fingers the points between which measurement has to be made, much more to get metal knobs into position and hold them in position while distances are being measured. Internal pelvimeters are, for this reason, practically useless. The best pelvimeter is the hand, and the time when the pelvis can be exactly measured is immediately after delivery. How to do this was first accurately and clearly described by Mr. Robert Wallace Johnson in his *System of Midwifery* published in 1796. The proceeding should, therefore, in justice be spoken of as "Johnson's method" of pelvimetry. It consists in introducing the whole hand into the pelvis, and noting the part of the hand which fills the pelvis in the diameter which it is wished to measure. The following measurements (given by Mr. Johnson) are those of a man's hand of average size; they should be tested and corrected, if necessary, by measurement of the hand of the operator.

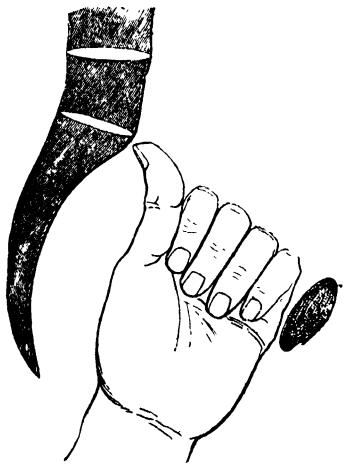


FIG. 46.—Direct pelvimetry, measurement 4 inches.

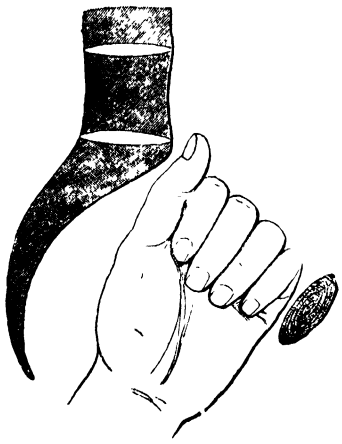


FIG. 47.—Direct pelvimetry, measurement 3½ inches.

1. The fingers being bent into the palm, and the thumb extended and applied close to the middle joint of the forefinger, the distance between the end of the thumb and the outside of the middle joint of the little finger is 4 inches (Fig. 46).

2. In the above position the distance from the thumb at the root of the nail, in a straight line to the outside of the middle joint of the little finger, is 3½ inches (Fig. 47).

3. The fingers being in the same position, and the thumb laid obliquely along the joints next the nails of the first two fingers and bent down upon them, the distance between the outside of the middle joint of the forefinger and the outside of that of the little finger is 3¼ inches (Fig. 48).

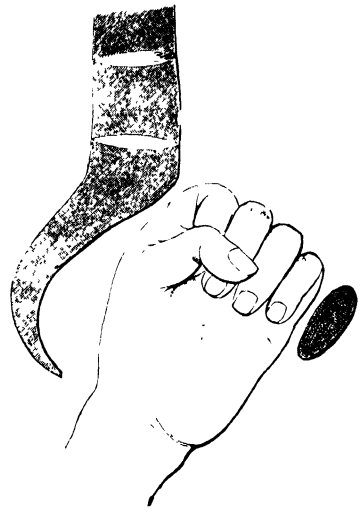


FIG. 48.—Direct pelvimetry, measurement 3¼ inches

4. The hand being opened and the fingers held straight, the whole breadth from the middle joint of the forefinger to the last joint of the little finger is 3 inches (Fig. 49).

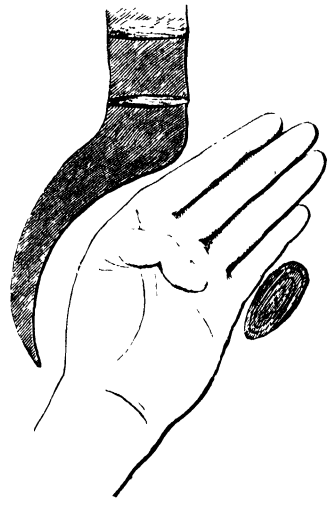


FIG. 49.—Direct pelvimetry, measurement 3 inches.

5. The fingers being so far bent as to bring their tips to a straight line, their whole breadth across the joint next to the nails is 2½ inches (Fig. 50).

6. When the first three fingers are thus bent their breadth across the same joint is 2 inches.

7. The breadth of the first two across the nail of the forefinger is 1¼ inches.

In any case in which labour has been difficult, the length of the obstetrical conjugate should

be measured during or after the third stage of labour in the way just described. If it is less than 4 inches, it can be measured by Johnson's method more accurately than in any other way; if it is more than 4 inches, its precise length ceases to be important. As the transverse measurement at the brim usually exceeds 4 inches, it can seldom be estimated in this way; but any measurement at the brim that is less than 4 inches can be taken in this way as accurately as the conjugate. It is difficult to measure the transverse at the outlet by Johnson's method, because the resistance of the perineum is so great; but if there is reason to believe this diameter contracted, its internal measurement should be attempted. In the slighter degrees of pelvic deformity, when the head is presenting at the brim, Johnson's method cannot be applied before delivery; but if neither the head nor the breech is presenting, or if the pelvic deformity is so great that the head cannot at all sink into the pelvis, Johnson's method can and ought to be applied before delivery with the assistance of anæsthesia. An experienced person may fall into error from failing to get his hand into the smallest diameter of the brim; but this is a mistake that a little care will guard against, and it is the only source of fallacy which attends Johnson's method of pelvimetry.

FIG. 50.—Direct pelvimetry, measurement 2½ inches.

It is often desirable to know the length of the obstetrical conjugate in cases in which Johnson's method is inapplicable before delivery. In that case the only way is to measure the *diagonal conjugate*, and infer from it the length of the obstetrical conjugate. This can be done either in the customary left lateral position or in the dorsal position. In the former position the left hand must be used; in the dorsal position either hand may be employed. With the patient on her left side, the middle and index fingers of the left hand must be introduced into the vagina and pressed up until the middle finger feels the sacral promontory. One finger cannot reach far enough for this. The difficulty in reaching the promontory lies in the resistance of the perineum, which must be pressed up by the knuckles of the third and fourth fingers, and this pressure on the perineum is painful to the patient. The amount of pressure exerted depends on the length of the diagonal conjugate and the thickness and firmness of the pelvic floor. One who is regardless of the pain he causes can feel the promontory in almost any woman; but it is in practice not needful to much hurt the patient, for if the promontory cannot be reached without very forcible upward pressure it may be safely concluded that the conjugate is not much, if at all, shortened, and its exact measurement need not be taken. When it is so contracted that its exact measurement is important, it can easily be felt. When the tip of the middle finger is in contact with the promontory the back of the right forefinger should be applied to the front of the pubic symphysis, and held at right angles to the radial border of the left index finger and palm, so that the nail of the forefinger marks the spot at which the left examining hand touches the pubic symphysis. Then the hands are removed, with the right forefinger still in contact with the left hand, and the distance measured from the right forefinger nail to the tip of the left middle finger. This distance is the diagonal conjugate; it forms one side of a triangle, the other side being the symphysis pubis and the true conjugate. The length of the symphysis pubis is easily measured, and if we could as easily measure the angle which it forms with the diagonal conjugate, we could then from these

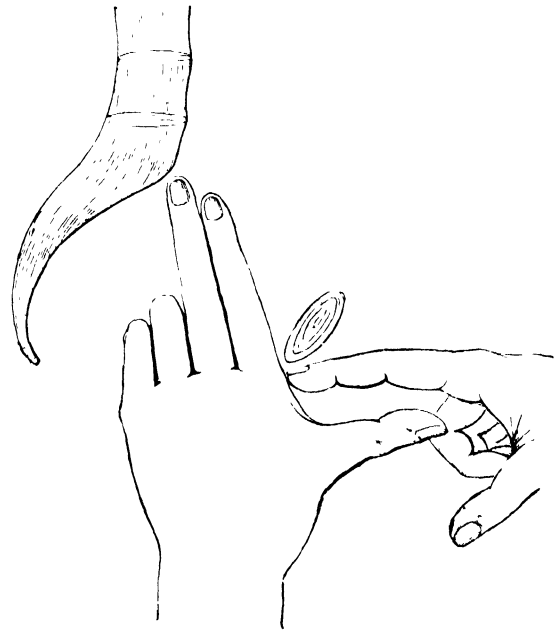


FIG. 51.—Mode of measuring the diagonal conjugate

data exactly calculate the length of the true conjugate. But to measure the inclination of the symphysis pubis requires special apparatus, an assistant, and an amount of exposure and manipulation of the patient that make this

measurement impracticable in ordinary practice. We are therefore obliged to be content with deducting from the diagonal conjugate the average difference between it and the true conjugate, which is a little more than half an inch, and thus inferring the true conjugate. It may seem as if we ought not to be content with so rough an approximation; but it must be borne in mind that the measurements themselves are only approximations, the points between which we have to measure and the measuring instruments (the fingers) are so indeterminate that it is impossible to get nearer than within about a quarter of an inch—that is to say, that different competent observers measuring the same patient, or a competent observer measuring the same patient on different occasions, will get results differing by at least a quarter of an inch from one another.

The accoucheur ought not in advising his patient to depend upon one kind of measurement alone. If consulted during pregnancy, he should take the external measurements and the diagonal conjugate; then judge of the size of the child by palpating the belly, measuring its girth, and the height of the uterus above the symphysis pubis; and, finally, estimate the relative size of the child and pelvis by trying how far he can press the head of the child down into the pelvis. After delivery, for sure guidance in future labours, he should accurately measure the true conjugate by Johnson's method, and check the result obtained by examination of the child's head. He should note any dints, grooves, overriding of sutures, red stripes on the skin, or other evidence of compression, and measure the diameter of the head where pressure has evidently been operative. From the information so obtained he will be able to advise the patient with precision as to the mechanical difficulties to be anticipated in subsequent labours, and as to the best mode of delivery. Midwifery can only be regarded as a branch of medical *science*, whence its practice is governed by a knowledge of the size and shape of the pelvis, the size of the child, especially of its head, and the movements which it ought to make in order to pass easily through the pelvis. Practice without this knowledge is not science, but rule of thumb. The treatment of difficult labour by persons who have not this knowledge is simply to lay hold of the child with instruments or hand, and pull till either the child comes out or the operator is exhausted. In the latter event he probably sends for assistance; and the person whose aid is sought finds a patient irreparably damaged, and so ill that an operation by which she might have been safely delivered early in labour, has become attended with extreme danger.

### The Treatment of Labour with Contracted Pelvis

Take first the most extreme case, a pelvis with a true conjugate of *not more than* 2 *inches*. Here there is no doubt as to the proper treatment. Cæsarean section is the only mode of delivery. It is true that in the past, when Cæsarean section was terribly dangerous, expert handlers of the cranioclast, vertebral hook, crotchet, and scissors have broken up and extracted a child through a pelvis with a conjugate a trifle less than 2 inches; but such operations are long and difficult, and entail a risk to the mother as great as that now attached to Cæsarean section. There is no longer occasion for such operations.

Deformity of the pelvis so great as this is, is usually accompanied with visible deformities of other bones; and therefore it is possible that the possessor of such a pelvis may suspect that her pelvis is misshapen, and be wise enough to consult her doctor before she marries, before she becomes pregnant, or in the early months of pregnancy. The suggestion may then be made that the necessity for Cæsarean section may be averted by the early induction of abortion. But multiple pregnancies ending in abortion cannot be gone through without a little risk, not to speak of the deterioration of the patient's comfort and happiness by the repeated necessity for fruitless operations. Cæsarean section, followed by sterilisation, seems to me preferable. It may also be suggested that the necessity for either abortions or Cæsarean sections may be avoided by the use of precautions to prevent pregnancy, such as are commonly employed in another country with a decreasing population. But these precautions are nasty; they often fail, and their prolonged use tends in many women to injure the nervous system, as is illustrated by the complex forms of hysteria common in the country to which I have referred, but happily rarer in England. But further, every woman with a healthy mind is fond of children. Even if she before pregnancy desired not a child, she will love and value it when she has got it. If treatment gives the patient a strong and healthy living child, it gives her the greatest treasure she can have. A patient may be so fond of children that she will be willing more than once to incur the risk of Cæsarean section, and if so, her wish ought not to be opposed. I think a patient cannot reasonably be expected against her wish to incur repeatedly a risk so much greater than that of natural delivery, and that therefore if the patient wishes that after Cæsarean section further pregnancy should be made impossible, it is proper to comply with this request.

Take next a pelvis having a conjugate diameter of *more than* 2 *inches, but less than* $2\frac{3}{4}$ *inches*, and without appreciable shortening of the transverse diameters. Here delivery by cephalotripsy, done by a skilful operator, in a patient not exhausted by protracted labour, and

in a place in which asepsis can be secured, is attended with no greater risk than that of labour at term. The immediate prognosis for the mother is therefore better if she is delivered by cephalotripsy than if Cæsarean section is done. The objections are (1) that the child's life is sacrificed, and the mother deprived of the happiness of maternity; and (2) if we look further into the future than the days of childbed, is the prognosis so much better for the mother? If she lives the life of a healthy married woman she will probably have pregnancy after pregnancy, each with its discomforts and dangers; and the sum of these dangers to her life (not to speak of the comfort of her life) will probably be as great as that of one Cæsarean section. I think that from the point of view of the mother's life Cæsarean section, followed by sterilisation, is to be preferred to cephalotripsy.

Consider next a pelvis with a conjugate of *from* $2\frac{3}{4}$ *inches to* $3\frac{3}{4}$ *inches*, and not appreciably contracted in the transverse diameters. Assume that the patient has been wise enough to consult you early in pregnancy. There are two alternatives. One considers solely the immediate interests of the mother. It is to induce labour before the child is too large to come through the pelvis. If this is done early enough the mother will have an easy labour, but the child will be puny and difficult to rear. The time to choose for the induction of labour is a compromise. The earlier the labour is induced the easier it is; the later it is postponed the stronger the child. Therefore it should be done at the very latest time at which it is possible for the child to pass through the pelvis.

Measure the pelvis, and measure the uterus. The greatest girth at the full term of pregnancy in a patient who is not fat, dropsical, or the possessor of a tumour, and whose uterus contains a child of average size, with an average quantity of liquor amnii, should not exceed 1 yard—36 inches. The measurement in such a case from the symphysis pubis to the top of the uterus, over its convexity, averages 13 inches. At seven months' pregnancy these measurements should be less. If the patient thinks herself only seven months pregnant, and yet her measurements approach those of an average full-term pregnancy, there is need for investigation as to the cause of her excessive enlargement. Palpate the abdomen, and find out where the fœtal head is. If it is not over the pelvic brim, perform external version, if possible, and get it over the brim. When the head is over the brim, try how easily it can be pressed down into the brim. If it can be pressed down easily into the brim, tell the patient to come again in two, three, or four weeks' time, according to the ease with which the head could be pressed down into the brim. As soon as the head just fills the brim tell the patient that the time has come to induce labour. This is the way by which the mother gets more safely and easily over pregnancy and labour, but not the way by which the birth of a strong and healthy child is best secured.

If the mother is willing, in order to have a strong and healthy living child, to incur a little more risk, you can add half an inch to the conjugate diameter by symphysiotomy. This operation, if done in suitable cases, and by the subcutaneous method, is almost without risk. The ill results that have occasionally followed symphysiotomy are either preventable, such as hæmorrhage and septic infection—risks almost abolished by the subcutaneous method of operating; or they have occurred in unsuitable cases—those in which the pelvis was so small, or the child so large, that it could not be pulled through the pelvis without excessive separation of the pubic bones. If the pubic bones are pulled farther apart than 2 inches there is risk of damage to the soft parts below them—urethra and bladder—and to the sacro-iliac articulation. Before symphysiotomy is undertaken the patient should be examined with as much care as before the induction of premature labour, and the relation of the equator of the head to the shortest diameter of the pelvis estimated. If the former diameter exceeds the latter by more than half an inch, symphysiotomy is not suitable.

When the head presents not, the relative size of the head and the pelvic brim cannot easily be determined. In this case try to turn the child by external or bimanual manipulation, and get the head over the brim. The possibility of this depends upon whether there is enough liquor amnii to enable the fœtus to move freely. If you cannot do this, the only guide as to the possibility of delivering a living child by symphysiotomy is the measurement of the pelvis and of the uterus. Measure the diagonal conjugate, and deduct half an inch from it to get the true conjugate. Measure the greatest girth, and the distance from the symphysis to the top of the uterus, measured over the anterior convexity of the uterus. If the girth exceeds not 36 inches, and the distance from pubes to top of uterus exceeds not 13 inches, you may safely assume that the child is not larger than the average—and may be smaller.

The objections to symphysiotomy are:—First, the immediate risk to life from (*a*) hæmorrhage, (*b*) septic poisoning. These risks attend every operation in which a large wound is made; but they are preventable; and if symphysiotomy is done by the subcutaneous method, and with a clean knife, they practically cease to attend it. Secondly, the risk of impaired power of locomotion from imperfect union of the symphysis. The experience of Ahlfeld and others shows that even when the two pubic bones are only united by fibrous tissue (and it is doubtful whether they ever unite in any way) and remain

separated by a larger interval than before the operation, the patient can nevertheless stand and walk well. In some cases symphysiotomy has permanently so enlarged the pelvis that the patient has been naturally delivered afterwards. The cases in which permanent lameness has followed have been those in which the two pubic bones have been separated excessively, so that the ilio-sacral articulation has been injured. Thirdly, the possibility of injury to the bladder or urethra, resulting in persistent want of control over the bladder. This ill consequence is liable to follow when the pubic bones are too widely pulled apart, and the soft parts between them too much stretched and consequently torn. It is to be prevented by estimating the relative size of the fœtal head and the pelvis before deciding on the method of delivery, and choosing symphysiotomy only if it is certain that the head can pass through the pelvis after this operation. Fourthly, the longer time during which the patient has to lie in bed after symphysiotomy; four weeks, as opposed to two weeks after natural delivery. There is really little in this objection; because, although a woman can get up two weeks after natural delivery, few women can fully discharge their household duties so soon. The time from delivery to restoration of full working power is about the same whatever the method of delivery, if the method chosen is the right one.

*Slight Pelvic Contraction.*—Consider, lastly, the case of a patient whose pelvis is not so much contracted that it can be said at once that an operation of some kind is necessary for delivery. Though the pelvis is under normal size, the disproportion between the pelvis and the head is not so great as to put natural delivery out of the question. The first point is to take greater care than usual to prevent premature rupture of the membranes. When the patient is upright the weight of the amniotic fluid helps the uterine contractions to burst the membranes; therefore the patient should be kept on her side in the semi-prone position. In this position the weight of the waters no longer is added to the force of the uterine contractions. If the patient can be got to rest on her knees and elbows, the weight of the waters opposes and partly neutralises the effect of the uterine contractions. The patient should be told not to strain. The attendant should be careful in examining not to injure the membranes. Next, it is needful to bear in mind the difficulty which the head has in engaging in a contracted pelvis; the ease with which in such a pelvis it may get displaced, even if it were over the brim at the beginning of labour; and the liability, even if the head present, of its being forced into an unfavourable position, such as a face or brow presentation. Therefore care should be taken to see that the long axis of the uterus is as nearly as possible a continuation of that of the pelvic brim. Lateral obliquity of the uterus should be corrected by making the patient lie on the side opposite to that towards which the uterus leans. If there be pendulous belly, the patient should be put on her back and a firm binder applied. If the position of the child is still unfavourable it should be, if possible, corrected by external manipulations. Contraction of the pelvis brings with it no special tendency to abnormalities of the pains, but the effects of too weak or too strong pains are more serious than if bony obstruction to the passage of the child is absent. Weak pains, which with a pelvis and child of average size would have only made the labour long, will, if the pelvis be contracted, fail to make the head enter the brim. On the other hand, if the pains are too strong, danger of rupture of the uterus will arise early. Hence in contracted pelves the course of the labour must be watched with greater care than usual, that an abnormal course of labour may be early perceived and early treated.

If the membranes rupture early, as is often the case, before the os uteri is near full dilatation, and the head does not come into the os uteri to stretch it open, the best course is to artificially dilate the cervix with Champetier's bag. Then when the os uteri is fully dilated, the child can be delivered either by forceps or turning.

The entry of the head may be made easier by putting the patient in what is called "Walcher's position"; that is, in the dorsal position on rather a high bed so that the legs may hang down with the toes just touching the floor. This position extends the pelvis upon the spine, rotating it about a transverse axis passing through the sacro-iliac synchondroses, and thereby enlarges the conjugate by about from $\frac{1}{5}$ to $\frac{2}{5}$ of an inch. It at the same time diminishes the antero-posterior diameter at the outlet; so that there is no advantage, but the reverse, in the patient's retaining this position after the head has entered the pelvic cavity.

There has been discussion as to the relative merits of delivery by forceps and by turning when the head is presenting in a flat pelvis. With a small round pelvis, and the head presenting, if the head can enter the pelvis no one questions that if help is needed it should be given with forceps. But as to the best way of delivery in flat pelves, there has been a difference in the teaching of different schools. It has been pointed out, and is admitted, that the passage of the head base first is easier, because when the parietal bones are pressed together from below upwards the angle they form at the sagittal suture is made more acute, and the transverse measurements of the head are diminished (see Fig. 24). When the child is born head first the resistance to the advance of the

vertex tends not to diminish the transverse diameters of the head, excepting by the over-riding of the posterior parietal bone by the anterior, and by grooving and dinting of the bones, and these changes in the shape of the head may be produced whether it comes first or last (Fig. 52). To this it is replied that delivery with the feet first involves so much risk to the child's life, from pressure on the cord, etc., that the results are better when the child is delivered by forceps. Statistics show that this has been the case in some maternity charities. The explanation is, that more skill is required to deliver a child alive by turning than by forceps. Forceps delivery only needs hard pulling; but for the delivery of a living child by turning it is essential that the right time should be chosen, and that extraction should be skilful and rapid. If the accoucheur fail to recognise the right time for version and bungles extraction, the child will be dead. I think that in flat pelves delivery by podalic version, skilfully done, gives better results than forceps. For success, the bag of membranes must, if possible, be preserved until the os uteri is fully dilated; if this cannot be done, the os should be dilated with the water-bag of Champetier de Ribes. When this bag has been expelled, the child should be turned and the head quickly extracted by combined jaw and shoulder traction.

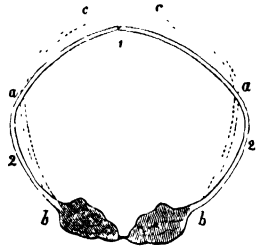



FIG. 52.—Change in shape of head produced by downward pressure with vertex in advance. Dotted lines, *aa*, *bb*, *cc*, normal shape of head; continuous lines, 1, 2, shape of head altered by pressure from above. (After Galapin.) (See also Fig. 24.)

There is one exception to the foregoing statement. If the head has engaged in the brim in the most favourable position for passing it, this state of things cannot be improved upon. The most favourable position is that in which the long diameter of the head is transverse, and there is so much Naegelé obliquity that the sagittal suture feels as if distant about ¾ of an inch from the sacral promontory. In this position the diameter which passes the brim is a transverse subparietal superparietal diameter, which is rather less than the biparietal, and is further diminished by the posterior-lying parietal bone being overlapped by the anterior, and being flattened by the pressure of the sacral promontory. Experience has shown that a head thus engaged in the brim can generally be delivered with forceps.

### Faults in the Soft Passages

Delivery may be delayed by *causes which prevent the proper dilatation of the soft parts*. By many writers (especially old writers) "rigidity" of the cervix, or, more briefly but incorrectly, "rigid os," has been described as a cause of lingering labour—a term which, as used, implies that there are cases in which delivery is morbidly delayed because a healthy cervix will not dilate; and various methods of treatment—drugs to be swallowed, medications to be applied, and manipulations to be performed—have been recommended, having for their object the more speedy opening up of the healthy cervical canal. It is rash to make negative statements, because they cannot be proved. But I must go as far as I can in this direction, and say that I have never seen a case of labour, otherwise normal, delayed because the cervix would not dilate; nor have I ever read a report of a labour described in such a way as to convince me that this was the case. I have read plenty in which it seemed to me that the only fault was that the doctor was in a hurry, and the dilatation was not quick enough to please him; but this is not enough to prove that it was abnormal. A healthy cervix in a natural labour will always dilate if time be given; and the time required depends on the forces which effect dilatation. The proper treatment of slow dilatation of the cervix is to search for the cause of slow dilatation, and treat that if treatment other than by time be required.

*The Causes of Slow Dilatation.*—It may be (1) from *uterine inertia*; the uterine contractions, which ought to pull up the lower uterine segment, and so pull open the os uteri, and afterwards drive the bag of membranes into it, are weak and infrequent. I have elsewhere described the treatment of this condition. It may be (2) that the bag of membranes which ought to enter the os uteri, and stretch it open with gradually increasing power, is absent, either because *the membranes have burst prematurely* (the common cause), or because there is *too little liquor amnii* (a rare thing). If so, dilatation is slow, because dilatation has to be accomplished solely by the vertical fibres of the uterine body pulling up the lower uterine segment until the os uteri is large enough to admit the presenting part of the child. The first stage of labour is then long, to the great annoyance of patient and accoucheur. If the head or breech of the child present, the pelvis is normal, and the child of average size, with time the presenting part of the child will come down into the os uteri and dilate it; and the only treatment required is to sustain the nerve force of the patient by food and sleep. The patient should take as much food as she can keep down; and if she feels tired, but cannot sleep, a sedative should be given, either opium or chloral. Some think that chloral has a specific effect, besides its utility as a sedative, in helping dilatation of the cervix. This may be so, but I know not that it has been proved. If used, a full dose, 3ss, should be given. A grain of opium may be given, or the opium and the chloral may be combined. With this treatment the cervix will in time

dilate, and the patient will be delivered naturally. If, however, from any cause, such as malposition or excessive size of the child, or contraction of the pelvis, the presenting part of the child comes not down to stretch open the os uteri, further help is needed, and this is best given by the insertion of Champetier de Ribes' water-bag. The mechanical action of this instrument in dilating the os uteri is the same as that of the natural bag of membranes, which it effectively replaces. It may be (3) because labour has come on *prematurely*. When labour comes on at the full term the os internum has already been dilated during the preparatory or so-called "secret" stage of labour, so that the first stage of labour consists in the dilatation of the external os only; but in labour which has come on, or been induced, prematurely, the os internum is not dilated, and the first stage consists in the dilatation first of the internal and then of the external os. It consequently is slow. The treatment of this consists in letting the bag of membranes, and then the presenting part of the child, have plenty of time in which to open up the cervical canal. If the bag of membranes bursts too soon, and the head or breech enters not the os uteri, then it should be dilated with Champetier's bag.

Contracted pelvis, large size of the child, and abnormal presentations are indirectly causes of slow dilatation of the soft parts, because they prevent the head from coming down into the cervix uteri, and thus lead to premature rupture of membranes, so that neither bag of membranes nor fœtal head dilates the os.

The natural shape of the *os uteri externum* is that of a transverse slit. In a few women it is *small* and round; and it may be so small that a probe will not enter it. If pregnancy take place in such a uterus, the bag of membranes cannot get into the os uteri. In such cases, labour pains may continue for twenty-four or thirty-six hours without producing any appreciable dilatation of the os uteri. The treatment of such cases is to dilate the os uteri, first with bougies, and then with the finger, until the bag of membranes can get into it. Labour pains that have been going on for many hours will have made the os uteri big enough to admit a bougie. When the bag of membranes is able to enter the os uteri dilatation goes on with normal rapidity.

The cervix uteri may dilate badly because it is diseased. It may be contracted by *cicatricial tissue*. The tears in the cervix by which a first labour is generally completed do not contract the os uteri, but rather widen it, for the scar tissue binds together the mucous membrane of the vaginal aspect and that of the cervical canal. But when part of the cervix has been destroyed by ulceration, syphilitic or other, or by sloughing, then cicatricial tissue may form part of the circumference of the os; and fibrous cicatricial tissue is incapable of stretching. The possibility of dilatation of the os uteri, and the rate at which such dilatation will go on, depend on the proportion of healthy tissue to scar tissue in the cervix uteri. If the scar tissue only forms a small part of the ring of the os, the healthy tissue may be capable of stretching enough to let the child pass. But if the whole or the greater part of the os uteri is bounded by cicatricial tissue, natural dilatation cannot be hoped for, and the os uteri must be enlarged by incision. A probe-pointed bistoury is the best instrument to use. The edge should be guarded by wrapping it with strapping up to the terminal inch. With it several incisions should be made radiating from the centre of the os, and then delivery completed by pulling upon the pole of the fœtal ovoid which presents: either upon the head with forceps, or upon the breech by means of the leg.

Pregnancy may take place in a uterus the subject of *cancer of the cervix*. Whether such disease retards dilatation of the cervix or not depends upon its hardness, not upon its extent. Some cancers are much harder than others. One case has been published in which the cancer was so hard that the cervix dilated not, labour pains, after long continuing ineffective, ceased, and the child was retained in the womb for ten months afterwards. Others have been observed in which the whole cervix and adjacent tissues were a mass of cancer, and yet delivery was quick and easy. Hence in the treatment of labour complicated with cancer of the cervix, the only guide is observation of the course of labour. If the cervix opens up quickly no treatment is required. If early in labour it is noticed that the cervix is very hard, and that the bag of membranes has no effect upon it, the patient should be delivered by Cæsarean section. The forcible dragging of a child through a hard mass of pelvic cancer entails a risk to the mother which is probably as great as that of Cæsarean section when performed under favourable conditions. Cæsarean section, if the child is living, will deliver it alive. In cancer of the cervix, unless the disease can be removed, the mother will die soon, probably after much suffering; so that this is a case in which the life of the child may be considered as more valuable than that of the mother. If the cancer is limited to the vaginal portion of the cervix, this should be amputated notwithstanding the pregnancy. If it is not discovered till the patient is in labour, and the greater part of the cervix is healthy, the healthy part of the cervix will dilate and the child be born naturally. When child and placenta have been expelled (assuming that there is no doubt as to the diagnosis, and as to the limitation of the disease to the uterus), the uterus should be at once removed by the vagina. Experience has shown that the uterus can be easily and safely removed immediately after delivery, for although the

uterus is very vascular, yet the genital canal is at this time so patent that the uterus can easily be pulled down, and the vessels secured.

The pregnant uterus with cancer of the cervix has more than once been removed entire by abdominal section. This proceeding subjects the patient to unnecessary risk. It is safer to induce premature labour or abortion, and then, after delivery, to remove the uterus through the vagina. This course is desirable, because cancer of the uterus grows faster during pregnancy owing to the increased vascularity of the uterus; and because if left the cancer will probably extend beyond the uterus, and then its removal will be impossible.

Delivery may be obstructed by an *ovarian tumour*. Such a tumour can only obstruct

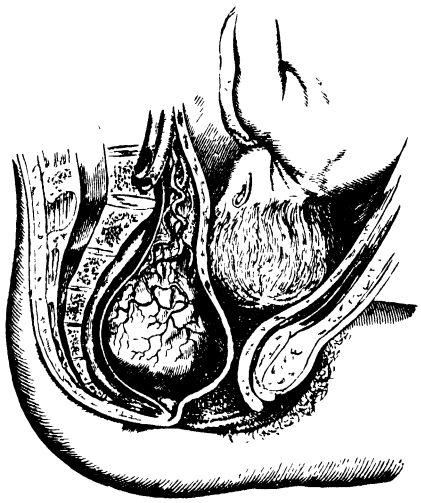


FIG. 53.—Ovarian tumour obstructing delivery. (After Tyler Smith.)

delivery if it be small enough to remain in the pelvic cavity. In that case, what happens depends on the size of the tumour. If it be small enough, the child may pass through the pelvis in spite of the presence of the tumour. If it be so large that the passage of the child, though possible, is yet difficult, the tumour will be squeezed and bruised during delivery; and this may cause hæmorrhage into the tumour, or inflammation of it, during childbed. Dermoid tumours are especially liable to this because they grow slowly, and are, therefore, more apt to remain long of small size and in the pelvic cavity. The bruising during delivery lowers their vitality, so that they become a prey to microbes, which cause suppuration. In the course of such suppuration they often rapidly enlarge; so that a tumour which was not discovered during delivery, even by repeated vaginal examinations, may within a fortnight become big enough to displace the uterus and cause retention of urine. A suppurated dermoid is the commonest cause of retro-uterine abscess in childbed. Dermoids sometimes grow in the pelvic cellular tissue; and when a dermoid behind the uterus has suppurated, it is not clinically possible to say whether the dermoid has grown in the cellular tissue, or whether it is an ovarian dermoid adherent in Douglas's pouch; and it is not very important.

If an ovarian tumour lies in the pelvic cavity, and is so large that the head cannot possibly pass it, the accoucheur should first try to push it up above the pelvic brim. Early in labour, before the membranes have ruptured, it will generally be possible to do this. If the head has advanced so far into the pelvic cavity that the tumour cannot be pushed up, the question is—Can the accoucheur remove it? The answer to this question will depend partly upon the features of the tumour, and partly upon the accoucheur's knowledge and experience in dealing with ovarian pedicles, and the instruments he has at hand. If he is familiar with the details of ovariotomy, and has the necessary instruments, he should, if possible, remove the tumour by the vagina. He should cut through the posterior vaginal wall (for the pedicle of the tumour will be behind the uterus), and thus expose the tumour, and bring it out into the vagina. Then transfix and tie the pedicle; next seize the pedicle on the distal side of the ligature with two strong pressure forceps, and then cut away the tumour. This done, carefully examine the pedicle to see that it is not bleeding, removing first one pressure forceps, then replacing it and removing the other. When satisfied that the pedicle has been securely tied, release it, and sew up the vaginal incision. This is the ideal treatment of an ovarian tumour which obstructs delivery. Its practicability will depend on the length of the pedicle, the presence or absence of adhesions, and the skill of the operator. Should the operator judge it wiser not to attempt the removal of the tumour, then he should make an incision into it, and pass a stitch on each side to unite the tumour to the vaginal incision. The resistance of the tumour will be removed by its evacuation; and by stitching it to the vagina, any infection of the peritoneum by the contents of the tumour will be avoided.

If pregnancy is complicated with a tumour too large to remain in the pelvis, the mutual effects of the pregnancy and the tumour may be important. The distension of the abdomen will be increased. The bearing-down efforts of the patient by which delivery should be helped, will be exerted at a disadvantage. Lastly, there is a liability to twisting of the pedicle of the tumour. For these reasons an ovarian tumour should always be removed as soon as it has been discovered, whether the patient be pregnant or not; and even if it has not been found out till the patient is in labour, it should be removed then, unless the labour is so far advanced that delivery is likely to take place during the operation.

Pregnancy sometimes takes place along with

*uterine fibroids.* Although fibroids are common, pregnancy with fibroids is not, because fibroids occur chiefly after the child-bearing age. If pregnancy occur with a fibroid, the tumour usually gets larger, softer, and more vascular during pregnancy, and then after delivery it undergoes involution—gets smaller, harder, and less vascular. I have known a fibroid disappear during puerperal involution of the uterus. A subperitoneal fibroid situated above the pelvic brim interferes in no way with pregnancy, labour, or lying in. A submucous or interstitial fibroid often causes changes in the endometrium which are inimical to the occurrence of pregnancy; but pregnancy may occur with such a tumour. It is often said that such tumours interfere with uterine contractions, make labour lingering, and cause post-partum hæmorrhage; but in my judgment the evidence in support of these statements is insufficient. It has not infrequently happened that the accoucheur has put his hand in the uterus, discovered the tumour, and without difficulty has enucleated and removed it. In some, the tumour has been spontaneously expelled after delivery; in others, which are rarer, the fœtal head has driven the tumour down before it, broken through its attachments, expelled it, and so cured the patient.

If a fibroid is situated in the cervix or lower part of the body of the uterus, and is so large that there is no possibility of the child's head getting past it, there are only two alternatives. One is to remove the tumour; the other to perform Cæsarean section. If the tumour is accessible it can probably be easily enucleated. The methods of enucleating uterine fibroids are described elsewhere. The only point special to enucleating a fibroid in a pregnant uterus is that its capsule will be very vascular, so care must be taken to see that after its removal the uterus drives the fœtal head down upon the site of the tumour. If this does not happen, the place of the fœtal head should be supplied by a dilating bag, that so the bleeding part may be pressed upon and hæmorrhage restrained. If the tumour is so large and so situated as to obstruct delivery, and it cannot be easily and safely removed, it is best to perform Cæsarean section, after which the uterine arteries can be tied, and the body of the uterus with the tumour removed.

Lastly, after delivery a fibroid may invert the uterus, just as it sometimes does an unimpregnated uterus. If the fibroid is so far driven down that its equator gets below the internal os, then when the internal os contracts the tumour will be driven farther down, and may pull the body of the uterus after it. The treatment is to peel off the fibroid and then reduce the inverted uterus. If it is undertaken soon, this can be easily done, by pressing the inverted fundus up with one hand, and at the same time steadying the cervix uteri and dilating the os internum with the fingers of the other hand on the abdominal wall.

Delivery may be obstructed by tumours of the pelvic bones. These may be *exostoses.* These are especially apt to grow where there is cartilage—at the symphysis pubis, the sacral promontory, and the sacro-iliac synchondrosis;

FIG. 54.—Sacral exostosis.

and where tendons are inserted—the psoas minor and Gimbernat's ligament. The pelvic bones may also be irregularly thickened by periostitis. Exostoses of the pelvis are seldom so large as to obstruct delivery, but they make laceration of the vagina more apt to occur, the mucous membrane being nipped between the head and

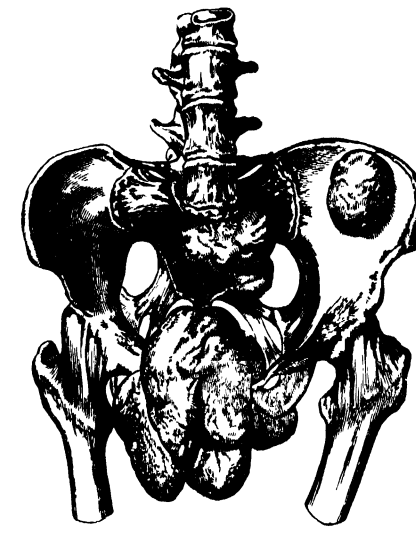


FIG. 55.—Cancerous growths from pelvic bones.

a bony spine. Pelves presenting exostoses have been styled "spiny" or "thorny" pelves, or "acanthopelys."

The commonest large tumours in the pelvis are enchondromata and sarcomata. Enchondromata usually grow from near the sacro-iliac synchondrosis, and are larger than most other

pelvic tumours. Sarcomata, especially osteo-sarcomata, sometimes completely block the pelvic canal. Fibromata grow from the periosteum; they are seldom large, but may be large enough to obstruct labour. Secondary growths of cancer may occur in the pelvic bones. Hydatids may invade the pelvic bones, and form a tumour bulging into and narrowing the pelvic cavity, although I know not of a case in which such a tumour has obstructed labour.

No detailed rules can be laid down for the treatment of cases of labour obstructed by a tumour; for the circumstances vary infinitely, according to the size, position, and nature of the tumour. All that can be said is, that if the pelvic space is so narrowed that a living child certainly cannot be born, Cæsarean section is generally indicated. This operation will effect the birth of a living child with less risk than that involved in dragging a mutilated child past a new growth.

## Faults in the Passenger

*Otherwise Labour obstructed by Abnormalities in the Ovum*

LIQUOR AMNII

(*a*) *Excess of: Hydramnios or Polyhydramnios* . . . . . 239
(*b*) *Absence of: Oligohydramnios* . . 240
(*c*) *Early Escape of: Dry Labour* . . 240

MEMBRANES

(*a*) *Thinness of* . . . . . 241
(*b*) *Toughness of* . . . . . 241
(*c*) *Adhesion of, to Lower Uterine Segment* . 241

PLACENTA

*Prolapse of* . . . . . 241

CORD

*Abnormalities of* . . . . . 241

CHILD

(*a*) *Death of: Post-mortem Rigidity: Emphysema* . . . . . 242
(*b*) *Large Size of: in Head Presentation* . 242
*In Breech Presentation; Impacted Breech* . . . . . 243
(*c*) *Unusual Ossification of Cranium* . 243
(*d*) *Malformations and Disease of: Hydrocephalus and Encephalocele* . 244
*Spina bifida, Hydrothorax, Ascites, Œdema, Distended Bladder, etc., Cystic Kidneys and other Tumours* . . . . . 244
(*e*) *Monstrosities: Acardiac* . . . 245
*Anencephalic* . . 245
*Exomphalic* . . . 245
*Conjoined twins* . . 245

LIQUOR AMNII

(*a*) *Excess of Liquor Amnii: Hydramnios, Polyhydramnios, or Hydrops Amnii.*—For a complete description of this condition, see "Pregnancy, Pathology of." We consider it here merely as a complication of labour. Though the quantity of liquor amnii is, as a rule, between one and two pints, it is impossible to observe two or three hundred cases of labour without seeing instances in which the quantity rises to two or three quarts, and occasionally several gallons of fluid may be found in the uterus. But while labour in some women is not appreciably affected by a very considerable excess of liquor amnii, a moderate amount of distension of the uterus by fluid causes marked disturbance in the labours of others. We cannot therefore define hydramnios as a complication of labour, by any reference to the quantity of fluid present, but only by the alteration in the ordinary course of labour which is observed to occur. Suppose we see a woman who is undoubtedly in labour, but whose pains are weak, infrequent, and ineffective. She complains of difficulty in breathing, and, perhaps, of nausea and vomiting. The abdomen is more distended than usual, and the whole abdominal swelling being dull on percussion, it is not partially due to flatus. If the distension were due to the presence of twins or to the complication of pregnancy by a fibroid tumour, palpation of the abdomen would reveal the presence of a solid body. If, however, there is fluctuation, it is necessary to distinguish between hydramnios, pregnancy complicated by ovarian or parovarian cyst, and pregnancy with ascites. The last named can be excluded by percussion with the patient in various positions; and when a large cyst accompanies pregnancy, the uterine contractions which can be felt in one part of the abdomen are absent over another. The diagnosis of hydramnios during labour is thus much easier than the recognition of the same condition earlier in pregnancy.

In making a forecast as to the result of a labour complicated by hydramnios, it is necessary to remember that such labours are generally premature; that the fœtus is generally ill-nourished or otherwise imperfect; and that, if not dead before labour begins, it often dies soon after birth. Further, as the fœtus floats freely in an enlarged cavity, no definite lie is assumed, and malpresentation is accordingly frequent. The prognosis is thus bad for the child. The mother suffers but slight risk to life. The distension of the uterus causes uterine inertia during all stages of labour. If left to nature there is a slow first stage, a slow or obstructed second stage, exhaustion, a risk of post-partum hæmorrhage, and the attendant risk of sepsis. If treated by early evacuation of the liquor amnii, there follows, as a rule, the need for artificial dilatation for the cervix, which is preferable to the risks to which the patient is exposed by delay. The prognosis for the mother is thus somewhat unfavourable, unless modified

by special care and exertion on the part of the accoucheur.

The abdomen must be supported by a firm broad binder, and the uterine contractions should be stimulated by friction. It is still usual to give ergot, as in other cases of uterine inertia; but quinine is rapidly becoming recognised as a more suitable drug for this purpose. Given in doses of 4 grains every hour for three or four hours, it favours alternate contractions and relaxations, much more useful than the somewhat permanent contraction seen after the administration of active doses of ergot. Indeed, in hydramnios, as in general, difficulties in delivering the child and the placenta are often avoided by adhering to the general rule never to give ergot until the uterus is empty. In slight cases, it is often possible to avoid breaking the general obstetric rule which forbids artificial rupture of the membranes before dilatation of the cervix is complete; in severe cases, on the other hand, the symptoms caused by distension may indicate evacuation of the fluid as soon as the diagnosis of hydramnios is made. It is usual to advise partial or gradual removal of the liquor amnii by means of an aspirating needle or a small "valved" opening high up in the uterus, the object being the preservation of the "fore waters." These suggestions, though traditional, are not practical; but before rupturing the membranes, it is always possible to secure, by artificial dilatation of the os, sufficient room to admit of bipolar version. Having passed one hand into the vagina and dilated until the os easily admits two fingers, simply puncture the membranes, check the flow of liquor amnii by plugging the vaginal outlet more or less completely with the wrist, and observe the lie of the fœtus. If the head presents and enters the pelvis in a good position, it may be left alone. The simplest way of avoiding future difficulty, however, is to secure and pull down a foot, and draw the half breech well into the partly dilated cervix. As a prognosis for the child is so poor, it is not worth while to attempt to secure head-first delivery. The case may now be left to nature, or may be terminated by manually completing dilatation and delivering the child. Abdominal pressure must be kept up while this is being accomplished, and extra care must be used during the third stage. Supra-pubic pressure should be made for some time after the removal of the placenta, in order to minimise the risk of post-partum hæmorrhage. Ergot may now be given to overcome the uterine inertia common in these cases. To obviate the tendency to subinvolution, quinine, iron, and strychnine may be given during the puerperium, and hot vaginal douches will also be found of service.

(*b*) *Deficiency or Absence of Liquor Amnii: Oligohydramnios.*—Sometimes the quantity of liquor amnii produced is less than a pint. This condition is discussed under "Pregnancy, Pathology of," but must be referred to here in so far as it affects the course of labour.

The fœtal parts are made out by abdominal palpation more easily than usual, and the abdominal tumour is small. Vaginal examination during a labour pain reveals the absence or small size of the bag of fore-waters. Periodic examination shows that the pains are not effective in dilating the cervix.

Nature's hydrostatic dilator, the bag of fore-waters, being wanting, the first stage is slow, and the risks attending exhaustion on the one hand and interference on the other are incurred.

During pains, the head should be pushed upward, so as to allow all the liquor amnii which is present to be forced past the head into the bag of fore-waters. Care must be taken to avoid rupturing the membranes during this manœuvre. If fluid is present only to the extent of a few ounces, enough can be collected in front of the head to form a useful bag of waters. Failing natural dilatation, the os must be opened by patient work with the fingers or by the use of hydrostatic dilators. The best of these for this purpose is the conical inelastic bag of Champetier de Ribes, which was originally designed for the induction of premature labour, and which is now largely used in cases of accidental hæmorrhage and placenta prævia. The bag can be introduced through the cervix uteri as soon as the os will admit the passage of two fingers. It is then almost, but not quite, filled with an aseptic fluid by means of a syringe. The conical bag then replaces the natural bag of waters, and when it has been expelled by uterine action through the cervix into the vagina, dilatation is sufficient to allow the second stage of labour to proceed. If labour pains are weak or infrequent, dilatation may be aided by gentle traction on the stalk of the bag.

(*c*) *Early Escape of Liquor Amnii: "Dry Labour."*—Premature rupture of the membranes may be caused by careless examination during a pain. If the membranes are unduly thin, they may break spontaneously under the pressure of uterine contractions at the beginning of labour. When the presentation or position is faulty, or when the shape of the pelvic brim is unusual, the presenting part does not fit accurately into the lower uterine segment, and may fail to shut off the fore-waters from the liquor amnii contained in the general uterine cavity. In these cases the whole "general contents pressure" during a pain acts upon the unsupported portion of membranes occupying the dilating os; the result is the descent of the bag of waters into the vagina as a sausage-shaped protrusion. Under these circumstances, if not very tough, the membranes will rupture early, and the rest of the labour will be "dry."

In "dry labour" the first stage is slow and painful. The head, unprotected by the bag of

waters, is more liable to injury from pressure than usual; the soft parts are also exposed to pressure from the uncovered head. The risks attendant upon exhaustion and interference must be remembered. The management consists in artificial dilatation, which may be accomplished as above mentioned, either manually or by hydrostatic dilators, the best of these, as in the previous case, being the bag of Champetier de Ribes.

### Membranes

(*a*) *Thinness of the Membranes.*—The obstetric interest of this condition depends upon the fact that unduly thin membranes are easily ruptured under circumstances such as those mentioned in the previous paragraph. In other words, the condition is a predisposing cause of early escape of the liquor amnii and subsequent "dry labour" (*q.v.*).

(*b*) *Toughness of the Membranes.*—Tough membranes do not rupture at the usual time, namely, as soon as dilatation is complete—in other words, at the commencement of the second stage of labour. Sometimes a child is born with its face and head covered by a portion of the membranes known as a caul; and occasionally birth is completed even at full time without any rupture of the membranes, the ovum being expelled complete, the membranes and placenta forming a sac containing the child and liquor amnii. Persistence of the membranes after dilatation is complete offers considerable resistance to the descent of the fœtal head (or breech). Artificial rupture of the membranes is therefore indicated as soon as the cervix and lower uterine segment are completely canalised. Some authorities hold that this method of hastening the second stage of labour should not be employed in first labours, as they consider that the bag of waters is of value in dilating the vaginal orifice. Artificial rupture of the membranes must be performed with aseptic precautions, not during a pain, lest the rush of waters should carry down a loop of the cord. Care must be taken to avoid injuring the fœtal scalp and the maternal passages. The safest method is to pinch up a bit of membrane and tear it outward. If this cannot be done with the fingers it may be managed with a pair of artery forceps: a sterilised hair-pin or any other blunt instrument may be used.

(*c*) *Adhesion of the Membranes to the Lower Uterine Segment.*—We define the lower uterine segment, for practical purposes, as that portion of the body of the uterus which is passive and becomes dilated during labour. Its surface is altered in shape during the first stage from that of a cup to that of a tube. It is clear that in the formation of the bag of waters and dilatation of the cervix, a movement of the membranes over the surface of the lower uterine segment is absolutely unavoidable. Thus if the membranes are adherent to the lower uterine segment, and do not break, dilatation cannot occur. Complete adhesion of this kind is, of course, theoretical; but adhesion sufficient to delay dilatation considerably is a practical difficulty, and results from inflammatory conditions of the endometrium during early pregnancy. Diagnosed by touch as soon as a finger can be passed through the *os internum*, this condition is easily removed by sweeping the finger round the os, separating the membranes from the uterine surface for a distance of about two inches all round.

### Placenta

*Prolapse of the Placenta.*—This rare occurrence occasionally causes mechanical obstruction to the course of labour. It presupposes premature separation of the placenta, and is generally met with in connection with the delivery of already dead children. The diagnosis is easy and the treatment is obvious. In placenta prævia (see "Pregnancy, Hæmorrhage during") delivery is frequently mechanically obstructed by the placenta. It is sometimes necessary to deliver through the organ, and this procedure is by no means always fatal to the child.

### Cord

(See also Abnormalities of the Cord, and Prolapse of Cord, p. 246.) Mechanical difficulty in labour may be caused by "absolute" or by relative or "accidental" shortness of the umbilical cord, which may be only a few inches long, or, while of full length, may be so wound round the child that its free portion is not long enough to permit of delivery without separation of the placenta. The second is the commoner variety of shortness. A strong cord may resist the expulsive powers to the extent of fifteen pounds' weight, and may thus greatly delay or completely arrest labour. Premature separation of the placenta may result seriously, endangering the life of the child, and exposing the mother to severe hæmorrhage. Inversion of the uterus is another possible result of shortness of the cord. The diagnosis of retardation of labour by absolute or accidental shortness of the cord is very difficult, especially before the head is born. If, however, the head recedes markedly between pains, and if labour lags without any other ascertainable cause, this condition will be suspected to exist. The escape of blood before the head is born suggests premature separation of the placenta and points to the same conclusion. In such cases forceps should be applied; or if the breech presents, delivery should be attempted by traction and abdominal pressure.

When the head is born the cord is generally within reach of palpation. It should be clamped or ligatured in two places and divided between them.

### Child

(*a*) *Death of the child* has long been associated in the minds of obstetricians with prolonged labour, and has often been mentioned as a cause of delay—the effect being mistaken for the cause. There are, however, certain carefully recorded cases[1] in which post-mortem rigidity has been present in the body of a dead child during labour, and has for some time prevented that undoing of the fœtal attitude, that unflexing of the trunk and limbs of the child, which is an essential part of the mechanism of labour.

The conditions under which post-mortem rigidity occurs *in utero* have not been sufficiently observed to permit of any statement as to the time and rate of onset, the duration or the termination of the condition. Its diagnosis is practically impossible, except by direct palpation. The treatment, were a diagnosis made, would be expectant, opium and other sedatives being employed to check the progress of labour and give time for relaxation of the fœtal body to occur.

Decomposition of the dead fœtus *in utero* sometimes causes various parts of its body to become distended with gas to such an extent as to impede delivery. This condition, which is known as *emphysema* of the fœtus, is recognised without difficulty by touch, the distended tissues yielding with a "crackling" feel, under pressure by the finger. The abdomen and thorax should be punctured, and the skin may be freely incised in accessible places in order to allow the escape of the gas. Delivery under these circumstances must be followed by energetic antiseptic measures.

(*b*) *Large Size of the Child.*—Though it is not one child in a thousand that weighs over twelve pounds at birth, there is no more common cause of delay during labour than relative largeness of the fœtal head, for cases are constantly met with in which the head is a trifle larger, while the pelvis is a trifle smaller than the average. In these cases, the second stage is prolonged, considerable time being demanded for the process of moulding, by which alone the passage of the head through the bony pelvis is rendered possible. Further delay is caused by the need for extra dilatation of the vaginal orifice, and tears of the perineum frequently occur. Though the head is usually the source of difficulty in the delivery of large children, broad shoulders sometimes become impacted in the pelvis, and frequently cause or increase perineal tears. Further, in pelvic presentations, the breech of a large child is liable to become impacted in the maternal passages. It is thus clear that the delivery of large children demands considerable care. Prolonged gestation is doubtless a common cause of excessive size of the fœtus. It is stated that pregnancy is prolonged over 300 days in at least 6 per cent of women. We may say, then, that the size of the child depends in part upon its age at the time of birth. The sex of the child must also be considered, as male children are well known to be slightly larger than females. Advanced age of one or both parents is said to favour large size of the child, but this may be simply because it tends to cause prolongation of pregnancy. Large size of one or both parents also conduces to overgrowth of the fœtus. It is certain that in many instances the size of the children increases in successive pregnancies. This is usually noticed in women with rather small pelves whose earlier children have been born spontaneously, forceps extraction, and occasionally craniotomy, being necessary at their subsequent confinements. It must be remembered that in the passage of a large head through an ordinary pelvis, the disproportion is exactly the same in nature as that which obstructs the passage of a normal head through a generally contracted pelvis (see Justo-minor Pelvis, p. 214). The modifications in mechanism and in treatment are accordingly the same in both conditions. The head cannot enter the brim with the vertex presenting, and therefore becomes more flexed than usual, the presenting part thus being behind the vertex. In extreme cases the presentation is, in fact, "occipital." This is important in diagnosis and in management; for unusual flexion of the head at the beginning of labour—presentation of a part behind the vertex—is a good and sufficient physical sign that the head is too large for the pelvis. Again, this mechanism of extra flexion implies that there is no room to spare in the sides of the pelvis; in other words, that the case is not one of flat pelvis. This at once contraindicates turning for exactly the same reasons which forbid version in justo-minor pelvis.

As to management, Hirst goes so far as to say that no woman should be allowed to exceed the normal duration of pregnancy (280 days) by more than a fortnight. Other authors advise that in cases where trouble from this cause has occurred in previous labours the patient should be examined periodically from about a month before full time, and that when it becomes difficult to make the head enter the pelvis by abdominal pressure labour should be induced (see "Labour, Operations"). Largeness of the head, however, is as a rule diagnosed only when unusual flexion of the head is discovered early in labour. The treatment consists in the application of the forceps as soon after the cervix is completely dilated as the operator deems consistent with the safety of the perineum. The advocates of symphysiotomy consider large size of the head to be a good indication for this operation, and unless it is certain that the child is dead, the symphysis should always be divided

[1] Ballantyne, *Edin. Obstet. Trans.*, 1894-95.

in preference to perforating the head of the child. But it is only very rarely that either of these measures is needful. It is almost always possible to deliver a large child alive with the forceps, if advantage be taken of the variations in the dimensions of the pelvis which can be produced by altering the posture of the patient.[1] Given a patient in the lithotomy posture, the outlet of the bony pelvis can be increased by pressing the thighs against the abdomen till the knees approach the shoulders. Similarly, the conjugate at the brim, *i.e.* the inlet to the bony pelvis, can be increased by allowing the legs to hang down (the feet not touching the ground), so that their weight draws the symphysis away from the sacrum, the whole pelvis rotating on an axis passing through both sacro-iliac joints. This "hanging legs position," or Walcher's posture, is of advantage whenever difficulty is met with in making the head enter the pelvis. The rules are as follows:—(1) Apply the forceps with the patient in the lithotomy posture. Place pillows under the buttocks, and while pulling the head through the brim into the pelvis allow the legs to hang down. (2) Remove the pillows, and while pulling the head out of the bony pelvis press the thighs against the abdomen, so increasing the pelvic outlet. (3) Whilst pulling the head, through the vulvar orifice allow the legs to hang down once more, as this relaxes the skin of the adjoining parts and minimises tearing of the perineum. After the birth of the head, some difficulty may be experienced in delivering a large body. Firm pressure on the fundus will favour rotation of the shoulders and their passage into and through the pelvis. When either of the axillæ can be reached, it forms a *point d'appui* for traction by the finger, which must be made carefully and in the pelvic axis, abdominal pressure still supplying the major part of the force employed.

In pelvic presentations large size of the child may act as a cause of impaction of the breech. If labour is delayed, although dilatation is complete and pains are good, this condition is to be suspected. The size of the presenting part should be reduced by bringing down one or both of the feet if this is possible, as it almost always is when the knees are flexed. Nature may then complete expulsion, or it may be necessary to deliver by traction and suprapubic pressure (see Management, p. 201). The attitude of the fœtus may, however, be the cause of impaction, for when the knees are extended, the feet being near the head and the pelvis flexed upon the trunk at the lumbar articulations, the trunk, pelvis, and legs form a wedge which cannot pass through the bony portion of the parturient canal. As flexion of the spine is essential to the formation of the wedge, the condition is immediately removed if a foot can be brought down, and this should therefore be done as soon as possible.

In any case in which the feet cannot be reached traction may be used, by means of a finger passed into the groin. A piece of aseptic material, such as a well-boiled handkerchief, may well be used for traction, one end having been slipped over the groin and pulled down between the legs. The blunt hook is not a safe instrument for this purpose, nor is the forceps seen at its best when applied to the breech. If it become necessary to break up the pelvis of a child the best instrument is a cranioclast or a cephalotribe. When this has been done the head must be perforated, lest the child should be born alive.

(*c*) *Unusual Ossification of the Cranial Bones.*—This condition causes difficulty in labour by preventing "head-moulding," which should reduce the head both laterally and in the antero-posterior direction. For the occipital bone slips under the parietals, and these in turn under the frontal, the head segments thus being telescoped; and also one parietal bone slips under the other, the upper one being that which is anterior (relative to the mother's pelvis) before rotation occurs. It is clear that when ossification has advanced to the sutures both these movements are prevented in some degree, and the diameters of the head during labour are those of a larger cranium.

Three reasons for undue ossification may be noted:—(1) Ossification may be precocious or premature. (2) If gestation be prolonged and the child is thus older than usual when born, normal ossification is further advanced than it generally is at the time of birth. (3) There are occasionally extra centres of ossification round which are formed the so-called Wormian bones between the usual cranial bones. These prevent head-moulding by interfering with the overlapping of the cranial bones at the sutures. Wormian bones are most often found in the posterior fontanelle and in that extra space known as the sagittal fontanelle, which is said to occur in 4 per cent of infants between the parietal bones, in a line joining the two parietal eminences. Peckham has recorded three cases of still-birth in which Wormian bones were the cause of death by preventing overlapping of the cranial bones.[1]

The diagnosis of undue ossification of the cranium is made by vaginal examination after dilatation has advanced sufficiently to permit of direct palpation of a considerable portion of the head. The prognosis is somewhat more unfavourable than when the head is merely large, because head-moulding cannot gradually improve the situation. In breech presentations it is even more difficult to save the child's life than when the head leads. The mother is exposed to the usual risks attendant upon delay

[1] Author's *Manual of Midwifery*, Edin., Clay, 1896, p. 413; also *Edin. Med. Journal*, July 1895.

[1] *New York Med. Record*, April 1888.

and interference. The management differs in one particular from that appropriate in cases where the head is large; for when the cranium is ossified firmly there is nothing to be gained by allowing time for head-moulding to occur, whereas when the head is merely large the longer it is possible to wait with safety before delivery, the easier is the extraction of the child. When undue ossification has been diagnosed, therefore, the operator should apply the forceps as soon as dilatation of the cervix is complete and the vaginal outlet is sufficiently soft; he should then deliver with the patient in Walcher's position, as described under the previous heading. If delivery is found to be impossible by this method, it is necessary to ascertain whether the child is dead or alive. If it is living, symphysiotomy is indicated; while if it is dead, the head should be perforated and extracted after comminution with a cranioclast. Walcher's position is, of course, as useful in delivering the after-coming head as in cases where the head leads. When perforation of the after-coming head is necessary the best position for the insertion of the perforator is the roof of the mouth.[1] The base of the skull is well broken up by this method, and the grip afforded to a cranioclast allows of easy extraction.

(*d*) *Malformation and Disease of the Child: Congenital Hydrocephalus* (see "Hydrocephalus").—Cases of congenital hydrocephalus as diagnosed after birth are much commoner than cases in which this condition causes difficulty in labour. The fluid occupies the cavities of the brain, or occasionally the sub-arachnoid space. In most cases the bones are widely separated, the sutures and fontanelles being greatly extended. In some cases of slight degree the bones reach the sutures and cover the whole cranium, but are much thinned. The presentation is said to be pelvic once in every five cases. In these breech presentations the base of the skull, which is not enlarged by disease, is first to enter the pelvis, and acts as the thin end of a wedge. Spontaneous delivery is therefore more frequent in breech than in head-first cases. Many heads, however, are so plastic as to pass through the pelvis after moulding has occurred. The cranium may burst under the pressure of the natural forces, or the fluid may pass from the cranial cavity and occupy a position under the scalp. As the large head stretches the cervix and lower uterine segment transversely as well as longitudinally, rupture of the uterus is the accident most to be dreaded. Out of thirty-eight maternal fatalities due to hydrocephalus, rupture of the uterus was the cause of death in no less than twenty cases.

On bimanual examination the head is felt to be large and soft, and is found to rest above the brim. The bones yield before the finger in a manner suggestive of brown paper. In breech cases the head does not follow the body, and its size and character can be recognised by abdominal palpation.

The prognosis is bad as regards the child. If the case is diagnosed early and actively treated, there is little risk to the mother; if unrecognised, these cases are grave, on account of the risk of ruptured uterus.

In the management of cases of hydrocephalus sufficiently marked to obstruct labour, no attempt should be made to save the life of the child. If born alive, these infants seldom survive long; the body is often small and shrunken, while malformations such as spina bifida are frequently present.

The use of the forceps should be avoided, as the grip is wide and unsatisfactory; the handles refuse to come together, showing that the blades are widely separated. Under these circumstances slipping and injury to the maternal soft parts are very likely to occur.

The perforator should be used at an early stage, and if natural expulsion does not follow in due course after the escape of the fluid, delivery should be completed by means of a cranioclast or a cephalotribe. In breech cases traction often bursts the head, or at least forces the fluid into an extracranial position under the scalp, so permitting delivery. If perforation of the after-coming head be necessary, it may be done through the roof of the mouth or behind the ear. If neither of these places is within reach, Van Heuvel recommends the removal of the fluid by means of a catheter, which is passed through an opening made into the spinal canal, and so upward through the foramen magnum into the cranial cavity.

*Encephalocele.*—Tumours of this nature are occasionally of sufficient size to delay or to completely obstruct labour. They may be either encephalocele proper or meningocele, the cranial substance being spread over the surface in varying degree. There may or may not be a communication between the sac and the cranial cavity through the pedicle, which may be either broad or narrow. Tumours of this kind are usually in the middle line, the occipital region being their commonest site, and the frontal the next in frequency. The extra fontanelles known as the cerebellar, the naso-frontal, and the medio-frontal correspond to weak points in the cranium, where outpushings of the membranes and the cerebral substance are liable to occur. The diagnosis may be very confusing, and demands careful examination, the whole hand being introduced into the uterus if necessary.

*Spina bifida* seldom causes actual difficulty in labour, as the tumour is seldom large. The condition may be myelocele or meningocele, and is often found along with hydrocephalus. It sometimes causes difficulty in diagnosis when the presentation is pelvic.

[1] Donald, *Trans. Obstet. London*, vol. xxxi.

*Hydrothorax* has very occasionally been recorded as a cause of delay in labour. In conjunction with ascites it is of more frequent occurrence.

*Ascites* has been observed in connection with other manifestations of syphilis, and also with new growths of various abdominal organs. It sometimes accompanies hydramnios. Apart from associated conditions it seldom obstructs labour.

*Œdema* of the fœtus may be caused by malformations of the fœtal circulatory organs, or may be associated with placental disease. It has been described by Spiegelberg as occurring in cases of congenital syphilis. There may be overgrowth of connective tissue and skin in addition to distension of the cellular tissue by fluid.

*Distended Urinary Organs.*—Owing to developmental errors the urethra is sometimes imperforate, when the fœtal bladder may become enormously distended. The ureters may also be imperforate, when the proximal portions may form tense tumours of considerable size. Hydronephrosis occurs under similar circumstances.

*Fœtal New Growths.*—The condition known as congenital cystic kidney may produce great enlargement of the fœtal body, and may completely obstruct labour. Ovarian cysts and various neoplasms of the liver, spleen, and pancreas have also been reported as having produced the same result. The occurrence of an "included fœtus" within the body of another must be remembered as a possibility, also the sacral teratoma.

The diagnosis of the above-mentioned conditions is, of course, extremely difficult, and indeed no definite conclusion as to the cause of obstruction can be arrived at, as a rule, until the difficulty has been overcome and the fœtus extracted. The general principles upon which such cases should be managed are as follows:—The life of the child must not be considered as of any importance. The life of the mother must not be exposed to risk by delay in ending labour. The means used to reduce the bulk of the fœtus vary according to the circumstances of each case. The choice lies between multiple incisions into the fœtal body, evisceration, and morcellement. A large pair of scissors will be found to be the most serviceable instrument; the blunt hook is also useful. The perforator and cranioclast may be employed as need arises.

(*e*) *Monstrosities: Acardiac Monsters.*—In twin pregnancy, when one embryo is less developed than the other, the heart of the stronger may so overpower that of the weaker that blood is forced from the single placenta up the umbilical arteries of the weaker embryo. This so disturbs its fœtal circulation that atrophy of the heart follows, the result being the production of an acardiac monster, whose lower parts, being nourished by the blood pumped into them through the umbilical arteries, grow rapidly, while the development of the upper portions is arrested for want of a proper blood-supply by the umbilical vein. The heart and upper parts are therefore represented by a mass of cellular tissue of low form not differentiated into organs, the lower limbs alone being recognisable as such. These monsters generally present by the feet, and are seldom large enough to cause serious difficulty during labour. Incisions may, however, be necessary, and occasionally the monster must be cut into several pieces and so removed.

*Anencephalic monsters* are characterised by absence of the brain and of the vault of the skull. The face looks upward, the neck being short and broad. The body and limbs are often very large and well developed. The absence of a properly shaped cranium tends to favour errors in presentation, and thus difficulty in labour is often caused. In head cases the face presents, and the diagnosis demands care. The small head does not dilate the passages enough to admit of ready delivery of the large shoulders. Again, owing to its size and shape, the head affords a very poor grip to the forceps. Delivery is easiest head last, and turning should accordingly be the treatment adopted, when this is feasible.

*Exomphalos and Ectopia Viscerum.*—Imperfect development of the anterior abdominal wall causes some portion of the abdominal viscera to lie outside of the body of the fœtus, occupying what is practically a dilated umbilical cord. In the extreme cases—those of complete ectopia viscerum—there is no cord at all, and the placenta forms one wall of the cavity in which the viscera are contained. In these cases the placenta must be separated before the child can be born, and considerable hæmorrhage is likely to occur. Serious difficulty is met with in those cases in which the fœtus is developed in a position of retroflexion; the fœtal attitude of flexion is lost and the back is hollowed, the upper portion of the fœtus being extremely rigid as a rule. Under these circumstances it is necessary to manipulate the fœtus in such a manner that the curve of its body shall correspond with the curve of the parturient canal, a manœuvre which is by no means easy.[1] Cases in which a minor degree of exomphalos occurs rarely present serious difficulty (see also "Teratology").

*Double Monsters.*—Conjoined twins obstruct labour in ways so varied that no definite rules can be laid down for their delivery. For this reason a detailed description of their varieties forms no part of practical obstetrics. Herman's classification, however, is useful; he arranges double monsters in three groups:—

(1) Those in which one end of the fœtus is double.

[1] Murray Cairns, *Trans. North of England Obstet. and Gyn. Soc.*, 1900.

(2) Those in which two fœtuses are loosely connected.

(3) Those in which two fœtuses are closely connected.

In (1) there are two heads more or less fused together (double-faced monster); or else the pelvis and lower extremities are duplicated. If seen early in labour, when the whole hand can be passed into the uterus, these conditions can be made out. A double head should be perforated; if there are two separate heads, one of them should be cut off. A reduplicated pelvis should be divided into portions with large scissors or a sharp hook.

In (2), the connection between the fœtuses being loose, labour is seldom seriously impeded, and any difficulty which may arise is of the same nature as those encountered in locking of twins (*q.v.*), and must be dealt with on similar lines. It is necessary, however, to make a complete diagnosis between "double monster" and "locked twins," in order to avoid destruction of both twins when it might be possible to save one alive. This remark also applies to class (3), in which the most difficult cases are likely to occur. The rule most generally applicable is to bring down the feet of one fœtus, and then proceed to embryotomy as circumstances may direct.

Playfair's classification of conjoined twins is also useful. It is as follows:—

(1) Two fœtuses united more or less completely face to face by thorax or by abdomen.

(2) Two fœtuses united back to back by the lower portion of the spinal column.

(3) Dicephalous monsters with single body and two heads.

(4) Two separate bodies, the heads more or less united.

Out of thirty-one cases collected by Playfair, twenty labours ended spontaneously, and parturition was fatal to the mother in only one case. Pelvic presentations were the most favourable, and turning was several times successful.

## Accidental Complications affecting the Child only


ABNORMALITIES OF THE CORD—
*Presentation and Prolapse* . . 246
*Knots of the Cord* . . . . 249
*Coils of the Cord* . . . . 249
*Torsion of the Cord* . . . . 249
*Rupture* . . . . . 249
*Undue Shortness of* . . . . 250
*Pressure on the Cord* . . . 250
PROLAPSE OF THE ARM . . . . 250
DORSAL DISPLACEMENT OF THE ARM . 250
PROLAPSE OF THE FOOT . . . . 251
INJURY TO THE FŒTUS DURING LABOUR . 251
STILL-BIRTH . . . . . 251


### A. ABNORMALITIES OF THE CORD

(i.) *Presentation and Prolapse.*—These are by far the most important complications, not only on account of their frequency, but because of the serious effect on the life of the child. The umbilical cord is a somewhat slender connecting line between the placenta and the fœtus, by means of which the nutrition of the child is maintained during pregnancy and parturition. Under the normal conditions of intra-uterine life the cord lies in a place of safety, free from injury and undue pressure, on the ventral aspect of the fœtus. Should it depart from this position, and come to lie over the presenting part, serious danger to the child will arise. It is important to clearly distinguish between the terms "presentation" and "prolapse" of the cord. "Presentation of the cord" is the term applied to those cases in which the umbilical cord can be felt lying over the presenting part of the fœtus, and in which the membranes are unruptured. The term "prolapse of the cord" is used for those cases in which the membranes are ruptured, and the cord projects through the cervix or descends into the vagina, or even appears at the vulva. If in any case the condition known as presentation of the cord is not recognised, the more serious complication of prolapse is certain to follow so soon as the membranes rupture. All cases of prolapse of the cord are not necessarily preceded by presentation. In some cases the abnormal position of the cord occurs quite suddenly at the time of rupture, and in other cases the cord is gradually extruded during the progress of labour along the side of the presenting part. The cord may form a tense band over the presenting part, or it may descend as a loop through the cervix into the vagina.

*Frequency.*—The frequency of prolapse of the cord appears to vary considerably, judging from the statistics obtained from the different maternity institutions in this country and abroad. According to Spiegelberg it occurs once in 86 cases. In the Dublin Rotunda Maternity the frequency was 1 in 200 cases. In other British maternity institutions it seems to be a less frequent complication, occurring about once in every 400 or 500 cases. Simpson has suggested that the increased frequency of prolapse in the German schools may be due to the dorsal position in which the parturient women are delivered. In this position the long axis of the uterus forms an angle of about 30° with the vertical, and the action of gravity would therefore assist the descent of the cord. In the left lateral posture, however, the long axis of the uterus is horizontal, and the fundus may be even on a lower level than the cervix, consequently there would be no tendency for the cord to descend towards the lower segment of the uterus under normal conditions of the fœtus and pelvis.

*Causes.*—Where the pelvis is of normal size, the child presenting with the vertex, and the muscular wall of the uterus not unduly lax,

there is very little tendency for the cord to present, owing to the close adaptation of the presenting part to the lower uterine segment. After rupture of the membranes this apposition of the presenting part to the lower segment of the uterus is still closer, as shown by the manner in which a considerable amount of amniotic fluid is retained after escape of the fore-waters, and therefore the chance of the cord becoming prolapsed is minimised. In cases of pelvic, including footling presentations, and in transverse or shoulder presentations, these conditions do not obtain to the same extent, so that presentation and prolapse of the cord is not uncommon. Again, in cases of hydramnios and twin pregnancies the excessive distension of the uterus interferes with the normal relation between the presenting part and the lower uterine segment. The same result may be brought about in cases of obliquity of the uterus and cases of pelvic contraction. Prolapse of the cord occurs more frequently in multiparæ than in primiparæ, because in the latter the head lies more deeply in the pelvis in the last few months of pregnancy and at the commencement of labour. The greater frequency of this accident in cases of excessive length of the cord and low insertion as in cases of placenta prævia, is easy to understand. Sudden rupture of the membranes occurring whilst the patient is in the erect posture may be a cause of prolapse of the cord, but it is more often associated with some other predisposing cause, such as undue length or contraction of the pelvis.

*Diagnosis.*—Before rupture of the membranes the cord can be felt as a movable coil lying over the presenting part, and may be overlooked unless a careful examination is made. After rupture the presence of a loop of cord in the vagina is easily recognised, and it is hardly conceivable that any mistake in the diagnosis could arise. It has been mistaken for a coil of small intestine ; but the absence of any mesentery is at once evident, and the presence of the pulsating umbilical artery confirms the diagnosis. Pulsation in the cord is, however, not always an available means of distinguishing the two, as it is absent where the fœtus is dead or where the cord is exposed to pressure. In the former case the cord hangs down as a flaccid loop. When pulsation in the cord cannot be felt, the death of the child should not be assumed till after careful auscultation of the fœtal heart.

*Prognosis.*—The mortality to infant life from prolapse of the cord is very high, and depends on the time at which it occurs, and also on the form of presentation. As a general rule, the danger to the child, so long as the membranes are intact, is not great. So long as the cord is only "presenting" it is only exposed to intermittent pressure, which does not endanger the vitality of the child. The later the cord becomes prolapsed the better the prognosis for the child, since the condition of the parts will be more favourable for rapid delivery. When, owing to early rupture of the membranes, the cord becomes early prolapsed, the danger to the child is very great. Prolapse of the cord is more serious in vertex presentations than in breech, since the cord is exposed to more dangerous compression against the hard surface of the head. In cases of placenta prævia and pelvic contraction the prognosis is very unfavourable. Prolapse of the cord is not in itself a cause of danger to the mother, except in so far as this complication calls for active interference in the course of labour, which otherwise might have been allowed to run its natural course.

*Treatment.*—All methods of treatment which are employed for this complication have for their object the saving of child life. Probably few cases occur in midwifery practice where the results depend more on the judgment and skill with which the necessary manipulations are carried out. Success in treatment—and by success one understands the delivery of a living child—depends on early diagnosis of the prolapse. The possibility of this complication makes a vaginal examination after rupture of the membranes a necessary routine practice. If the cord can be felt presenting, make a careful examination in order to decide whether the child is alive or dead. Seeing the comparatively small danger to the child so long as the membranes are intact, one important indication is to avoid their premature rupture. For this reason the woman should be kept in bed during the period of dilatation. An attempt should be made to remove the cord from its unfavourable position by placing the patient in the genu-pectoral position. In this attitude the fundus uteri becomes the most dependent part, and the cord gravitates towards the fundus. The woman should be kept in this position for about ten minutes, and should then be instructed to lie well over on her side with the hips raised by means of a pillow. In some cases, when the head descends after rupture of the membranes, it may force upwards a loop of cord previously presenting out of the way ; but this favourable result cannot be relied upon. A careful watch must be kept on the fœtal heart, and the obstetrician must be prepared to interfere if the condition of the fœtus calls for it. Should the cord again present after its replacement by the postural method it is best to perform bipolar version, bringing the leg down into the vagina. The half-breech will act as a plug in the lower segment of the uterus and prevent further prolapse.

When the membranes are found ruptured when the woman is first seen, and the cord prolapsed, the treatment to be adopted depends on the presentation and also on the extent to which the cervix is dilated. It will be convenient to consider cases of vertex presentation first.

If the cervix is only large enough to admit two fingers, an attempt may be made to replace the cord by means of a repositor. Replacement with the fingers is rarely satisfactory at this stage, as it is seldom possible to push up the cord into a position where it will remain, unless the whole hand can be introduced through the os. Special instruments are made for the purpose of replacing the cord, called repositors; but a new English catheter, size No. 10, answers the purpose well, and has the advantage of always being available. If a catheter is used it is necessary to cut a small hole in the end of the catheter opposite the eye, and to pass a piece of tape through it. The piece of tape is then passed round the loop of prolapsed cord near its end, and fixed to the end of the catheter sufficiently tightly to prevent its slipping through, but still allowing for circulation in the umbilical vessels. The stilette is now introduced along the catheter to give it the necessary stiffness, and it is passed up with the loop of cord into the uterus. The stilette should then be withdrawn, but the catheter is left *in situ*, as it in no way interferes with the course of labour. The fœtal heart must be carefully watched, and should it show signs of failing, other means must be resorted to. Should the cord again prolapse after its reposition, further manipulation is harmful to the child, and it is safer to have recourse to bipolar version while there is still a chance of saving the child.

If the os is sufficiently dilated to admit the hand when the case is first seen, two methods of treatment are possible. In the first place, an attempt may be made to carry up the loop of cord in the palm of the hand past the head, and to hook it over one of the lower limbs. When this has been done, the further progress of the case may be left to the natural forces should the cord remain *in utero*. Should the cord, however, again descend, the safety of the child will best be ensured by passing the hand up into the uterus, seizing a leg, and bringing it down into the vagina.

Lastly, certain cases are met with where the os is dilated to three-fourths its full size when the prolapse is discovered. Under these circumstances rapid completion of the delivery by means of forceps is indicated. It is probable that in this operation a certain amount of laceration of the cervix will occur, and in the case of a primipara the perineum may also be extensively lacerated as a result of the rapid delivery. It must be remembered, however, that these injuries to the mother are capable of immediate repair, and are not followed by any permanent injury, whereas in the case of the child its life is in serious jeopardy. It is, therefore, not only justifiable, but it is good practice, to risk these possible injuries to the mother in the interests of the child. In this country all are agreed that the first duty of the medical attendant is towards the mother, where the question of treatment concerns the life of the mother *versus* the life of the child. In the present instance this is not the point at issue. What we have to weigh in the balance is the *life* of the child as against a *traumatism* to the mother, which with proper antiseptic treatment is readily repaired, and for this reason the life of the child claims our consideration.

It is necessary now to consider what line of treatment should be adopted where prolapse of the cord is associated with presentations other than vertex. In cases of breech presentation the pressure on the cord, and, therefore, the danger to the child, is rarely so great as in vertex presentations, owing to the less resistant character of the presenting part. The best treatment is to pass the hand up and bring down a leg. This not only diminishes the size of the presenting parts, but the half-breech readily adapts itself within the lower segment of the uterus, and prevents the cord from again becoming prolapsed. Further, the presence of the leg in the vagina enables the child to be delivered rapidly by traction upon it should the fœtal heart show signs of weakness or slowing of the beats. In the case of oblique or shoulder presentations, the treatment called for to correct this abnormal presentation will remove the cord from its position of danger.

Where prolapse of the cord is associated with a flattened pelvis, reposition of the cord should not be attempted, as it is unlikely that it will be followed by any permanent results, owing to the shape of the pelvic inlet, which prevents the descent of the presenting part, and so interferes with its adaptation to the lower uterine segment. The best result will be obtained by performing bipolar or internal version, according as the os admits only two fingers or the whole hand. If the contraction of the pelvis is of such a kind that the delivery of a living child after the performance of version is not to be expected, it is best to replace the cord by means of a repositor, and to extract with forceps as soon as the cervix is sufficiently dilated to admit the passage of the child without undue risk to the mother.

Lastly, cases occur in which the prolapse of the cord is a complication of placenta prævia. The best method of treatment in these is to replace the cord with the hand, the patient lying in the latero-prone position on her left side, with the hips slightly raised. After pushing up the cord out of the way, introduce a de Ribes' bag into the lower segment of the uterus and distend it with boiled water or weak carbolic solution. The distended bag not only checks further hæmorrhage during the dilatation of the os, but its presence in the lower uterine segments prevents the cord from again becoming prolapsed. After expulsion of

the bag into the vagina the delivery of the child can be rapidly completed.

It is hardly necessary to add that in all cases where no pulsation can be felt in the cord, and where, after careful auscultation of the abdomen, no evidence of a living child can be obtained, the case must be treated according to the presentation found, and the fact that the cord is prolapsed may be entirely disregarded.

(ii.) *Knots of the Cord.*—This complication is usually associated with abnormal length of the cord. Knots are primarily caused by the fœtus slipping through a loop of the cord during the active movements that occur in the course of pregnancy. The knots may become tightened either during pregnancy or at the time of labour. Those which occur during pregnancy are usually more lightly knotted, and the constrictions produced in Wharton's jelly are readily observed after expulsion of the placenta. Cases in which the knotting has occurred during labour are more readily unravelled, and leave no indentations. Occasionally a double knot is caused by the fœtus passing through two loops, either consecutively or with one movement, owing to the two loops being apposed. It rarely happens that the knots are drawn so tightly as to interfere with the circulation through the umbilical vessels, but in rare cases this may be a cause of intra-uterine death.

(iii.) *Coils of the Cord.*—Coiling of the cord round the neck of the child is an exceedingly frequent occurrence, being observed as often as once in every ten cases. One or more coils may be found. When there is only a single coil there is little probability of any serious trouble arising in the course of parturition. Where the cord is coiled two or more times round the neck abnormal presentations may result, owing to the acquired shortening of the cord, resulting from the coiling interfering with the normal lie of the child. Again, the constriction caused by the cord may lead to interference with the blood-supply to the fœtus, and in this way asphyxia or even death of the fœtus may result during delivery.

Where coiling of the cord occurs round the limbs of the fœtus it may give rise to marked constrictions of the limb, which may extend through the soft tissues down to the bone. Under these circumstances the limb presents a curious appearance at the time of birth. Cases have occurred where the distal portion of the limb has become entirely separated in consequence of this gradual constriction. Where several coils of the cord encircle the neck of the child, a further danger may arise during the expulsive stage of labour owing to the shortening of the cord causing premature detachment of the placenta. Severe hæmorrhage may then occur, both during labour and after the expulsion of the child. It is important in all cases of labour, as soon as the head is born, to pass the finger round the neck of the child to see whether this complication is present. Unless the finger is passed up to the neck, coiling of the cord may easily be overlooked, as the swollen labia and perineum may conceal it from view. Where one or more coils are found, it is usually quite easy to draw the coil down so as to slacken the loop and slip one or more coils, as the case may be, over the head. In some cases where the child is being rapidly born there may not be time to draw down the coil and slip it over the head, and it is necessary to slacken the loop and allow the shoulders to be delivered through the loop. In very exceptional cases it may be necessary to divide the cord with scissors and deliver the child forthwith.

(iv.) *Torsion of the Cord.*—This may occur to an abnormal extent in the last few months of pregnancy, and may cause death of the fœtus. It does not occur during labour, and cannot lead to any interference with the normal course of labour; it is not, therefore, necessary to consider it in the present article.

(v.) *Rupture of the Cord.*—This accident occurs only in cases of precipitate labour, when the patient is suddenly and unexpectedly delivered in the upright posture. The effect on the child is twofold. In the first place, the child is deprived of the additional amount of blood which passes into its circulation, where ligation of the umbilical cord is delayed till ten minutes after the delivery of the child. Furthermore, hæmorrhage may occur from the torn end of the cord, though this is not usually serious, owing to the retraction of the walls of the vessels. The rupture usually occurs a short distance from the umbilicus. This accident rarely happens in cases of natural labour, and it can only occur when the cord is abnormally thinned or coiled round the fœtus.

To produce rupture of the cord rapid escape of the fœtus is essential. It is not necessary for the placenta to be still adherent, as the contraction of the cervix after expulsion of the fœtus is quite sufficient to hold back the placenta, and in this way fix the opposite end of the cord. Where the cord is much twisted, and presents thinner parts in places, the liability of rupture occurring in consequence of some sudden strain is greatly increased. The reason why rupture more often occurs near the umbilicus is found in the fact that abnormal torsion and thinning of the cord are more often found in this situation. Rupture occasionally takes place in the course of instrumental delivery and during the performance of version. As soon as the child is born, the torn ends should be ligatured so as to prevent further hæmorrhage. Where it is not possible to ligature the cord as a whole, owing to the proximity of the rupture to the umbilicus, the bleeding vessels should be picked up separately and tied.

(vi.) *Undue Shortness of the Cord.*—This is a rare complication of delivery. It is more common for apparent shortness to occur as a result of coiling of the cord round the neck of the child in vertex presentations, or owing to the child riding on the cord in the case of pelvic presentations. In the latter case the cord is stretched down between the thighs and up over the back of the child towards the placenta. Shortness of the cord, either actual or relative, can only affect delivery during the later stages of expulsion. During the early stages of expulsion progress is gradual, and the uterus retracts down as the child descends through the parturient canal. In the later stages of delivery a short cord may interfere with further progress, owing to the stretching of the cord between the umbilicus and the placental site preventing further advance of the presenting part. Certain signs are said to be suggestive of this condition, but none are actually diagnostic. These are descent of the presenting part during the pains with some hæmorrhage, followed by recession in the intervals, in cases where there is no marked resistance of the soft parts of the pelvic floor; also dragging pain referred to the placental attachment of the cord. Certain diagnosis is usually only possible when the cord can be felt tense and stretched. Examination per rectum may assist the diagnosis, when the shortening is due to coiling of the cord round the neck, and the descent of the head is prevented. The treatment of relative shortness of the cord due to coiling round the neck has already been described. Where the child rides on the cord the treatment consists in drawing down a loop of the cord, flexing the posterior knee, and slipping the loop of cord over it. After this the fœtal heart must be watched, and should it show signs of failing, rapid delivery must be effected. Where there is actual shortening of the cord it may in rare cases be necessary to divide the cord with scissors, following this up by immediate delivery.

(viii.) *Pressure on the Cord.*—In all cases of labour where the breech or lower extremities present, the umbilical cord is necessarily exposed to pressure during the expulsion of the child. In normal delivery the amount of pressure is not sufficiently great to interfere with the circulation in the umbilical vessels, owing to the latter being embedded in the elastic envelope formed by the jelly of Wharton. Where, however, there is any undue resistance in the parturient canal, as in the case of primiparæ, the pressure on the cord may be a serious complication, and it may be especially injurious during the passage of the head through the pelvis. Great danger is present where the after-coming head is delayed in cases of pelvic contraction. In these cases the cord is likely to be compressed between the resistant head and the bony rim of the pelvis and the child will soon perish from asphyxia unless immediate delivery is possible. In some cases of forceps delivery, where the cord is coiled round the neck of the fœtus, death of the fœtus has been caused by one blade of the forceps pressing on the cord and obstructing the circulation. Such an injury can usually be avoided by careful vaginal examination previous to application of the forceps.

### B. Prolapse of Arm

Prolapse of one or other upper extremity occurs under two conditions. It may be prolapsed and occupy the vagina in cases of shoulder presentations, or it may be prolapsed in cases of vertex presentations. The former will be considered in the article on shoulder presentations, the latter is an accidental complication of what might otherwise be a normal delivery. Slight descent of the upper limb may be found in the early stages of dilatation, and may disappear as the head engages more deeply. When the arm is found more deeply prolapsed by the side of the head there is usually some want of adaptation between the head and the lower uterine segment. It is found in cases of contracted pelvis and in lateral deviation of the head, and may suddenly occur at the time of rupture of the membranes. Death of the fœtus predisposes to prolapse of the arm, inasmuch as the normal attitude may be lost. Prolapse of the arm does not necessarily interfere with the progress of labour, though it may in some cases. The posterior part of the pelvis affords the most available space, and is therefore the most favourable position for the prolapsed limb. If the arm becomes prolapsed in front of the head it is more likely to cause the head to be wedged into the brim of the pelvis. Further progress of the head is thus prevented, and the prolapsed arm may be damaged or even fractured. The diagnosis of the condition is readily made, and by careful examination of the head it is not difficult to decide which arm is prolapsed.

The treatment consists in pushing up the arm, if this is possible, special care being taken to press the arm towards the ventral aspect of the child. Failing this, perform internal version. If the head has passed the pelvic brim, and is lying in the cavity of the pelvis, leave the case to nature, as delivery may follow without interference. If delay occurs at this stage it is best to complete the delivery with forceps, taking special care to avoid damaging the prolapsed extremity during the application of the blades.

### C. Dorsal Displacement of the Arm

This is a rare complication of pelvic presentations, and occurs still less frequently in some cases where the vertex presents. In pelvic presentations this displacement may be caused by injudicious attempts to rotate the body of the child during the delivery of the trunk, or it

may occur in the absence of any manipulations, in which case it is due to failure of the arm following the rotation of the trunk. The forearm of the child in this way comes to lie behind the nape of the neck, and interferes with the descent of the after-coming head through the pelvic brim. The treatment consists in attempting to rotate the body of the child in the opposite direction to that which caused the displacement, then seizing the forearm, and bringing it down over the front of the chest. Where the displacement occurs as a complication of vertex presentations, the projection caused by the arm interferes with the descent of the head through the cavity of the pelvis. Diagnosis is often very difficult. It may sometimes be possible to feel the arm above the pubes, but in other cases the delay in descent of the head can only be explained by passing the hand up past the head and ascertaining the position of the arm. Having found this condition, the best means of treatment is to perform internal version.

### D. Prolapse of Foot

This is a frequent occurrence in case of pelvic presentations, and may be artificially produced when version has been performed. These will not be discussed now. The cases of prolapse of the lower extremity which may be considered as an accidental complication are those in which the foot is prolapsed by the side of the head. The causes are similar to those already described as giving rise to prolapse of the arm, being especially frequent in dead and premature children. Where the condition is found associated with vertex presentations an attempt may be made to push upwards the limb, and to press the head down into the pelvis. Failing this, it is best to pull one foot, and at the same time press the head upwards toward the fundus, in this way producing a footling presentation.

Injuries to the Fœtus during Labour.—It will be convenient here to consider the numerous injuries to which the fœtus is exposed during the course of delivery. The majority of these occur in connection with pelvic presentations, especially where pelvic contraction is present in the same patient. Wherever labour is obstructed by abnormal conditions of the fœtus or of the pelvis, the delicate tissues of the fœtus are liable to suffer injury.

In *breech* presentations, injury to the vessels and soft parts about the groin and damage to the external genital organs may result from traction with the fillet and blunt hook :—Hæmorrhage into the liver, spleen, or around the kidneys may be caused by forcible traction on trunk; fracture of femur or humerus may be due to attempts to bring down a leg or arm; fracture of clavicle may arise from the same cause; bruising of the muscles of the spine and back of the neck from traction on the legs and over the shoulders; hæmorrhage into the sterno-mastoid muscle is also caused by traction over shoulders; injury to the articulation of the lower jaw from traction by means of the finger on the lower jaw. Dislocations are rare.

In *face* presentations, damage to the eyes may result from want of care in making a vaginal examination. Injury to the muscles and soft structures of the neck may be caused by extraction with forceps in mento-posterior positions.

In *vertex* presentations, bruising and hæmorrhage into the brain or beneath the dura mater may result from difficult forceps deliveries. Fracture of the cranial bones may be produced by precipitate labour. In cases of contracted pelvis, grooving and indentations of the scalp and cranial bones may be seen with or without hæmorrhage beneath the pericranium or within the skull. Paralysis of the facial nerve may be due to forceps delivery in vertex presentations. The paralysis usually disappears shortly after labour.

*Still-Birth.*—By still-birth is understood that condition in which the child after birth does not show the ordinary signs of life, but at the same time the signs of life may return either permanently or for a time if suitable treatment is employed. Cases in which intra-uterine death has occurred during pregnancy from various causes are not included under this title. Under normal circumstances, at the moment of birth the fœtus enjoys a condition of apnœa. Very soon—within a minute or two—a sense of want of oxygen is experienced, which acts as a stimulus to the medullary centre, and respiratory movements are initiated. The alteration in the surrounding medium may also act as a peripheral stimulus to the respiratory centre in the medulla. Any condition which interferes with the supply of oxygen to the fœtus during labour, and anything that prevents the entrance of air into the lungs, when the pulmonary circulation is established after the birth of the child, causes asphyxia to supervene, and the child is then said to be still-born. The onset of asphyxia is accompanied by expansion of the thorax, with opening up of the pulmonary circulation in the fœtus; this is followed by the inspiration of any media which happen to surround the child at the time. If it occurs while the head still lies in the cavity of the pelvis, even though it may be possible for some air to find its way into the lungs, liquor amnii mixed with mucus, hairs, etc., may be drawn into the lungs, and oxygenation of the fœtal blood will be interfered with. With the opening up of the pulmonary circulation, less blood passes along the umbilical arteries to the placenta, and as a consequence the centres in the medulla become less well supplied with blood, and their irritability is diminished. The respiratory movements gradually cease, and asphyxia results. In other cases asphyxia is

caused by prolonged pressure on the head, frequently produced by forceps, causing injury to the centres in the medulla, or causing hæmorrhages into the brain or beneath the dura mater.

The *causes* of still-birth comprise, first, those conditions in which the supply of oxygenated blood from the mother to the fœtus is interfered with; among these may be mentioned pressure on the cord in breech presentations, where the after-coming head is delayed, and compression of the cord where it is prolapsed or coiled round the fœtus; second, those cases in which the mother's condition during labour becomes very grave, either as a result of severe ante-partum hæmorrhage, eclamptic convulsions occurring during labour, or exhaustion of the mother from prolonged labour; third, those cases in which there is direct injury to the centres in the brain from prolonged pressure, as in cases of forceps delivery in case of difficult vertex presentations, or in case of prolonged traction where the after-coming head is delayed.

The signs of still-birth depend on the degree of asphyxia. For the characteristic signs and special treatment of asphyxia livida and pallida the reader is referred to the special article on this subject, Asphyxia (*Resuscitation*), vol. i.

## Retention of Placenta


Definition . . . . . 252
Simple Retention in Uterus . . . 252
Morbid Adhesion of Placenta . . 253
Spasmodic Contraction of Uterus . 253
Retention in a Fibroid Uterus . . 254
Retention of Fragments . . . 254
Retention in Cervix or Vagina . . 254
Method of Removal . . . . . 255


Definition.—When the placenta is not discharged from the genital canal within a certain period following the birth of the child, it is said to be *retained*. The duration of the third stage of labour is, however, subject to such wide variations in normal cases that any definition of "retention" must of necessity be more or less arbitrary. From the Strasburg Maternity 100 cases have recently been reported in which the separation and expulsion of the placenta were left entirely to nature, and in 44 of these cases the third stage occupied an hour or less; in only 80 cases was it concluded within three hours; in the remaining 20 cases it occupied various longer periods up to twelve hours. Ahlfeld states that if the expulsion of the placenta is left entirely to nature, in only 5 to 8 cases per 1000 does retention occur. Strictly speaking, retention of the placenta ought, therefore, to be a rare complication of labour. The tendency of modern practice, however, is, and has been for some time, to shorten the natural duration of the third stage by assisting the expulsion of the placenta by compression and manipulation of the uterus, while, if the placenta is not delivered within an hour, the case is regarded as one requiring more active interference on the part of the medical attendant. Retention of the placenta as thus understood has consequently become one of the most frequent complications of labour with which we have to deal. But it should be clearly understood that delay is the only fault in a large number of such cases.

*Varieties.*—There are two stages in the normal process of expulsion of the placenta: A, its separation from the uterine wall and passage into the lower uterine segment and cervix; B, its expulsion from the body through the vagina and vulva. In cases left entirely to nature it is found that stage A (stage of separation) seldom occupies more than fifteen to twenty minutes; while stage B (stage of expulsion) is usually very much longer. It follows from this that a retained placenta may lie, A, in the uterine cavity (*i.e.* above the retraction ring); B, in the lower uterine segment and cervix, or in the vagina. Since the expulsion stage is naturally much longer than the separation stage, it follows that the placenta will be oftener found retained in the cervix or vagina than in the uterus. The cause of retention in the uterus is always non-separation of the placenta, either complete or partial; the causes of retention in the cervix or vagina are (1) non-separation of the membranes, (2) deficiency of the expulsive forces. The frequency of the two varieties (A and B) is in inverse ratio to their importance: retention in the uterus is relatively rare, but of major importance; retention in the vagina is relatively frequent, but of minor importance.

A. Retention in the Uterus; Retention with Non-Separation either complete or partial.—This is due to some hitch in the normal process of separation of the placenta. As a rule, either uterine contraction and retraction are incomplete, or they are unable to effect separation owing to the abnormal firmness of the placental attachments. The former is well designated "*simple retention in the uterus*," and is dependent upon some degree of uterine inertia; the latter is the condition known as "*morbid adhesion of the placenta*." These two are the commonest varieties of retention of the placenta in the uterus. A much rarer variety is that dependent upon *spasmodic contraction of the uterus*, or, as it is usually called, "*hour-glass contraction*." In this curious condition there are two factors—(1) non-separation (complete or partial) of the placenta; (2) spasmodic closure of the lower part of the uterine cavity. Another rare condition is retention in the uterus from the *mechanical obstacle* offered by a fibroid tumour occupying the lower part of the uterine cavity. These four varieties of retention in the uterus must now be noticed in detail.

I. *Simple Retention in the Uterus.*—This con-

dition is not truly pathological at all; the placenta would probably, in all such cases, be expelled spontaneously if only sufficient time were allowed. As already stated, however, modern practice authorises the removal of the placenta by artificial means if its delivery is delayed beyond an hour; hence the frequency of simple retention. Cases are recorded where the placenta has been expelled spontaneously several days, or even a week, after the birth of the child, without any untoward result to the mother. Two clear risks attend prolonged retention—(1) the risk of hæmorrhage; (2) the risk of decomposition of the placental tissue. As long as the placenta remains completely undetached there will be no hæmorrhage, because no uterine vessel has been laid open. Should any part, however, become detached, serious bleeding may occur from the denuded part of the uterine wall, and interference may become urgently required at very short notice. Decomposition of the placental tissue would not occur if atmospheric organisms could be rigidly excluded from the genital tract; but as this is impracticable, dead organic matter in the uterine cavity is rightly regarded as a source of grave danger. On the other hand, manual removal of the placenta under antiseptic methods introduces no additional risk, while at the same time it relieves the patient from the dangers mentioned. Non-appearance of the placenta within an hour after the birth of the child is therefore an indication for interference on the part of the medical attendant. If there is unusual bleeding, the placenta has been partially detached; if not, the placental attachments have been undisturbed. Serious hæmorrhage is, however, unusual in this form of retention. On passing the fingers into the uterine cavity the detachment of the placenta can be effected with great ease, and herein lies the diagnostic distinction between this form of retention and that next to be described.

II. *Morbid Adhesion of the Placenta.*—It is obvious that the pathological lesion which determines this condition must lie in the stratum through which the line of cleavage passes in the normal process of separation, *i.e.* in the ampullary layer of the decidua serotina. It is a fact not very creditable to obstetrics that the precise nature of these changes has never been determined. The question can only be profitably studied in the placenta *in situ*, *i.e.* before the morbid attachments have been destroyed, and, of course, opportunities of obtaining a uterus with an undetached adherent placenta must be extremely rare. It is easy to surmise than an inflammatory process attacks the serotina, rendering it thicker, denser, and tougher than usual; but there is no actual evidence that such a change ever occurs in the decidua, and it is better to admit that the causes are entirely unknown. The morbid condition, whatever it may be, rarely involves the entire placenta; some portion of it is usually separated by the normal process, while the affected part remains attached. The result is that smart hæmorrhage occurs from the stripped part of the placental site, although at the same time the uterus may feel hard and firmly retracted. While the placenta remains wholly or partly in the uterine cavity complete retraction is impossible, and for the closure of the uterine sinuses it is essential that complete retraction should occur. We find this condition is usually indicated, therefore, by hæmorrhage, while the uterus remains large and is fairly well retracted; it is thus readily distinguished from hæmorrhage due to uterine inertia. Hæmorrhage is almost invariable with morbid adhesion of the placenta, because the adhesion is practically never universal, but affects portions of the placenta only. If universal adhesion be present there is, of course, no hæmorrhage. Neither is there external bleeding in the rare cases in which the circumference of the placenta is adherent while the central part becomes detached; a large retro-placental hæmatoma may then be formed. Sometimes the membranes as well as the placenta are adherent.

*The diagnosis* of this condition depends upon the recognition by the fingers of the morbid adhesions. Firm, dense bands and strings are found uniting the placenta with the uterus; these usually have to be torn through with the fingers or finger-nails, as they are so firmly united to the uterine wall. Sometimes portions of placental tissue cannot be removed at all, and must be left to break down and become discharged with the lochia. Cases have been recorded (Morgagni, Tarnier) where scissors have been required to cut through bands of unusual strength.

*The treatment* is to receive the placenta without delay. The method of removing the retained placenta will be dealt with in the last paragraph of this article.

III. *Spasmodic Contraction of the Uterus.*—The absurd and meaningless name which is still usually applied to this condition is "*hour-glass contraction of the uterus*," a name supposed to indicate the peculiar alteration in the shape of the organ which was supposed to be induced by it. The original diagrams are still faithfully copied into obstetric text-books, although they represent, not the condition actually found, but the theory by which their author sought to account for what he found. It is essentially a deviation from the normal processes of retraction and contraction of the uterine muscle which obtains during the third stage of labour. A transverse zone of spasmodic contraction occurs, usually just above the retraction ring, which narrows the cavity so much that it may be impossible to pass the finger through it, and the cord may be tightly gripped. Very rarely

the entire organ is affected, and the whole uterus so firmly closed that nothing can enter it, even under anæsthesia. Reliable observers have stated that sometimes the cervix itself becomes closed by spasmodic contraction; but this appears to be rare, and is *primâ facie* improbable. More commonly a transverse zone is alone affected; the placenta lies above it, and may be separated by the normal process of retraction in the uterus above the zone of spasm. Its expulsion is, however, prevented by the narrowing of the canal, and free bleeding consequently occurs. The condition is not usually recognised by abdominal examination, but on passing the fingers through the cervix they encounter the obstruction formed by the narrowed part of the uterine cavity. Sometimes a portion of the placenta is caught in the constriction, and the cord can always be felt passing through it.

Of the causes of this condition nothing is known. It is certainly not due, as was once supposed, to the exhibition of ergot during labour, for in most of the recorded cases no ergot had been previously given. Ahlfeld is probably right in regarding it as due to irritability of the uterine muscle when more or less exhausted by labour, and he considers that too early and too vigorous attempts to express the placenta are the commonest exciting cause.

*The treatment* is to dilate the constriction and remove the placenta if the amount of bleeding is serious; if there is little or no bleeding the uterus may be allowed a few hours' rest, when the spasm will pass away, and the placenta will then probably be spontaneously expelled. Usually the amount of bleeding is too great to allow of the expectant treatment being adopted. Even under anæsthesia great difficulty may be experienced in dilating the constriction, and the removal of the placenta may have to be effected piecemeal, as only one or two fingers can be passed up to the fundus. Once the placenta is removed the uterus usually retracts firmly, and there is no more hæmorrhage.

IV. *Retention in a Fibroid Uterus.*—An interesting example of this rare condition has been recorded by Dr Haultain,[1] in which, after a miscarriage, the placenta was retained, and all attempts to remove it failed owing to the insuperable obstacle offered by a fibroid in the lower uterine segment. It had to be left, and the patient died of septicæmia from placental decomposition.

Retention of Fragments of Placenta or Membrane.—If a small portion only of a placenta is morbidly adherent, while the attachments of the remainder are healthy, the non-adherent part may be expelled by the uterine contractions, leaving the adherent part *in situ*. Occasionally the entire chorion may be thus left in the uterus, being torn off round the placental margin; the amnion generally goes with the placenta, as it is much tougher than the chorion, and less firmly united to that membrane than to the umbilical cord. Portions of the chorion may be thus retained. The decidua is so thin and friable that it is probably seldom expelled entire, but its retention is of no importance. Outlying portions of placental tissue (*placenta succenturiata*) when present are, of course, often retained either by morbid adhesion or simple non-detachment.

*Diagnosis.*—If the portion of retained placenta or membrane be not very large, and if in addition uterine contraction and retraction are efficient, there may be no immediate hæmorrhage, and the fact of their retention may then be overlooked. Later on, in the puerperium more or less severe secondary hæmorrhage will occur during their separation. A minute examination of the whole afterbirth ought always to reveal the occurrence of retention of fragments; this is comparatively easily seen in the case of a portion of the placenta, but not so easy if a succenturiate placenta or a piece of chorion be retained. Excessive bleeding from a contracted uterus after the delivery of the afterbirth depends either upon lacerations or upon retention of fragments, but only by passing the fingers into the uterus can it be definitely settled that there are retained portions present.

*Treatment.*—Retained placental fragments must always be sought for at once and removed; in the case of the chorion, the subsequent risks—secondary hæmorrhage and decomposition of lochia—are much less. Small fragments of chorion may therefore be allowed to remain; but if a piece of any considerable size be retained, it should be sought for and removed in like manner.

B. Retention in the Cervix or Vagina; Retention of a Detached Placenta.—This condition is very frequent, and of comparatively little importance, as it does not occasion much hæmorrhage. It may be due to deficiency of the expulsive forces, when expression suffices to deliver it; or it may be due to morbid adhesion of the membranes (Fig. 56), when digital removal is called for. The fact that the placenta is not retained in the uterus may be noticed by observing the size and position of the uterine body. When the placenta leaves it an obvious diminution occurs in its size; while, as Varnier has pointed out, the level of the fundus often rises a little when the placenta lies below it, thus preventing the uterus from sinking into the pelvis (see Figs. on pp. 167, 168). If the condition be not recognised on abdominal examination, the finger passed into the vagina will at once feel the placenta bulging through the external os, or perhaps lying free in the vaginal canal.

[1] Allbutt and Playfair, *System of Gynæcology*, p. 592.

Method of Removing a Placenta retained in the Uterus.—When the fingers must, for any reason, be passed into the parturient uterus,

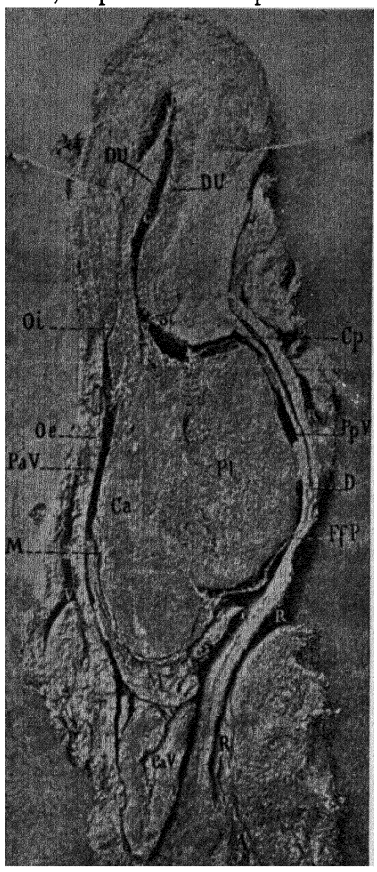



Fig. 56.—DU, adherent decidua; Oi, os internum; Oe, os externum; Pl, placenta; Ca, blood-clot; M, M, fœtal membranes detached.

the strictest antiseptic precautions are required; it is unnecessary to enumerate them here; the reader will find them set forth in the section on the management of labour. In removing an undetached placenta, the natural process should, as far as possible, be closely followed—*i.e.* first, the placenta should be completely detached from the uterine wall; and second, it should be expressed or withdrawn from the body. Unless detachment is completed before the removal of the organ is begun, fragments or larger portions of placental tissue will remain attached, and the complete evacuation of the uterus thus rendered more difficult.

The entire hand having been passed into the vagina under anæsthesia, the fingers first seek the lower edge of the placenta, and if no abnormality exists in the placental attachments, these are very readily torn through by sweeping the finger between the placenta and the uterine wall. One hand upon the uterus steadies it while the fingers gradually pass upwards to the fundus or across it, where the opposite placental edge is reached. Outlying lateral portions have then to be dealt with, and not until it is clear that the placenta lies quite free in the uterine cavity should its removal be commenced. It may then be withdrawn by the fingers into the vagina, or preferably the hand may be removed and the placenta delivered by expression. It is not necessary, in most cases, to detach the membranes with the fingers; they are peeled off when the placenta passes into the vagina, just as in the natural process of delivery of the afterbirth.

Difficulty in detaching the placenta may arise from morbid adhesions at the placental site, or from constriction of the lower part of the uterine cavity due to muscular spasm or to an encroaching fibroid tumour. Morbid adhesions can generally be broken through with the aid of the finger-nail, but much time and patience may be required for the task. Cutting instruments, such as scissors or the curette, should not be employed; it is better to leave small portions of placental tissue in the uterus than to run the risk of injuring the uterine wall. Adherent membrane may be even more troublesome than adherent placenta, but, fortunately, there is less risk in leaving it. Constrictions often cause very great difficulty, as it is unsafe to use any form of mechanical dilator.

A good deal of bleeding always attends the artificial separation of the placenta, because retraction is impeded by the fact that the fingers

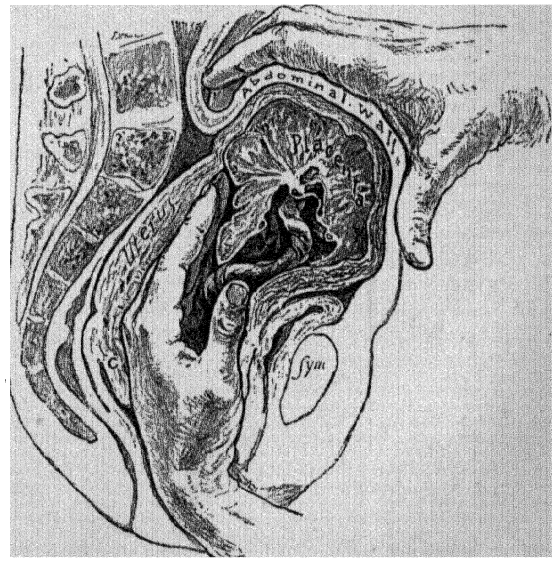



Fig. 57.—Method of detachment.

as well as the whole placenta are in the uterine cavity during the process. When the cavity is completely evacuated retraction usually follows, and the bleeding then ceases. A hot antiseptic intra-uterine douche should always be given afterwards, and massage practised through the abdominal wall, till all relaxation of the uterus has been overcome.

## Post-partum Hæmorrhage[1]


PRIMARY POST-PARTUM HÆMORRHAGE . 256
*Varieties* . . . . . 256
TRAUMATIC HÆMORRHAGE . . . 256
*Varieties* . . . . . 256
EXTERNAL TRAUMATIC HÆMORRHAGE . 256
INTERNAL TRAUMATIC HÆMORRHAGE . . 257
ATONIC HÆMORRHAGE . . . . 258
CONCEALED POST-PARTUM HÆMORRHAGE . 261
SECONDARY POST-PARTUM HÆMORRHAGE . 261
POST-HÆMORRHAGIC COLLAPSE . . 262


### I. PRIMARY POST-PARTUM HÆMORRHAGE

Primary post-partum hæmorrhage is the term applied to hæmorrhage occurring at any time within six hours after the birth of a child. It is one of the commonest accidents met with in midwifery. It occurs in two distinct varieties:—

A. Traumatic hæmorrhage.

B. Atonic hæmorrhage.

### TRAUMATIC HÆMORRHAGE

Traumatic hæmorrhage is the term applied to hæmorrhage due to laceration of any part of the genital tract, the result of direct or indirect violence. Bleeding due to rupture of the uterus is not, however, included under this head, as in the majority of cases of rupture hæmorrhage is only one of several symptoms, and consequently is better dealt with under the head of Rupture of the Uterus.

*Varieties.*—Two varieties of traumatic hæmorrhage are met with:—

1. External traumatic hæmorrhage.
2. Internal traumatic hæmorrhage.

1. EXTERNAL TRAUMATIC HÆMORRHAGE.—External traumatic hæmorrhage, in which the blood escapes externally, is very much the more common of the two forms.

*Ætiology.*—External hæmorrhage may result from lacerations occurring about the clitoris, perineum, or cervix, during the expulsion of the child. Perineal lacerations very rarely bleed to an extent sufficient to justify the name of hæmorrhage.

*Symptoms.*—The symptom of the case is hæmorrhage of a varying degree, which is not affected by the contractions of the uterus.

*Diagnosis.*—External traumatic hæmorrhage has to be distinguished from atonic hæmorrhage, that is, from hæmorrhage due to failure of the uterus to contract. Practically, we find that as a rule we commence to treat all cases as if they were atonic hæmorrhage, and that it is owing to various points which are determined during this treatment that we make the diagnosis of traumatic hæmorrhage. The first of these points is that the bleeding is found to be unaffected by the contractions of the uterus, the patient bleeding as rapidly when the uterus is contracted as when it is lax. The second is that while we are douching out the vagina or uterus with a double-channel catheter—Bozemann's—we notice that though blood is coming from the vulva, the fluid which is returning through the catheter is colourless. If the hæmorrhage is coming from a laceration of the clitoris or perineum, this latter fact is noticed when the nozzle of the catheter is in the vagina; if from the cervix, when the nozzle is in the uterus. As soon as we have in this manner roughly localised the site of the hæmorrhage, by carefully examining it the exact bleeding spot can be found.

*Treatment.*—If the hæmorrhage is found to come from a laceration of the clitoris, the easiest and most effective method of checking it is to pass a silk suture deeply below both ends of the laceration with a small curved needle. These sutures, which may if necessary be passed right down to the bone, are then tied tightly, and as a rule the hæmorrhage immediately ceases. If the tear is of great length, a third suture may be passed between the other two. These sutures are removed on the eighth day. Occasionally bleeding follows their removal, but if so, it can always be checked by means of a firm compress applied for a few hours.

If the hæmorrhage is coming from the perineum, it will be checked by the ordinary sutures which are inserted to bring together the lacerated perineal body.

Hæmorrhage coming from a cervical laceration is the most troublesome to check on account of the difficulty of exposing the laceration. If we have an American bullet-forceps or any form of volsella at hand, the cervix is drawn down by means of them. If, however, as frequently happens, we have not a volsella, an extemporised form of cervical tractor can be made in the following manner:—Thread a small curved needle with a long ligature of No. 8 or No. 10 silk. Pass two fingers of the left hand into the vagina to touch the most prominent portion of the cervix. Introduce the needle—held in a needle-holder—into the vagina under cover of the fingers of the left hand, and pass it through the cervix. The ends of the ligature are then knotted together, and by traction upon them the cervix can be exposed. The descent of the cervix will be very much facilitated by firm suprapubic pressure upon the fundus. As soon as the source of the hæmorrhage has been exposed, the latter is checked either by the ligation of a spouting vessel or by the suturing of a laceration. If the site of the hæmorrhage cannot be found, the bleeding can be stopped by plugging the utero-vaginal canal with iodoform gauze. Cervical sutures are to be removed on the eighth day, unless they have also been inserted with the object of bringing together the edges of a laceration; in such a case they may be left *in situ* until the fourteenth day.

[1] For accidental and unavoidable hæmorrhage during labour, see "Pregnancy, Hæmorrhage during."

*Prognosis.*—The prognosis of external traumatic hæmorrhage is always good unless the case is either neglected or improperly treated. It is especially bad in cases of low insertion of the placenta, owing to the proximity of the uterine sinuses to the laceration.

2. Internal Traumatic Hæmorrhage. — Internal traumatic hæmorrhage is the term applied to traumatic hæmorrhage in which the blood instead of escaping externally flows into the perivaginal or perivulvar tissues. If this occurs, a hæmatoma forms of varying size, and from this the condition has been given the name of *hæmatoma vel thrombus vaginæ et vulvæ*. It is said to be one of the rarest accidents in midwifery.

*Frequency.*—Internal traumatic hæmorrhage sufficient in amount to require treatment is a very rare occurrence. Statistics of its relative frequency are difficult to obtain. Winckel estimates its frequency at 1 in 1000, Hugenberger at 11 in 14,000. At the Rotunda Hospital there were 6 cases in 13,549 deliveries.

*Ætiology.*—The direct cause of the condition is the rupture of a vein in the tissue beneath the lowest part of the vaginal wall, more rarely beneath the vulvar mucous membrane (Winckel). The cause of the rupture is to be sometimes found in great stretching of the vaginal walls, especially when very rapidly accomplished; in the existence of vulvo-vaginal varices; or as the result of subsequent sloughing of the coats of a blood-vessel the result of long-continued pressure. However, in the majority of cases of this condition no assignable cause can be found. In such cases the rupture of the vessel may have been due to a pre-existing abnormal thinness of its coats, or to the gliding of the vaginal wall as it is drawn upwards during labour over the deeper structures—a gliding which may be associated with laceration of a vessel (Perret). A strong predisposing element to rupture, which is present in all labours, is the obstruction to the venous return which occurs during the descent of the head, and which tends to produce thinning of the walls of the veins by over-distension.

*Pathological Anatomy.*—These hæmorrhages may occur either below or above the pelvic diaphragm, and consequently can be divided into infrafascial and suprafascial. Infrafascial hæmatomata usually form, as has been said, at one or other side of the lower portion of the vaginal canal. If they form externally they are most frequently situated in the labia majora, more rarely in the labia minora, or in the remains of the hymen or perinæum. Usually a well-defined tumour results, varying in size from that of a hen's egg to that of a fœtal head. In some cases the hæmorrhage may extend in all directions, surround the whole vulva and vagina, and extend downwards upon the thighs. Sometimes, as the result of perforation of the pelvic fascia from sloughing, such hæmorrhage may extend upwards, as in suprafascial hæmatomata. Primary suprafascial hæmatomata are very rare. If a vessel ruptures in this region blood may collect round the upper part of the vagina, and then extend upwards in all directions beneath the peritoneum, reaching the kidneys behind, the level of the umbilicus in front, and the iliac crests laterally.

*Symptoms.*—A hæmatoma may form during delivery; but, as will readily be understood, although the vessel may be torn prior to the expulsion of the child, the pressure of the head will most usually prevent the escape of blood until after that event. Whether the child has been expelled or not, the first symptom of the condition is intense pain, associated with swelling in the neighbourhood of the ruptured vessel. In a short time a small tumour forms, elastic to the touch and of a blue colour, and gradually increases in size. If the hæmorrhage continues and the case is not treated, this tumour may rupture and the bleeding become external. At the same time, the patient becomes collapsed and anæmic in proportion to the amount of blood lost.

*Terminations.* — Internal traumatic hæmorrhage, if allowed to remain untreated, may terminate in one of the following ways:—

(1) The tumour may rupture, and free external hæmorrhage result which may or may not prove fatal.

(2) The hæmorrhage may extend interstitially—upwards towards the abdomen, or downwards towards the perineum—according as the ruptured vessel is above or below the pelvic fascia. The patient may thus bleed to death into her subcutaneous tissue.

(3) The tumour, if small, may be absorbed aseptically.

(4) Suppuration or decomposition of the contents of the tumour may occur.

*Treatment.* — If the condition is recognised before the birth of the child, the latter should be delivered immediately. If the amount of effused blood is still small, the forceps can be applied in the ordinary manner. If, however, the size of the tumour is so great as to obstruct delivery, its walls must be incised, its contents turned out, a piece of iodoform gauze placed over the opening, and the child delivered as quickly as possible. If the tumour has not been incised, and it increases slowly in size after delivery, the effects of firm pressure upon it may be tried; if this fails, or if the increase in size has been very rapid, it will be necessary to incise its wall and turn out the contents. In any case in which incision is practised, and the cavity is of large size, the latter should be douched out and then firmly plugged with iodoform gauze. This plugging is changed every day until the cavity is obliterated. If the latter was found to be of small size on opening it, deep sutures

passed beneath it, so as to bring its walls together when they are tied, will be found to be as satisfactory as, and less troublesome treatment than, the plug.

If the tumour is of small size, it may be left to absorb. Suppuration should never occur; if it does, the abscess must be opened at the spot at which it points, the pus evacuated, and the cavity plugged with iodoform gauze.

*Prognosis.*—The prognosis depends upon the treatment adopted and on the situation of the hæmorrhage. Suprafascial bleeding is very much more dangerous than is infrafascial, on account of the difficulty of checking it if it does not cease of its own accord. In either case the patient may die of hæmorrhage or sepsis. In the common form of hæmatoma, neither should occur if the case is properly treated.

### Atonic Hæmorrhage

Atonic post-partum hæmorrhage is the term applied to hæmorrhage due to the failure of the uterus to contract. Loss of blood occurs to a very slight extent in almost all cases of labour, as it is impossible for the placenta to be detached and expelled without such an occurrence. It is only when the amount lost becomes excessive that the term post-partum hæmorrhage can be applied to it. The average amount of blood lost, taking clots and fluid blood together, is four ounces before the placenta is delivered, and six ounces with the placenta and membranes (Dakin). According to Winckel, as soon as the patient has lost from 400 to 500 grams (fourteen to seventeen ounces) of blood, active treatment with the object of preventing further loss must be commenced.

*Frequency.* — The frequency of atonic post-partum hæmorrhage depends entirely upon what amount of hæmorrhage we consider can be called post-partum hæmorrhage. In the Rotunda Hospital, amongst 13,549 confinements there were 167 which required some form of treatment more radical than the massage of the fundus and the administration of ergot—that is, one case in 81·13. Amongst these a few cases of traumatic hæmorrhage are included.

*Ætiology.* — Before starting to discuss the causes of atonic post-partum hæmorrhage it is well to understand the factors which normally prevent its occurrence, as by so doing its ætiology will be rendered more obvious. The hæmorrhage which occurs during the detachment and expulsion of the placenta is normally checked by the united action of three factors :—

(1) The Contraction of the Muscular Coat of the Uterus.—The contractions of the muscular coat of the uterus bring about a temporary cessation of hæmorrhage during their occurrence. Each fibre of the uterus diminishes in length, and as a result the whole organ becomes almost as firm and hard as a billiard ball, and all the supplying arteries are compressed. As soon as the contraction passes off—and it only lasts a very short time—the uterine fibres return to their original length, the compression of the vessels ceases, and the hæmorrhage would recommence if another factor quite distinct from, but in a manner dependent on, the contraction was not also occurring. This factor, which is the most potent agent in causing the permanent cessation of the hæmorrhage, is the retraction of the uterine muscle fibres.

(2) The Retraction of the Uterine Muscle Fibres.—By the retraction of the uterine muscle fibres is meant a process which implies a permanent change in the relationship of the fibres to one another. During every contraction not only does each fibre shorten, but it becomes drawn upwards a very minute distance towards the fundus, *i.e.* it retracts. As a consequence, fibres which at the commencement of a contraction were end to end, at the completion of the contraction may have their ends overlapping one another, and after a few more contractions may have come to lie parallel. This new position of the muscle fibre, brought about by its gradual retraction, is a persistent position. It brings about the progressive diminution in size of the uterus, which is required to suit the diminution in the uterine contents as the fœtus is expelled during labour; and, after delivery, it brings about a final reduction in size, which is sufficiently marked to cause a permanent kinking and compression of the placental vessels. Accordingly, retraction is the process to which the final and permanent checking of hæmorrhage is due; but it must also be remembered that retraction itself is due to the occurrence of contraction. Contraction alone is not sufficient to check hæmorrhage permanently, but it is the means by which a permanent check is provided.

(3) The Clotting which occurs in the Mouths of the Vessels.—The clotting which occurs in the mouths of the vessels is so unimportant a factor in the checking of hæmorrhage that it may be almost neglected. It may be the direct cause of the cessation of hæmorrhage in a few very small vessels, but it will probably be more correct to consider its occurrence as being the result of the hæmorrhage ceasing rather than as a cause of its doing so.

The above are the normal agencies by which the occurrence of post-partum hæmorrhage is prevented. Accordingly, we are now in a better position to understand what are the conditions which will favour the occurrence of hæmorrhage. Speaking generally, the latter may be said to be anything which tends to prevent the due retraction of the uterine muscle fibres, either directly as a retained adherent placenta, or indirectly, by preventing contraction from taking place, as degeneration of the fibres from some pathological condition.

The following are the principal causes of post-partum hæmorrhage:—

(1) Retained Placental Fragments, Membranes, or Blood-Clots.—Such a condition is generally due to bad management of the third stage. Fragments of placenta and membranes may, however, also be retained owing to their too firm adhesion to the uterine wall, the result of a former endometritis.

(2) Uterine Inertia.—This may in turn be due to: (*a*) previous over-distension of the uterus, as in hydramnios twins; (*b*) metritis; (*c*) prolonged labour; (*d*) weak muscular development of the uterus; (*e*) faulty shape of the uterus—maldevelopment; (*f*) tumours.

(3) Precipitate Labour.—During a precipitate labour the uterus has not had time to undergo the normal amount of retraction, and consequently is not ready—so to speak—for the third stage.

(4) Placenta Prævia.—In this condition the hæmorrhage results from a portion of the placenta being attached to the non-contractile lower uterine segment.

(5) Tumours of the Uterus.—These, as well as causing uterine inertia, act by preventing the uniform retraction of the fibres.

(6) Any Condition which Weakens the Patient.—Such are: (*a*) previous hæmorrhages; (*b*) any form of wasting disease.

*Diagnosis.*—The diagnosis of atonic hæmorrhage is made by finding hæmorrhage coming from the interior of a non-contracted or badly contracted uterus.

*Treatment.*—The treatment of post-partum hæmorrhage falls under two headings:—

(i.) Prophylactic Treatment.
(ii.) Curative Treatment.

(i.) Prophylactic Treatment.—The prophylactic treatment of atonic hæmorrhage consists in the proper management of the third stage. The writer considers this to be a point of so great importance that he offers no apologies for giving a brief account of it in this place.

As soon as the child is born the patient is turned upon her back, and the doctor or nurse "controls" the fundus with one hand. To do this, the hand is placed horizontally over the fundus of the uterus with its ulnar border sunk down into the abdomen so as to touch the promontory of the sacrum. It is thus in a position to note the occurrence or cessation of uterine contractions, and during the latter to prevent the accumulation of blood in the cavity by exerting firm pressure when necessary. If the bladder is full it ought to be emptied, as pressure over a distended bladder causes pain, and also makes the future expression of the placenta more difficult. Nothing further is done, if everything progresses in a normal manner, until the placenta has left the contractile part of the uterus. As soon as this occurs the placenta is expressed from the vagina by the "Dublin method," or, as it is more commonly, though incorrectly, termed, Credé's method. As the placenta passes through the vulva it is seized in the hands and gently rotated, so as to twist the membranes into a rope, and thus bring them away entire. Any slight hæmorrhage which may occur is checked by massage of the fundus and the administration of ergot. As soon as it has ceased the binder is firmly applied; and, until the last pin which fastens it is in process of insertion, the controlling hand should remain upon the fundus.

If the third stage is correctly managed, the frequency of post-partum hæmorrhage is reduced to a minimum. It is said that the number of cases of this form of hæmorrhage which occur in a doctor's practice are in inverse proportion to the skill with which he manages this critical period.

(ii.) Curative Treatment.—The curative treatment of post-partum hæmorrhage is most satisfactory, if it is intelligently carried out. It is essential to have a definite plan of action laid down in our minds which we know so thoroughly that we shall follow it mechanically. Such a plan should be graduated so as to commence with the mildest measures, and then pass on—if they fail—to others which will be more radical. The following is such a plan in the order that should be adopted, and presupposing that the failure of each measure in turn requires the adoption of the subsequent one:—

(1) If hæmorrhage starts after the birth of the child which is not checked by massage of the fundus, ascertain whether the placenta is in the uterus or vagina. The signs which tell us that the placenta has left the uterus are:—

(*a*) The lengthening of the portion of cord which is outside the vulva.

(*b*) The rising of the fundus upwards from a finger-breadth or two above the pelvic brim almost to the umbilicus.

(*c*) The increased mobility of the body of the uterus owing to its upward displacement and consequent loss of support.

If the placenta is in the uterus, try the effects of massage for a little longer. If this does not check the bleeding, or if the placenta was already in the vagina—

(2) Express it by the Dublin method, if possible. To express the placenta, grasp the fundus with one or both hands during a pain, and press it downwards and backwards in the direction of the last piece of the sacrum. By this means the uterus is displaced downwards into the vagina, and the placenta driven out in front of it. Then stimulate the fundus to contract by friction and the administration of ergot. Up to three drachms of the liquid extract of ergot may be given by the mouth, but more certain and rapid in its action is the hypodermic administration of citrate of ergotinin. From $\frac{1}{50}$ to $\frac{1}{25}$ of a grain of the

latter may be injected. If this still fails to check the bleeding, or if the placenta could not be expressed at the start—

(3) Place the patient in a cross-bed position, wash her externally, and douche the vagina with a solution of creolin (℥ss to a gallon), at a temperature of 110° to 120° F., having first passed a catheter, if this has not been done already. If the placenta is still in the uterus, remove it manually. The removal of a placenta is a comparatively simple operation as far as the operator is concerned, but it is by no means as straightforward for the patient. In the first place, it is rarely if ever possible to give an anæsthetic, and consequently the operation is attended with a considerable amount of pain. In the next place, it is an operation during the performance of which it is specially easy to inoculate the patient with septic infection, owing to the intimate relationship of the fingers to the uterine sinuses, while detaching the placenta. It is performed as follows:—The preliminary steps as described above having been carried out, the hand is introduced into the uterus, taking care to keep outside the membranes, at the same time applying firm counter-pressure over the fundus with the other hand. Feel for the edge of the placenta, and then with a to-and-fro sawing motion of the fingers separate it from the uterine wall, gradually working up from below. Endeavour if possible to detach it in one piece, and then, grasping it in the hand passed above it, draw it out. Then douche out the uterus thoroughly, and administer ergot as directed above.

If the placenta has been previously removed by expression, and the vaginal douche fails to check the hæmorrhage, a hot uterine douche is given, creolin solution being used as before. If the bleeding still continues—

(4) Compress the fundus firmly between the fingers of one hand in the anterior fornix and the other hand upon the abdominal wall, thus squeezing out any clots that may be retained, and then repeat the intra-uterine douche.

(5) Introduce the hand into the uterus and remove any fragments of placenta or of membranes, and all clots. Then repeat the intra-uterine douche.

(6) In those cases in which hæmorrhage resists the above treatment, there are still two final measures before us from which a choice can be made. These are, either to plug the utero-vaginal canal with iodoform gauze, or to inject perchloride of iron into the uterine cavity. Of the two, the former is preferable, as will be seen later.

The uterus is plugged with iodoform gauze in the following manner:—Place the patient in the cross-bed position, if she is not already in it, and seize the anterior lip of the cervix with an American forceps and the posterior lip with another. If a short posterior speculum is to hand it may be introduced, and will facilitate the proceeding; it is not, however, absolutely necessary. Then pass the end of a long strip of iodoform gauze, about 2 inches in width, up to the fundus, by means of a special plugging forceps or with the end of the Bozemann's catheter. The remainder of the strip is pushed up piece by piece until it is finished. A fresh strip is then knotted on to the former, and introduced in a similar manner. As soon as the uterus is full the forceps are removed, and the vagina also plugged. As a rule, three to four strips of gauze 6 yards long and about 2 inches wide are required. It must be remembered that it is not the large cavity of a dilated uterus which we have to plug, but rather the comparatively small cavity of a contracting one, because on the introduction of a small piece of gauze the hitherto flaccid uterus quickly contracts upon the foreign body. Finally, a tight abdominal binder is applied in order to compress the uterus from above, and more ergot may be given. The gauze must be removed in from twelve to twenty-four hours, and if there is any rise of temperature, a uterine douche administered.

The use of perchloride of iron was introduced by Barnes. He recommended that a few ounces of Liq. ferri perchlor. (B.P.) be injected into the uterine cavity from which all clots have been removed. Another and perhaps easier method of applying the iron is to add Liq. ferri perchlor. fort. (B.P.) to warm water until a light sherry-coloured fluid is produced. The uterus is douched out with this and then with ordinary creolin solution. Barnes claims that iron acts in the following manner:—

(*a*) It coagulates the blood in the mouths of the vessels.

(*b*) It constringes the tissues round the mouths of the vessels, and so compresses the latter.

(*c*) It provokes some contraction of the muscular wall of the uterus.

The great advantage of iodoform gauze over iron is that it has no tendency to interfere with the nutrition of the superficial portions of the uterine wall. Iron, on the other hand, causes a very considerable superficial necrosis, and, if saprophytic germs gain entrance to this dead tissue, they have a very suitable pabulum on which to live. Again, iodoform gauze is as certain as anything can be in its action, and even if the hæmorrhage is coming from a large vessel which has been torn across owing to a laceration of the uterus, it will in all probability prevent it from bleeding. Iron may and sometimes does fail, and if it does it is impossible to resort to plugging as, owing to the manner in which the tissues have become constringed, gauze could not be introduced. If iron is used, the uterus must be douched out next day, and every subsequent day if there is any rise of temperature.

The above s the line of treatment which the writer considers is most suitable in cases of atonic post-partum hæmorrhage. It is, of course, impossible to follow a regular stereotyped plan in all cases ; special cases call for special variations in the treatment, and in some instances it may be necessary to resort immediately to the plug owing to the condition of the patient. However, in the great majority of cases in which the accoucheur has been in attendance from the commencement of the hæmorrhage, it will be possible to follow a system such as the above, and so save the patient from the risk of intra-uterine manipulations in all but the most serious cases.

There are two proceedings which are very frequently recommended that have not been mentioned. They are of use in some cases, and if they will not finally check the hæmorrhage they will at all events gain a little time. The first of these is compression of the aorta. It is comparatively easy—unless the patient is very stout or strains very hard—to compress the aorta through the abdominal wall against the lumbar portion of the spinal column. It is a proceeding which is of use, if we have an assistant capable of performing it, while preparations are being made for intra-uterine treatment. The second proceeding is the bimanual compression of the uterus, not as recommended above with the object of expressing clots, but rather with the object of preventing further hæmorrhage by compressing the bleeding vessels. It is carried out as follows: —Pass the right hand into the vagina and place two fingers behind the cervix in the posterior fornix. With these fingers press the cervix forwards in such a manner as to fold it beneath the body of the uterus ; then compress the latter as firmly as possible between the vaginal hand and the left hand upon the abdominal wall. This is also only of use in order to gain time, as it will rarely if ever arrest the hæmorrhage finally ; as such, however, it may sometimes be found of use.

There are a few methods of treatment which the author would like to warn against. Such are the intra-uterine injection of vinegar, the freedom of which from bacteria can never be assumed ; the application of ice or the pouring of cold water on the patient's abdomen, a practice which is sufficient to determine the death of a collapsed patient by increasing the collapse ; the introduction of ice into the uterus, both on account of the risk of sepsis and of the shock it causes ; the injection of ergot before the placenta has left the uterus, unless we are prepared to remove it immediately ; and the plugging of the uterine cavity with any material which is not absolutely sterile.

*Prognosis.* — The prognosis of post-partum hæmorrhage is always good if the case is taken in time. A woman can lose an amount of blood immediately after delivery without being very much affected, which at another time would bring her to the point of death.

Concealed Post-partum Hæmorrhage.—Concealed post-partum hæmorrhage is the term applied to post-partum hæmorrhage when the escaped blood is stored up in the uterus instead of pouring out through the vulva. It is to a large extent an artificial condition, that is to say, it is caused by the attendant compressing the lower uterine segment instead of the fundus, and so placing an obstruction in the way of the escape of the blood. It may also occur behind a detached placenta which is blocking the lower uterine segment, if the fundus is not properly controlled. If it occurs, it is recognised by the increase in size of the uterus. Its treatment consists in immediately removing the obstruction to the escape of blood and then emptying the uterus by expression. If the hæmorrhage still continues, the further treatment of the case is the same as that of the more usual form of post-partum hæmorrhage.

### II. Secondary Post-partum Hæmorrhage

Secondary post-partum hæmorrhage is the term applied to bleeding coming on more than six hours after the completion of labour. It is also known as puerperal or late hæmorrhage.

*Frequency.* — At the Rotunda Hospital, in which patients remain for eight days after their confinement, 13 cases of secondary hæmorrhage occurred in 13,549 confinements—a proportion of 1 in 1042·23.

*Etiology.* — Secondary post-partum hæmorrhage may arise in three ways :—

(1) Owing to the separation of the thrombi in the mouths of the uterine blood-vessels. This may occur owing to some sudden increase in the blood-pressure, or to the sloughing of the coats of a vessel as a result of a previous long-continued pressure.

(2) Owing to a congested condition of the endometrium. The commonest cause of congestion of the endometrium during the puerperium is a relaxed condition of the uterus. This condition, which is known as subinvolution, may be caused by the retention of pieces of placenta or membrane, malpositions of the uterus, fæcal accumulations, or getting up too soon.

(3) Owing to the presence of tumours, either pre-existing or arising subsequent to delivery. Amongst pre-existing tumours, myomata of the body of the uterus are the commonest. The only tumour which is likely to form subsequent to delivery is that known as deciduoma malignum (*vide* article "Puerperium").

*Treatment.*—If the hæmorrhage is slight, the administration of ergot in full doses, the expression of all clots from the uterus, and absolute rest in bed, may be sufficient to check it. If it does not respond to this treatment, or if it is severe from the start, the vagina and uterus should be douched out with hot creolin lotion,

and the latter explored with the fingers in order to ascertain the cause of the hæmorrhage. If a retro-deviation of the uterus is present it must be corrected, and a pessary inserted if the uterus will not remain in a normal position of its own accord. If a portion of placenta has been left behind, it must be removed with the finger or blunt curette. If hæmorrhage still continues, the uterine cavity must be plugged with iodoform gauze. In addition, the bowels must be regulated, and the daily administration of ergot continued for some days. If the hæmorrhage is due to the presence of a myoma, and the bleeding cannot be checked by the use of ergot, hot douches, and plugging, it may be necessary to discuss the advisability of hysterectomy or myomectomy, according to the situation of the tumour. If the latter is pedunculated it can, of course, be easily removed; indeed, this should be done in all cases as soon as the condition is recognised, as the risk of such a tumour sloughing after delivery is very considerable. Deciduoma malignum admits of but one treatment—immediate and complete hysterectomy.

Post-hæmorrhagic Collapse.—The very favourable results which attend the early recognition and treatment of post-hæmorrhagic collapse are so marked that it is deemed advisable to devote a separate paragraph to this condition.

*Symptoms.*—The symptoms of collapse due to excessive loss of blood are most characteristic. At first there is no noticeable change in the condition of the patient save a slight increase in the frequency of the heart. As the hæmorrhage continues this becomes more marked, and the pulse at the same time becomes small and feeble. Gradually the aspect of the patient becomes blanched, the conjunctivæ especially being of a pearly white; respiration is more hurried, and the patient frequently sighs. This condition, which is known as air-hunger, is the result of the lessened amount of oxygen which the diminished blood-stream carries to the tissues and the medulla oblongata. If the temperature is taken, it is found to have fallen from one to three degrees. As the hæmorrhage continues, the above symptoms become more marked. The pulse becomes uncountable and finally imperceptible, and the body is covered by a cold sweat. Hurried respiration is replaced by dyspnœa, and the patient, struggling for breath, requests to be raised as high as possible. If this is done she probably loses consciousness momentarily, or the sudden elevation of the head may be even sufficient to cause the final failure of the heart. She gradually becomes more and more restless, complains of inability to see, and finally becomes comatose, with perhaps occasional convulsive movements.

*Treatment.*—When a patient loses a large quantity of blood, death threatens. This occurs, not because there is an insufficient quantity of blood in the body, but because the blood-vessels have not as yet had time to suit their capacity to the diminished amount of fluid which they now contain. As a matter of fact, a woman greatly collapsed from post-partum hæmorrhage is said to have as many red blood-corpuscles in her body as an anæmic girl. In consequence of the unfilled condition of the vessels, blood does not return to the heart in sufficient quantities; the latter has nothing to contract upon; as a result its contractions become more and more feeble, and an insufficient quantity of blood is sent to the brain. In consequence of the resulting anæmia of the brain feeble stimuli are transmitted to the heart, which fails still more, a vicious circle being thus established. Reasoning from this we see that, to successfully combat the tendency to cardiac failure, our treatment must be directed towards three points:—

(1) The heart must be directly stimulated. Direct stimulation of the heart can be performed by the administration of alcohol by the mouth; by the hypodermic injection of ether, strychnine, or brandy; by the rectal injection of brandy or coffee; and by the use of hot fomentations over the præcordial area. In administering alcohol by the mouth, we must be careful not to give it in such large quantities as to cause vomiting. Half an ounce may be given at first of a mixture of one part of whisky or brandy in two parts of water, followed by a teaspoonful of the same every five or ten minutes. From twenty minims to a drachm of ether may be injected hypodermically, and from $\frac{1}{25}$ to $\frac{1}{10}$ of a grain of sulphate of strychnine. Several syringefuls of brandy may be used instead of ether; the latter is, however, preferable. From half an ounce to an ounce of brandy or whisky, mixed with from four to eight ounces of strong, hot coffee, may be injected into the rectum.

(2) The diminished quantity of blood must be limited as far as possible to the vital organs of the body, *i.e.* the brain and viscera. This is a most important point, and one which is frequently forgotten during the carrying out of the necessary measures for checking the hæmorrhage. The even momentary diminution in the amount of blood which is going to the brain, due to some sudden elevation of the patient's head, may prove fatal. While the patient is in the cross-bed position all pillows must be removed from beneath her head; and if her condition is serious, the limbs must be tightly bandaged from below upwards, in order to drive the blood from them to the more important parts of the body. So soon as the bleeding has been checked, and the patient has been returned to bed, the bottom of the latter must be raised from six inches to a foot by placing bricks or other sufficiently firm support beneath the legs. Subsequently, as the patient improves the bandages may be removed, and the foot of the bed brought gradually back to its former level.

(3) The amount of fluid in the blood-vessels must be increased. The amount of fluid in the blood-vessels can be increased in the following ways: by administering abundance of fluid by the mouth; by rectal injections of salt and water; by infusing saline solution directly into a vein, or into the subcutaneous connective tissue. As thirst is always present to a marked degree in these cases, it is never difficult to get the patient to drink large quantities of fluid as soon as she has rallied somewhat from her collapse. It is not, however, a method of increasing the fluid in the body which can be adopted at first, as sufficient quantities to have any effect in this direction would almost certainly cause vomiting. Rectal injections of saline solution of the same strength as that infused into a vein (0·6 per cent; roughly, a tea-spoonful of salt to a pint of water) will be absorbed most quickly. From one to two pints may be given, and it must be injected very slowly, as otherwise the patient will not retain it. The difficulty of retention of the fluid is often hard to overcome, and consequently in urgent cases one or other of the two remaining proceedings is usually adopted.

Direct intravenous infusion of saline solution is the most rapid method of increasing the amount of fluid in the blood-vessels. It is a course of procedure which, while it has many supporters, has also a number of opponents on the grounds of its danger and uselessness. If it is carefully carried out, the risk attending it is by no means great, while doubts with regard to its usefulness are most probably due to the fact that it is suffering at present from the results of previous over-estimation. Intravenous infusion will not bring a patient who is in the last stage of collapse from hæmorrhage back to life, but, if it is performed before this stage is reached, it will in all probability prevent her from even falling into such a condition. To render the proceeding of use, a sufficient quantity of fluid at a proper temperature must be infused. The necessary amount will vary between three and six or even eight pints; no definite quantity can be fixed which will suit all cases, but the infusion must be continued until there is a marked increase in the volume and strength of the pulse. The solution is used at a temperature of 100° to 102° F. In order that the proceeding may be as free from danger as possible, everything used in the operation must be sterile, and due precautions must be taken to prevent the entrance of air along with the fluid. The apparatus used consists of the following: a glass or metal funnel capable of holding at least two ounces; a rubber tube of about three feet in length; a small silver or white metal cannula with a blunt point; and a scalpel, dissecting forceps, small needles, needle-holder, and fine silk. The operation is performed as follows:—Tie a bandage round the upper arm sufficiently tightly to compress the veins but not the arteries. By this means the veins below the bandage stand out sufficiently to be seen, and a suitable one can be selected. Expose the latter by means of an incision about an inch in length made directly over it, isolate a small portion of it, and slip two silk ligatures beneath it; the distal ligature is tied to prevent hæmorrhage. A longitudinal incision of sufficient length to admit the tip of the cannula is made in the vein, and the cannula is introduced, care being taken that it is filled with saline solution. Next tie with a single knot the proximal ligature in such a manner as to compress the vein against the cannula, in order to prevent the escape of fluid, and remove the bandage which was compressing the arm. Before the cannula is introduced the entire apparatus must be filled with saline solution, its escape being prevented by pressure upon the tube. The fluid is now allowed to flow, an assistant taking care that the funnel is always full, and that no air gains admission. By holding the funnel from 10 to 18 inches above the patient, a sufficient pressure is obtained. As soon as the required quantity of fluid has been infused the cannula is removed, the vein cut across, the second ligature tied tightly, and the skin wound closed with sutures.

Infusion into the cellular tissue has been substituted by many for intravenous infusion on account of the greater ease with which it is carried out. Kelly, who prefers it to all other means of infusion, injects the fluid into the sub-mammary cellular tissue. For this purpose he uses graduated bottles capable of holding a couple of pints, to which a tube 6 feet in length is connected. A long, slender, and sharp aspirating needle is fastened to the other end of the tube. The solution used is the same as for intravenous infusion, and a head of 6 feet is required to make the fluid run. To perform the operation, the breast after careful disinfection is seized in the hand and lifted as far off the chest wall as possible. The needle, with the saline solution flowing, is then passed through the skin at the base of the breast and deeply into the connective tissue, taking care to keep clear of the gland structure. The fluid then runs in of its own accord, and as soon as no more will flow the needle is withdrawn. A piece of adhesive plaster fastened over the opening will prevent its subsequent escape. The breast will hold from a pint and a half to two pints, and the time required to infuse this amount is about twenty minutes. A similar amount can be infused under the other breast at the same time, if necessary. Instead of the breast, the fluid may be infused into the connective tissue of the buttock; but the former site is preferable.

The above is a short description of the immediate treatment necessary in post-hæmorrhagic

collapse. It must not, however, be thought that, as soon as the patient has rallied, all danger is at an end. The resultant enfeebling of the circulation carries in its train many dangers from which she cannot be regarded as safe for a considerable time. The most common of these is cardiac syncope, coming on at any attempt at exertion. Pulmonary embolism may also occur, due to the detachment of a thrombus whose formation has been favoured by the weak action of the heart. Crural phlegmasiæ may occur from a like cause, and, as happens in all debilitating conditions of the patient, the natural resistance of the system to septic invasion is so lowered that the risk of infection is greatly increased. In consequence of the tendency to cardiac failure, the patient must not be allowed even to sit up in bed during the first week or so, and all attempts at raising herself must be strictly forbidden. The process of getting up must be a most gradual one, and even after she is able to walk about all sudden or violent exertion must be carefully guarded against. In order to promote her convalescence, the administration of iron in tolerably large doses will be found of considerable benefit. Careful attention to the dietary and the judicious use of stimulants are also matters of, perhaps, vital importance.

## Injuries to the Generative Organs during Labour

A. Lacerations during Labour—
1. Rupture of the Uterus—
*Causation* . . . . . 264
*Position of Rent* . . . . 265
*Varieties* . . . . . 265
*Evidences* . . . . . 265
*Diagnosis* . . . . . 265
*Prognosis* . . . . . 266
*Prophylaxis* . . . . . 266
*Treatment* . . . . . 266
2. Laceration of the Cervix Uteri (Infravaginal portion) . . . 267
3. Laceration of the Vagina—
*Causation* . . . . . 268
*Position of Rent* . . . . 268
*Evidences* . . . . . 268
*Dangers* . . . . . 268
*Treatment* . . . . . 268
4. Injuries to the Pelvic Floor—
*Anatomy* . . . . . 269
*Position of Injuries* . . . 269
*Prophylaxis* . . . . . 269
*Treatment* . . . . . 269
5. Injuries to the Perineum—
*Varieties* . . . . . 270
*Causation* . . . . . 270
*Results* . . . . . 271
*Prophylaxis* . . . . . 271
*Treatment* . . . . . 271
6. Injuries to the Pelvic Articulations . . . . . 273
7. Injuries to the External Organs of Generation—
*Laceration* . . . . . 274
*Vulva Hæmatoma* . . . . 274
B. Injuries the Result of Prolonged Pressure—
*Sloughing* . . . . . 274
C. Acute Inversion of Uterus . . 275

Although injuries to the genital tract during labour are actually more common in multiparæ, they occur more frequently in primiparæ when the cause is pelvic obstruction, or rapid labour, for the passages are being dilated for the first time. This is especially the case when the obstruction is in the soft parts, and the "pains" are strong.

The consideration of the subject will be discussed under two main heads, viz. :—

A. Lacerations and injuries during labour.

B. Sloughing, due to crushing or to prolonged pressure during labour.

### A. Lacerations during Labour

1. Rupture of Uterus.—This accident is said to occur about once in 3000 cases.

*Causation.*—(*a*) Predisposing Causes.—Previous operations on the uterus, involving discontinuity of the uterine muscle fibres. Irregularities of the pelvic walls, such as bony ridges on the sacral promontory, near the pectineal eminences or prominent ischial spines.

(*b*) Direct Causes.—(1) *Rapid Labour*, especially in primiparæ.

(2) *Prolonged*, especially *obstructed labour*, such as occurs with contracted pelves, pelvic tumours, cervical or vaginal constrictions, or where there are fœtal malpresentations or deformities. In obstructed labour, when tonic contraction of the uterus is being produced, the course of events is as follows :—

The muscles of the fundal zone and of the uterus are acting vigorously, and the lower zone and cervix, relaxed by the process of polarity, are being drawn up, stretched over the presenting part, and getting constantly thinner. Bandl's ring, the lower limit of the retraction area, not felt at all in normal labours, is becoming more and more marked, and after a time can be felt some fingers' breadth above the pubes by the external hand. The "pains" gradually lose their intermittency, and a cramp-like continuity of pain is established, and all the local and constitutional evidences of tonic contraction of the uterus, already described in the article on Precipitate and Prolonged Labour, p. 207 *et seq.*, are observed. It is this thinned-out portion of the uterus which may rupture. Under such circumstances, version, or other inappropriate operation, may cause rupture.

In such cases ergot greatly increases the risk of rupture, for it tends to cause continuous

uterine action, and tonic contraction is more speedily produced.

(3) *Direct Violence.*—Instances have been recorded of women being kicked or run over, or tossed by bulls, with resulting rupture of the uterus.

(4) *Criminal Attempts at Abortion.*—In such cases the uterus is more usually perforated than incised or lacerated.

(5) *Spontaneous Rupture.*—This somewhat obscure accident may occur as early as the eighth or tenth week from the rupture of an "interstitial" gestation, but need not be further detailed here; nor need much be said of those very rare cases of spontaneous rupture, stated to have occurred in the later months, before any evidence of labour has appeared, and which are impossible to explain by merely assuming fatty or other degeneration. A possible explanation of such cases is that the uterus had been some time previously operated on, *e.g.* "rapidly" dilated, and that partial rupture had then occurred, with subsequent union by cicatricial unyielding tissue. The author knows of one case where a woman died suddenly at the beginning of labour, who a year previously had the inverted cornu of the uterus removed unintentionally by the wire écraseur during the removal of a fibroid polypus.

*Position of the Rent in Ruptured Uterus.*—The rent is usually in the lower zone of the uterus, and posteriorly and to the left. The line of laceration is at right angles to the direction of greatest tension, and may therefore be either longitudinal or transverse; but is usually obliquely longitudinal, and as such likely to extend through the cervix into the vagina. If the cervix is nipped between the head and the pelvic brim, the lower segment of the uterus will give way first; but if the cervix be drawn up, as usually occurs, the rupture may begin in the cervix, and may, unless it is a transverse one, extend downwards to the vagina, or upwards into the uterine lower segment.

*Varieties of Rupture.*—If the rupture is "complete," *i.e.* through all the coats and covers of the uterus, the peritoneal cavity is generally opened up posteriorly. If the tear is oblique or lateral, the cellular tissue of the broad ligament may be opened up, or, if the rent is anterior, the base of the bladder may be torn, in either case without the peritoneum being reached. In "incomplete" ruptures, where the peritoneal investment is not torn, it may nevertheless be extensively stripped off from the uterine muscle, and the sac thus formed may be distended by blood, by the placenta, or even by parts of the fœtus itself.

*Action of the Uterus after Rupture of the Lower Segment.*—If empty, the uterus would contract as firmly down as if its contents had been normally evacuated, and its size would be that of the normal uterus at the end of the third stage.

*Symptoms and Signs.*—Occasionally rupture occurs without any premonitory symptoms having been noticed, owing to absence of skilled observation. As a rule, however, if rupture be imminent, the "pains," previously intermittent, will have become continuous, and will be felt mainly in the lower abdomen owing to the continuous tension of the uterine muscles and ligaments. There will be constitutional and local evidences of tonic uterine contraction, with Bandl's ring well marked below the navel. When rupture has occurred there will be sudden pain and collapse following an ordinary "pain," if intermittency was still present. There is usually internal hæmorrhage, and, unless the presenting part filled the passage, some would also escape *per vaginam*. The presenting part may be felt to have receded, or to have totally disappeared, but, if fœtal impaction had occurred, no difference would be noticed. In "complete" rupture both the child and placenta might be in the peritoneal cavity. If rupture had taken place over an after-coming head, it might not be at first suspected, especially if the patient were anæsthetised, and might only be discovered when the hand was inserted to remove what appeared to be a retained placenta. More rarely the child escapes into the peritoneal cavity at the moment of rupture, and the placenta is subsequently normally expelled. If the rent is "incomplete," the child may have partially escaped from the uterus into a sac formed by the stripped-off peritoneum. In "complete" rents the bowels may protrude into the vagina, or even appear externally.

*Diagnosis of "Complete" Rupture.*—In the event of being suddenly called to a patient suffering from sudden collapse during labour, the diagnosis has to be made mainly between ruptured uterus and concealed accidental hæmorrhage. The distinction is, however, obvious; for whilst the aspect of the patient, the severe and prolonged shock, the evidences of internal hæmorrhage and the recession of the presentation, may be common to both disasters, the occurrence of the collapse in the second stage of labour, and the small size of the retracted uterus, point strongly to ruptured uterus and away from concealed accidental hæmorrhage, where the collapse occurs before or during the first stage, and the uterus is over-distended and tense. If, in addition, the child is felt to be outside the retracted uterus, or if the rent can be felt *per vaginam*, the diagnosis of rupture is certain.

*The diagnosis of "incomplete" rupture* is often impossible, and may not be suspected before delivery, and would then only be known by the passage of the hand into the uterus, and the detection of the partly stripped-off peritoneum, or the formation of a subperitoneal or broad ligament hæmatoma. If the placenta or a portion of the fœtus lies outside the uterus in

the sac thus formed, the shock would approximate to the severe shock of "complete" rupture, and that accident would be suspected.

*Prognosis.*—In all cases, probably 60 per cent of the mothers die either from shock or hæmorrhage, or at a later stage from septicæmia, and at least 90 per cent of the children.

*Prophylaxis.*—Whenever possible, the accoucheur should satisfy himself that any woman wishing to be attended by him at her approaching confinement has not a contracted pelvis. If a cursory abdominal examination and a manual palpation of the pelvic crests do not satisfy him, precise external and internal measurements should be made, and if pelvic contraction be discovered, labour should be prematurely induced at the appropriate date. If not seen till "in labour," examine early, and rectify any malpresentation promptly, and deal at once with any obstruction by version, perforation, decapitation, or by other indicated operation. If tonic contraction be present, avoid giving ergot or attempting version, but at once evacuate the uterus by forceps, or perforation if the head presents, or by decapitation if the lie is transverse. Rupture may be said to occur almost always in cases in which the earlier significance of the physical signs has not been observed or appreciated.

*Treatment.*—(1) *When the rupture is "incomplete"* and there is not much stripping off of the peritoneum, antiseptic drainage *per vaginam* is usually all that is required.

*Drainage* should be effected as follows:—First carefully wash the external genitals, and gently douche the vagina and the lower end of the rent (the peritoneum being unopened), taking care to allow all the injection to return at once. Drainage may be adopted by means of iodoform (10 per cent) gauze, or by india-rubber tubing. If tubing is used, it should be stitched to the lower end of the rent; but, as a rule, gauze lightly packed into the rent and allowed to loosely fill the vagina and appear at the outlet, is the best drain. It may be possible to suture the vaginal part of the rent if the tear has extended downwards. The gauze may be left in for as long as a week, if the temperature shows that drainage is effectual. If the temperature rises, remove the drain, syringe out the cavity still remaining, and drain again. If the bladder be torn, and the accident were at once discovered, an immediate operation might be tried, and a retention catheter tied in; but, as a rule, it would be best to await the partial healing and contraction of the wound, dealing with it subsequently as a secondary operation for vesico-vaginal fistula.

(2) *When the Rupture is "Complete."*—If the child is born, and the placenta has escaped into Douglas's pouch, it can usually be easily removed by the hand, with antiseptic precautions, and the case treated by vaginal drainage; but if the rent is extensive, or there is evidence of internal hæmorrhage, and the shock already present be not very severe, abdominal section is indicated. If the child is not already born, and the bulk of it is evidently in the uterus, attempts may be made to extract by forceps; but if this fail, knowing that the child is almost certainly dead, the head should be perforated, or if it be a transverse lie, decapitated, the body being then extracted by the arm, and the head by digital traction on the mouth if the pelvis be normal, or by perforation and crushing if contracted. If the child be in the abdominal cavity, or being partly in the abdominal cavity, is gripped by the uterus, it should be at once removed by abdominal section. If the child be already born, or has been delivered by the accoucheur, let the hand be passed up and the passages carefully examined under ether, so that the extent and nature of the laceration and the indication for treatment can be accurately determined upon. In a word, if the rent be very extensive, and hæmorrhage is evidently going on, if the child be in the abdominal cavity, or if the bowels protrude, abdominal section is essential; otherwise, as Drs. Herman and Herbert Spencer have recently shown, vaginal gauze drainage is all that is required. The following are the conclusions come to by Dr. Spencer:—

In the treatment of rupture of the uterus—

(1) *Abdominal section* is rarely required, and almost solely in cases where the fœtus has passed completely or in great part into the peritoneal cavity. It should be performed rapidly under local infiltration anæsthesia, and should be followed by flushing of the peritoneal cavity with normal salt solution and by suture of the tear, if possible, or, if this be not possible, by packing the tear with iodoform gauze and draining by the vagina or abdomen.

(2) *Abdominal hysterectomy* is hardly ever necessary; when the broad ligaments are so much damaged as to endanger the vitality of the uterus, vaginal hysterectomy should be performed.

(3) All incomplete tears implicating the broad ligament, and most complete tears, should be treated by *packing the rupture per vaginam with iodoform gauze* after removing clots and fluid blood.

If the abdomen is opened primarily for the extraction of the child, or for the arrest of internal hæmorrhage, and it is found that the torn surfaces can be accurately adjusted by suturing, it would be advisable to do so, and the following rules may be worth noting:—

*Suture of Uterine Laceration.*—This operation can only be done thoroughly after abdominal section. Suturing *per vaginam*, if the tear is above the vaginal portion of the cervix, is impossible. Let the abdomen be opened in the mid-line in the usual way, and if the rent is

anterior it is at once seen, and can often be sutured with the uterus *in situ*. If the tear is posterior, it is best to turn out the uterus and bring it well forward, so as to expose the torn surface. Suture the rent as in Cæsarean section, using deep sutures of silk or silk-worm gut, two-thirds of an inch apart, passing through the peritoneum and muscles, and avoiding (according to present-day teaching) the decidual lining. Superficial sutures of catgut or silk to accurately adjust the peritoneum should then be passed between the deep sutures. If the torn surfaces are not very accurately united, pockets are left, and the suturing will do more harm than good. (See Fig. 62, p. 273.)

There seems no good reason why the decidual lining should not be included in the deep sutures; for if it is not included, the decidua gapes, and allows the uterine secretions to reach the sutures, the danger of which is the main reason why it is advised that the lining membrane should not be touched.

When the tear is fundal or anterior, and the uterus does not need to be turned out of the wound, the Trendelenburg position is advantageous, as it keeps the intestines out of the way. If gauze draining *per vaginam* is adopted after abdominal section, a strip of gauze should be passed from the abdomen and drawn down *per vaginam* to the vulvar outlet. In such cases the catheter should be used till the gauze is removed.

2. Laceration of the Cervix Uteri—(Infra-vaginal portion).—Slight unilateral tears (usually on the left side) or bilateral, or even slight stellate tears, are almost universal in first labours, and, being so common, have proved valuable as probable indications of the previous passage from the uterus of some large body, such as a viable fœtus. Such tears hardly ever produce symptoms, but will, of course, add to the risk of sepsis if the lochia should become infected. The more serious cervical rents will now be considered.

*Causation.*—Rigidity of the cervical tissue is one cause, and this may be due to so-called spasm, the result of absence of the normal polarity, or to the presence of old cicatricial tissue, or to a fibroid in the cervical wall, or to malignant disease. The lacerations are usually longitudinal, and, as a rule, on the left side; but sometimes, in malignant disease, a complete ring of cervical tissue may be torn off, owing to its extreme friability. If these conditions are present, or if the rigidity is so marked that there is insuperable obstruction, the uterus is apt to give way in its lower zone, and the cervix might then only be torn secondarily. If the cervical rent be primary, it may extend outwards into the cellular tissue at the base of the broad ligament, upwards into the uterus, downwards into the vagina, or forwards into the bladder.

Obstetrical operations, such as version with forcible extraction, or the use of forceps when the cervix is undilated, may also cause severe rupture of the cervix.

*Dangers.*—If the rent is confined to the infra-vaginal part of the cervix the danger is small, though troublesome hæmorrhage from division, or partial division, of a branch of the uterine artery may ensue. The risk of subsequent sepsis is also increased. If the tear extends beyond the cervix, the risk is increased both as regards immediate hæmorrhage and subsequent septic absorption through the wound, producing probably a septic parametritis. Subinvolution not infrequently follows cervical tears.

*Evidences of Cervical Laceration.*—A tear of the cervix without extension into the uterine body does not produce shock. The evidences, if any, would be sudden and unexpected progress being made in a somewhat protracted labour, with subsequent greater rapidity. There might also be rather smart arterial hæmorrhage immediately after the birth of the child, often before the placenta is born; whilst the uterus is nicely retracted, showing that the hæmorrhage is not coming from a relaxed placental site. Careful vaginal digital examination would reveal the cervical rent, and the hot blood could be felt coming from the apex of the tear. If a duck-bill speculum were used, and the cervix drawn down by a volsellum forceps, the exact nature and extent of the tear would be apparent.

*Prophylaxis.* — A rigid cervix often relaxes after warm water injections, or after passing up a tampon of cotton-wool soaked in glycerine. This encourages glandular secretion, and makes the somewhat dry cervix moist. A physiologically active organ is always in a state of relaxation. Cocaine is said to enhance the effect of the glycerine by allaying any local hyperæsthesia; chloral in doses of thirty grains, repeated in an hour, also encourages relaxation. If a fibroid is present in the cervical tissue, it may be enucleated. If malignant disease be present, the question of Cæsarean section must be considered. Cicatricial contractions may need division. Forceps should never be used during the first stage of labour merely with the object of shortening its duration. No obstetrical operations should be attempted, and ergot should never be given until the cervix is fully dilated, or at all events dilatable. The first stage of labour should not be interfered with in normal cases, and the membranes should be left intact till the completion of their functions.

*Treatment.*—There is no need to suture all tears as a matter of routine, unless they have extended into the broad ligament, or there be serious hæmorrhage. If the uterus remains firmly contracted, the hæmorrhage must be proceeding from some laceration below the retracted portion of the uterus; and the finger may at once detect the cervical tear, and the

surfaces by means of a Hagedorn's small half-circle needle with silk-worm gut, as in Fig. 59, or by a rectangular needle set in a handle (see Fig. 61). Drainage and antiseptic douching are especially necessary if the pouch of Douglas, or the cellular tissue of the broad ligament, is opened up. The gauze drain should be left *in situ* for at least three days.

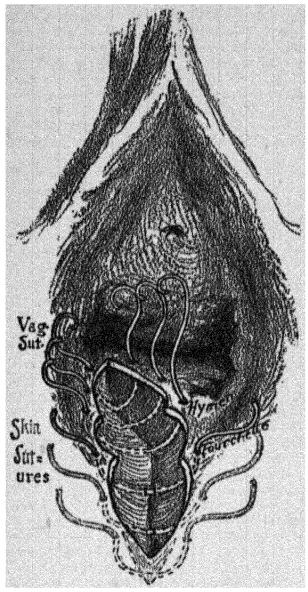



FIG. 59. — Laceration of the pelvic floor extending half-way to the rectum, with sutures properly placed ready for tying. (Norris.)

4. INJURIES TO THE PELVIC FLOOR. — *Anatomy.* — The pelvic floor, bounded externally by the skin and internally by the peritoneum, consists of a diaphragm of muscles with coverings derived from the pelvic fascia, supported from below by a more superficial series of smaller muscles, fascia, and connective tissue padding, the whole covered by skin.

*The pelvic diaphragm* consists of the powerful levatores ani and coccygei muscles, which practically shut off the pelvic outlet, allowing the rectum and vagina to pass through, and to be supported by rather intimate fusion of their muscle elements with those of the diaphragm. These two muscles constitute a sling, attached to the pubes in front, and, sweeping almost horizontally backwards, embrace the vagina and rectum, and are attached posteriorly to the coccyx. The levatores ani are attached along both sides from the back of the pubes, the "white line" of pelvic fascia, the ischial spines, and lesser sciatic ligaments, and then unite with each other and with the coccygei muscles along the middle line to complete the diaphragm. The muscles then curve downwards and inwards to the lower ends of the vagina and rectum, helping to form the internal sphincter of the latter, and uniting behind the rectum along the mid-line of the perineum till they reach the coccyx.

*The pelvic fascia* divides into two layers along the "white line." The upper, visceral, or recto-vesical fascia covers the upper surface of the levatores ani, and is a structure of great value in enabling the pelvic floor to resist undue intra-abdominal pressure.

The lower layer of the pelvic fascia is the obturator fascia, covering the obturator internus muscle, and forming the external investment of the ischio-rectal fossa. A thin sheet is also given off from the pelvic fascia at the "white line"—the anal fascia—to cover the under surface of the levatores ani muscles.

The more superficial structures consist of accessory smaller muscles, the transversi perinei, the bulbo-cavernosi, and the erectores clitoridis, with the superficial pelvic fascia, continuous with the triangular pubic ligament, whose two layers fill in the pubic arch, support the urethra, and form an attachment to the anterior fibres of the levatores ani. The perineum, largely composed of the above-named structures, will be described later on.

The pelvic floor may be said to be composed of two segments, an anterior or pubic, and a posterior or sacral, the vaginal cleft being between. In labour the anterior segment is drawn up, whilst the posterior is forced down, and is stretched by the presenting part. Injuries, therefore, to the tissues of the pelvic floor during labour almost always occur in the posterior segment, which includes part of the perineum and the perineal body, whose injuries will be hereafter discussed.

*Nature and Position of Injuries to the Pelvic Floor.*—There is no doubt that fibres of the levatores ani may be torn or unduly stretched, or their attachments to bony or ligamentous points loosened. Occasionally it would seem that the pelvic fascia itself is injured, for the whole pelvic floor lies at a lower level than before labour, and is more influenced by intra-abdominal pressure than it should be, moving too freely with inspiration, coughing, etc. Schatz and Howard Kelly are firm believers in such injuries; but it must be remembered that although they undoubtedly occur, the fact that gaps are felt in the levatores ani after labour does not prove injury, for those muscles run in distinct bundles with spaces between, and these can sometimes be felt even in primiparæ. No post-mortem proofs of such lacerations have yet been published.

*Prophylaxis.*—The efficient treatment of obstructed labour and of too rapid labour should prevent these injuries.

*Treatment.*—If injury to the pelvic floor has been diagnosed, the patient must be kept in bed longer than usual; but if, as would usually happen, the diagnosis is not made until after some weeks, when secondary symptoms have arisen, she must be warned against prolonged standing, and against all occupations which produce downward pressure on the pelvic diaphragm. Moderate rest, avoidance of constipation, and of tight corsets or abdominal bands, should be insisted upon, and in some cases a suitable vaginal pessary may be worn till the pelvic floor is able to take its full part in supporting the uterus. Such patients often find temporary relief from a bandage with a perineal pad to support the perineum.

5. Injuries to the Perineum.—*Anatomy.*—The female perineum includes the very important "perineal body," which is a portion of the posterior segment of the pelvis, and its elastic yet resistant properties enable the recto-vaginal septum to undergo great distension during labour.

The perineal body is triangular in vertical section, and its boundaries are the posterior wall of the vagina in front, the anterior wall of the rectum behind, and the integument covering the perineum between the vagina and anus below. It may roughly be said to extend laterally as far as the ischial tuberosities.

The structures contained in the perineal body are well seen in Fig. 60, and consist of fibre of

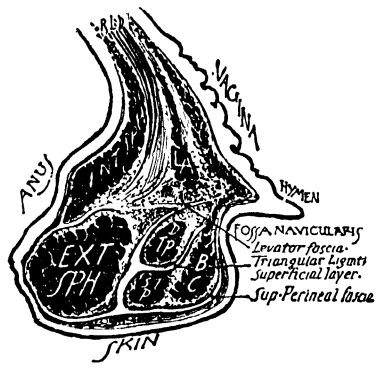



Fig. 60.—Sagittal section of the perineal body showing its component structures. (Norris.)

the levatores ani, superficial and deep transversi perinei, and bulbo-cavernosi, with the internal and external sphincter ani muscles, and layers of fascia from the anal and superficial perineal fascia, and from the triangular ligament.

*Nature of the Laceration.*—In primiparæ there is almost always a tear through the hymen, generally a little on one side of the central line, and often at several other points round its vaginal attachments. The main tear in the mid-line extends into the fossa navicularis, and usually will pass beyond this and lacerate the anterior edge of the perineum, the posterior fourchette. Deep hymeneal tears necessarily pass through the mucous membrane and invade the subjacent connective tissue, and, during the process of healing, the pieces of hymen are separated as small islets, or tubercles, by intervening cicatricial tissue, or modified mucosa, and are called *carunculæ myrtiformes*; the presence of these is evidence of the passage of a large body through the vaginal outlet, and would usually indicate the passage of a child of at least a viable age and size.

Tears which involve the perineum usually begin as above stated, extending backwards along the line of the median raphé through the sphincter and into the anus, or they may pass along the side of the mid-line, and follow the outer edge of the sphincter without actually destroying its integrity.

*Varieties.*—Perineal rents may be "complete," *i.e.* lacerating the vaginal mucous membrane, perineal body, and sphincter ani, so opening the rectum anteriorly; or "incomplete," where the vaginal mucous membrane and the integument with a larger or smaller part of the perineal body has given way, leaving the rectum intact. More rarely a tear, termed "central," occurs, where the rent seems to begin either on the vaginal mucous membrane or on the perineal surface, and extends deeply into the perineal body, working right through, leaving the sphincter ani and the posterior fourchette intact. Cases have been described where the child has been born through this "central" tear; but in the majority of such cases the anterior margin of the perineum would ultimately give way, and probably the sphincter ani as well. Still more rarely the perineal body seems to have given way in its centre, with a resulting perineal hæmatoma, which may suppurate a few days after the child is born.

As an apparent result of the hardening of the skin of the perineum by bicycling, extensive tears of the deeper structures of the perineum may occur. The author has seen a very tough perineal skin drawn forward over the head as it extended from under the pubic arch, and has subsequently found the skin to have been completely separated from its subjacent structures almost down to the rectum, accompanied by tearing of the vagina longitudinally, exposing the rectum laterally also.

*Causation.*—Perineal injuries are commoner in primiparæ, especially after thirty-five years of age, and the more rapid the labour the more likely is the tear to be severe. A large "passenger," especially a large head, predisposes, especially if the pains are strong. Sometimes the perineum is too long, for though its average length, from margin of anus to posterior fourchette, is just over an inch, it may vary from $\frac{5}{8}$ of an inch to over 2 inches. Rigidity of the parts, the presence of a small subpubic angle, or an altered inclination of the pubes, may lead to severe tears. In a vertex presentation the tear usually occurs when the sub-occipito-frontal diameter is passing through the outlet, especially when the supra-orbital ridges are emerging. In unreduced occipito-posterior presentations the perineum runs a great risk of rupture; for here, instead of the vulvar outlet being stretched by the circumference of the head when its diameter is the sub-occipito-frontal (4 inches), it has to make room for the head to pass when its diameter is the occipito-frontal (4½ inches). Sometimes the head seems to be passing through normally, and yet a severe rent suddenly appears, and it is found that the child's hand was applied to its chin, and that the sharp ridge of the fore-arm

had cut the stretched perineum. The shoulders also are very apt to commence or to increase a perineal tear, and may convert an "incomplete" tear into a "complete" one.

*Results.* — If the laceration is superficial, hæmorrhage is slight. Unless accurately sutured, raw surfaces, over which all lochial discharges must pass, are left, and, quite apart from distinct evidences of septicæmia, it is not infrequently found that there may be some pyrexia, 100° to 102° F., about the fourth or fifth day, when the lochia are a little faint in odour, and probably contain some chemical toxic elements. Pyrexia is usually absent if the wounds have begun to granulate up, absorption not taking place readily through granulation tissue.

If the laceration is "complete," incontinence of flatus and fæces is soon noticed, and the tendency of the sphincter ani, divided anteriorly, to pull itself and its adjacent structures backwards is soon apparent, the anus getting nearer the coccyx, and the antero-posterior length of the vulvar outlet becoming longer. This may produce a distressing sensation of gaping of the vulva, even permitting air to enter the vagina when the patient stoops.

If the perineum is severely torn, and no attempt at union is made, the anterior vaginal wall loses some of its posterior support; and if it is itself relaxed and stretched, it is apt to prolapse a little, and a cystocele may ensue. If this prolapse continues, secondary elongation of the supravaginal cervix, or prolapse of the whole uterus may follow, owing to the continuous dragging action of the cystocele. These secondary phenomena are, of course, commoner in cases where the vagina or pelvic floor has received injury; but there can be no doubt that in women obliged to follow laborious occupations a ruptured perineum is sometimes the starting-point of uterine prolapse.

*Prophylaxis.*—In ordinary labours with the vertex presenting, the accoucheur should wait till the occiput is protruding or causing perineal bulging, and until the nape of the neck is pressing against the arch of the pubes. Then the perineum should be supported with the palm of the hand, and the occiput should be urged forward, and at the same time too rapid delivery of the head should be prevented. This encouragement of the occiput to rotate under the symphysis can best be done just as a "pain" is passing off, before the recession of the head occurs. When the head has reached the outlet as far as its supra-orbital ridges, recession is less marked, and then the largest circumference of the head can sometimes be coaxed through as one "pain" is ceasing, and before the next commences. If, notwithstanding, the perineum threatens to give way, tell the patient to cry out, in order to avoid the reflex bearing-down; give chloroform deeply, and apply pressure on the occiput with both hands to keep the head back if possible. The best way to exert pressure upon the occiput is to apply the right hand from the perineal aspect, and the left between the legs from the vulvar aspect, and let the fingers interdigitate. Even then it is difficult to exert sufficient resistance.

*Episiotomy.*—If a tear seem inevitable, the small operation of episiotomy may be performed. This should be done exactly at the right time, or not at all. Wait till the supra-orbital ridges are about to be born, and then, if the perineum is going to give way, a thin, white, very tense line is seen almost all round the margin of the vulvar orifice. Take a probe-pointed knife; pass it on its flat, along the child's head, until its point has passed under this thin white line, about one-third of the distance between the central line of the perineum and the base of the vestibule. Then gently turn the handle till the cutting edge of the knife meets the tense line, which immediately gives way, and a notch is made, which, though afterwards looking only a quarter of an inch long, gapes to three times that length as the head is being born. Make a similar notch on the opposite side, and the head will almost certainly pass through at the next "pain," with very little deepening of these two notches, and with the perineum saved. Scissors may be used if preferred. It is best to put a single suture in afterwards, on each side.

Care should be taken that there is no complex presentation present, such as a hand near the mouth, with a sharp fore-arm to tear the perineum, and that the posterior shoulder, as it is born, does not deepen or cause laceration.

If the forceps are being used, rapidity of birth can usually be controlled, and the head can be coaxed over the perineum, and round from under the pubic arch between the "pains" at the right moment. When the head is distending the perineum the forceps may be removed, if it is thought that nature will then effect delivery without accident.

Anæsthesia, to the surgical degree, is very desirable where there is risk of laceration, and should always be given when any prophylactic manipulations are being adopted.

*Treatment.*—Immediately the child is born let the perineum be carefully examined, and let the nurse, and the patient too, if not under anæsthesia, understand what is being done, so that no charge of carelessness may be subsequently brought against the accoucheur. This particular charge, neglect of a torn perineum, is one so frequently brought against medical men, with a view to damages, that special care should be taken in making the examination, and if in doubt as to the need of a suture, let the accoucheur err on the safe side and insert one.

A good time to make the examination is immediately after the birth of the child, even before the placenta has been expelled, for the

patient is probably still under anæsthesia. It is quite possible to insert (tying them afterwards) the necessary sutures at this time also, especially if only perineal sutures are required, for the parts are then somewhat numbed.

Although trifling tears will heal without sutures if the legs are tied together, and the wound kept aseptic, all wounds of the perineum, except those which have merely torn the posterior fourchette, should be at once sutured.

The more extensive the tear, especially if the rectum be involved, the more necessary is it to suture the rent at once, though union will often take place if the operation is unavoidably postponed for a few hours.

*Preparations for the Operation.* — Let the patient be kept under anæsthesia if it is considered that sutures are needed. For the insertion of sutures for "incomplete" perineal tears anæsthesia is not essential, for the only pain is as the needle enters and leaves the skin, and that is often somewhat numbed at first. Keep the patient in the lithotomy position, by means of a Clover's crutch or an improvised roller-towel or sheet-sling. Clean the parts well with antiseptic lotion, and dam back any hæmorrhage coming from the uterus with antiseptic vaginal tampons. Let an assistant separate the labia well, and expose the torn surfaces. The parts should be shaved, if hirsute.

(1) *The Operation for "Incomplete" Rupture.* —The best needle is a rectangular one fixed in a handle, as recommended by Dr. Cullingworth (Fig. 61). Pass the sutures in such a way,

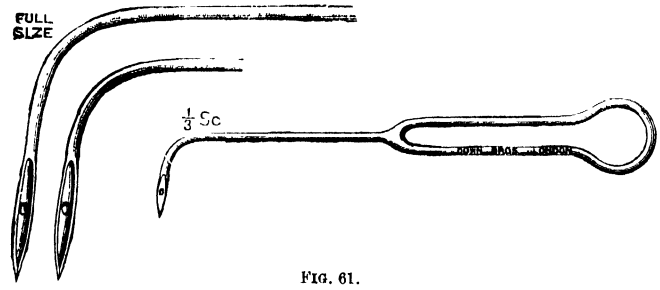



FIG. 61.

that when they are tied, the wound is entirely obliterated without any incurving of the skin or mucous membrane. In introducing the sutures, notice carefully if any spots in either of the raw surfaces are depressed or cupped. If so, it shows that muscle-fibres, *e.g.* of the sphincter ani, are cut through and retracted. These must be carefully caught in the sutures, and drawn to the surface. The rectangular needle, unarmed, should enter the skin or mucous membrane (Fig. 59) quite close to the edge of the tear, should pass deeply into the tissues under the base of the rent and out at the other side, "buried" throughout. The needle should then be threaded with silk-worm gut and withdrawn. The first suture should be inserted opposite the posterior angle formed by the tear, nearest the anus, and the other stitches, one-third of an inch apart, should then be inserted anterior to the first one till the anterior margin of the perineum is reached. After all the sutures have been inserted they should be tied in the same order. The following diagrams show the faulty and the correct methods of inserting sutures for the repair of incomplete ruptures (Figs. 62, 63).

The sutures should be removed in seven days, after the bowels have been well opened.

(2) *The Operation for "Complete" Rupture.* —When it is found that the sphincter is torn, and the rectal mucous membrane exposed, the patient must be arranged in the lithotomy position, in a good light. The rectal tear must be sutured first with catgut, the sutures being introduced first at the apex of the tear, the highest point from the anus. The best way to insert the sutures is as follows:—Let the rectal wound be put on the stretch by an assistant's hands, one on each side of the vulva. Steady the apex of the wound with dressing forceps, and take a rectangular, or, if preferred, a half-curved needle set in a holder, and pass it, unarmed, from the rectal aspect at the apex of the tear, as close as possible to the torn surface, at its very edge, without actually passing through the rectal mucous membrane itself, and pass it sideways into the tissues in such a way that it includes a little bunch of submucous and muscular tissue, and comes out again at the edge of the vaginal mucous membrane without actually entering the vagina itself. Then thread the needle, the eye of which is at its point, and withdraw it, leaving the catgut in the track made. Then pass the unarmed needle in a similar manner on the opposite side of the apex of the rent, and thread it when passed with the vaginal end of the catgut which was left in the tissues on the opposite side, withdraw it, and tie the catgut suture, so that its knot is in the rectum. Then pass succeeding sutures from above downwards, till the anus is reached, tying each before the next is passed. When tied, each suture may be at once cut short, or, as is found more convenient in practice, hold the one just tied in the left hand to steady the parts till the next suture is introduced and tied, and then cut short the previous one. If the sphincter ani is only partly cut through, a so-called purse-string suture, as advised by Dr. Percy Boulton, may be used, either without inserting any rectal sutures, or in addition to them, to add to their security. The purse-string silkworm-gut suture is passed completely round the rectal wound. It is made to enter at the edge of the skin at the anal end of the tear, and is passed along, buried, parallel with the cut rectal surface to the apex, and back again along the other side, and out again at the anal edge of the wound. It should be

buried sufficiently deeply at its first insertion to include the muscle of the divided sphincter (see Fig. 59, p. 269), for then, when tied, it accurately draws the sphincter forwards, so that

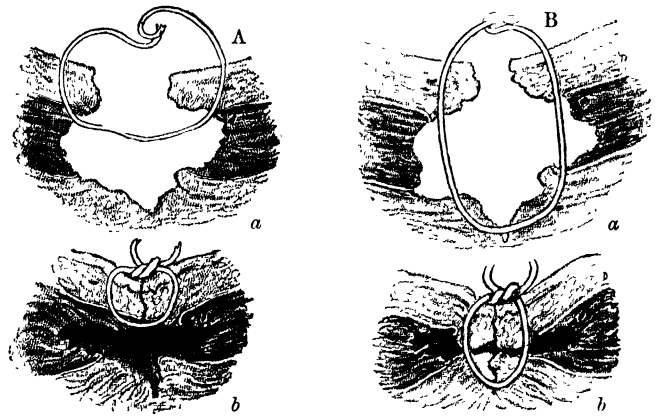



FIG. 62.—A, faulty method of suture, falling short of the bottom of wound and not catching all the muscle-ends: *a*, before tying; *b*, after tying. The latter figure shows dead space at the bottom of wound after tying; perineal body only partially restored. B, suture improperly placed: *a*, before tying; *b*, after tying. The suture *a* has too little lateral sweep, and it does not include the ends of all the retracted muscle-fibres at the sides of the wound; *b* shows the result, the pelvic floor being imperfectly restored. (Norris.)

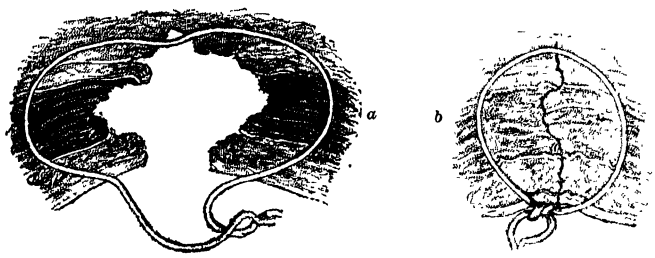



FIG. 63.—Shows full sweep of a properly placed suture: *a*, before tying; *b*, after tying. Even though the tear runs in different planes at different depths, the muscle-ends are held in apposition throughout the entire depth of the wound (Norris.)

the divided strands are in close contact, and at the same time it affords great support to the catgut rectal sutures already passed. As a rule, it should be inserted before the rectal catgut sutures are passed, but should not be tied till afterwards.

6. INJURIES TO THE PELVIC ARTICULATIONS.—*Causation.*—The pelvic joints are the sacro-iliac, the pubic, and the sacro-coccygeal. They are all united by cartilage, and are rendered more mobile during pregnancy owing to softening and hypertrophy of the cartilage, with increased development of the existing synovial pouches. The result is that during labour there is a yielding of the bones united by these joints, and a certain definite though inconsiderable gliding motion is permitted. This has been proved by Walcher, Pinzani, and others, and is the *raison d'être* of "Walcher's position" in difficult labour. The joints are unduly strained if there is any marked disproportion between the pelvis and the passenger. The pubic joint is necessarily divided into symphysiotomy, but unites promptly with fibrous union, unless antiseptics have broken down, when suppuration and destruction of the cartilage would ensue, and bony union eventually would take place. In this same operation the sacro-iliac joints are liable to be seriously injured if, at the moment of division of the inter-pubic cartilage, the legs are unsupported and allowed to fall outwards. The sacro-coccygeal joint is rarely injured, for it allows of very free movement. If, however, there has been previous dislocation and fixation of the joint, and especially if the coccygeal apex points forward, difficulty at the end of the second stage may arise, and fracture at the joint has been known to take place. More rarely dislocation of the coccyx has been known to occur during labour, the point of the coccyx then pointing backwards.

*Evidences of Injuries to the Pelvic Articulations.*—The main evidence of serious injury to one of the pelvic joints is dyskinesia — difficulty in walking—associated with local pain and tenderness. If the patient is not seen till some weeks after labour, the local pain and tenderness will have passed off, and no evidence may be left except the dyskinesia. To examine the pubic joint let the patient be placed on her back. A thumb placed in the vagina and applied to the back of the symphysis, whilst the other fingers of the same hand are over the joint, will enable intra-pubic mobility or tenderness to be determined, especially if one leg is passively moved up and down by an assistant. To examine the sacro-iliac joint, the patient should stand, supporting herself against a fixed point, and the physician should apply his ear to each joint in succession, whilst the patient flexes and extends the thigh of the same side. If there is much movement in the joint some creaking or crackling will be heard. In all such cases the effect of a tight girth round the pelvis—a large towel, for instance—is almost diagnostic; for the patient, who just before wobbled about, unable to walk without support, can now walk very fairly across the room, experiencing great support and relief. If the sacro-coccygeal joint is affected, an examination with one finger in the rectum, and the thumb outside, will enable the physician to grasp the bone, and judge at once of its mobility, its relations, and the tenderness of its joint. The main symptoms, at first, if the joint is inflamed, are coccygodynia (painful sitting), with some dyschezia (painful defæcation), and often pain when coughing or sneezing, owing to

traction on the coccyx by the muscles of the pelvic floor. After a time all these symptoms, except coccygodynia, disappear.

*Treatment.*—If the pubic or sacro-iliac joints are very tender, a blister will be the best treatment, with rest in bed. Later on, a firm binder round the pelvis, with avoidance of all exertion, is indicated, and will soon lead to the joint becoming normal again. If the sacro-coccygeal joint be affected, a blister often cures; but if the coccygodynia persists, or the coccyx is found fixed and displaced forwards or backwards, it may require to be forcibly readjusted, or more rarely to be excised.

7. Injuries to the External Organs of Generation.—(*a*) *Laceration of the Vulva.*—Tears through the hymen have already been discussed; but tears may also take place in the vestibule, or through the labia minora, or even extend into the labia majora, or the urethra may occasionally be injured near its external meatus.

*Evidences.*—The tears in these cases, unless the swelling of the parts should prevent, are at once seen on inspection. The hæmorrhage, unless some varicose vein or the venous plexuses in the labia majora are torn across, is not severe; but bleeding from a superficial tear in these vascular tissues may continue for many hours or days, and be overlooked owing to the presence of the lochia, and may produce profound anæmia.

*Treatment.*—Any tear should be at once closed with catgut, or silk, to check hæmorrhage, and to ensure primary union.

(*b*) *Vulvar Hæmatoma.*—Occasionally a pudendal vein gives way during the second stage of labour, and if the skin remains intact a vulvar hæmatoma results. Such an accident usually occurs in primiparæ, because in multiparæ the veins are more varicose and superficial, and tend to burst externally. If a large hæmatoma is formed, obstruction to the presenting part may result.

*Evidences.*—The usual symptom is severe pain in one labium majus, felt suddenly during a "pain," in the later part of the second stage, and not infrequently some shock results. On examination the swelling of the labium is found to be irreducible, dark in colour, bulging over the labium of the other side, tense and fluctuating, but gradually getting less elastic as the blood coagulates, and finally getting boggy from œdema round the effusion. There is no impulse on coughing. It is distinguished by its history, and by its physical signs, from distended Bartholini's gland, labial abscess, hernia, and varicose veins.

*Treatment.*—If there is definite, though slight, obstruction, and the head presents, deliver with forceps. If the obstruction is such that forcible delivery would bruise or tear the swollen parts, the tumour, after the vulva has been shaved, must be incised on the skin aspect, along the long axis of the labium. Turn out the clot, and apply pressure till the child is born, tying any bleeding point. If the wound can be made quite clean and free from any adhering clot, a few buried purse-string sutures will approximate the surfaces, and an attempt may be made with outside pressure to promote primary union. As a rule, such cavities do not thus heal, and may be packed with gauze and allowed to granulate up.

B. Injuries due to Prolonged Pressure on the Internal Generative Organs

*Causation.*—In some cases of obstructed labour there is extensive nipping of some parts of the uterus, cervix, or vagina, between the head and some bony point of the pelvis, most commonly the pubic symphysis. We have seen that obstructed labour often leads to tonic contraction of the uterus, and subsequently to thinning of the lower zone, and to laceration of the stretched tissues; but delivery may be effected without laceration, and yet the tissues, swollen from œdema and hæmorrhagic extravasation, lose their vitality. A slough then forms, and is thrown off in from four to ten days by a process of ulceration between the dead and living tissue; and if the slough be deep, it may include the lining membrane of a neighbouring viscus, such as the bladder, and a urinary fistula would result. If, in a contracted pelvis, the anterior lip of the cervix is nipped between the head and the brim, the part below the pressure becomes swollen, and this further delays labour. The part nipped soon loses its vitality, and eventually sloughs, and a utero-vesical fistula would result; or, if the whole anterior lip of the cervix sloughed, the fistula would practically be a vesico-vaginal one. In either case some bladder irritation and, possibly, some cystitis may follow, and some induration from cellulitis may also be found round the margins from which the slough had separated. When a ureter-o-vaginal fistula is formed, it is more usually the result of a laceration than of a slough. When a recto-vaginal fistula is formed it is generally at the perineal end of the vagina, and is usually due to a complete perineal rupture, with partial union by a bridge of tissue between two stitches, the others having given way. More rarely a slough forms opposite the sacral prominence, and an opening may be made into Douglas's pouch; but if so, the general peritoneal cavity is effectually protected by rapidly effused lymph.

*Evidences of Sloughing.*—Superficial sloughs along the vagina, and at the orifice of the vulva, are not uncommon, and are the result of pressure, and of the "glissading" of the tissues owing to the child's head pushing the tissues in front of it, detaching the mucous membrane from its deeper attachments, and depriving it of blood-supply. With antiseptic care these

superficial sloughs are unimportant. If the bladder has been laid open by a slough, it would be noticed that in from four to ten days urine would be coming away from the vagina, and the bladder would be found to be more or less empty. The exact position would probably not be known till some time after the fistula was diagnosed, for a satisfactory examination could hardly be made till the parts had involuted.

*Prophylaxis.*—All such cases of fistula, the result of delayed labour, are now relatively rare, owing to the forceps being used earlier, and to the adoption of other measures to prevent undue delay.

*Treatment.*—This would have to be delayed, and the fistula treated by a secondary operation after its precise character had been determined. To surgically treat a fistula, due to pressure, immediately after the separation of the slough, would certainly prove a failure, owing to the induration and lack of vitality round the margins of the wound, and the presence of some cystitis. Time should also be given for the wound to cicatrise, for it invariably gets smaller, and the operation therefore becomes less severe. In the meantime, all that can be done is to adopt some suitable paliative treatment, for, although a radical operation is useless during the puerperium, much relief can be afforded to the patient, and septic consequences can usually be averted. The vagina should be douched with some sedative solution, such as borax (dr. 2 to three pints), lysol (dr. 1 to three pints), or diluted Condy's fluid, and some antiseptic wool or wool-pads can be kept constantly applied to the vulva, or a suitable urinal can be worn. Women accustomed to wear diapers at menstruation do not object to such appliances as much as one would expect. In a few cases patients do not need to wear anything when recumbent, but all depends upon the exact position and extent of the fistula.

Six weeks after the labour an operation may be performed. (See "Vagina.")

### C. Acute Inversion of the Uterus

*Definition* . . . . . 275
*Morbid Anatomy* . . . . . 275
*Causation* . . . . . 275
*Evidence and Diagnosis* . . . . 276
*Prognosis* . . . . . 276
*Prophylaxis* . . . . . 276
*Treatment* . . . . . 276

*Definition.*—This form of uterine displacement is a more or less complete turning "inside out and upside down" of the body of the uterus, so that its lining membrane becomes external and its fundus the lowest portion of the body.

The fundus may be "completely" or only "partially" inverted.

*Morbid Anatomy.*—When the fundus becomes completely inverted it draws down with it, into the peritoneally-invested cup, part of the broad ligaments, with their pampiniform plexus of veins, the round ligaments, the ovarian ligaments, and sometimes the ovaries themselves, with part of the Fallopian tube, and more rarely part of the omentum. These so completely fill up the hollow that the physical examination may fail to feel the cup-like depression which theoretically exists.

Prolapsus of the uterus or vagina may be also present. In such cases the fundus, even when the placenta is not attached, may be quite outside the vulva.

*Causation.*—This accident is said to occur once in 200,000 labours, and may be both artificially and spontaneously produced.

Inversion cannot occur if the uterus is contracted. The body of the uterus must be completely relaxed.

(*a*) *Artificial inversion* may be caused in two ways:—(1) *Forcible expression during uterine relaxation.*—Expression of the placenta during the third stage of labour should only be practised during a "pain," otherwise indentation of the fundus may be induced. Partial inversion seems to temporarily paralyse that portion of the uterus, so that when a "pain" arrives the whole uterus contracts, except the inverted fundus, which is grasped and driven downwards in the line of least resistance, and a complete inversion may ensue. This may take place during the next pain, or after the lapse of some hours, or even some days after a partial inversion has been produced.

(2) *Traction on the umbilical cord* during uterine inertia. The fundus, with its adherent placenta, may be thus partially inverted, and this may be converted into a complete inversion either by "expression" or by further traction on the cord, or spontaneously.

(*b*) *Spontaneous inversion* may occur in several ways. As already stated, a partial inversion may be converted spontaneously into a complete inversion, but there is no doubt also that an inversion may be induced spontaneously *ab initio*. A short umbilical cord, or a cord rendered relatively short by being wrapped round the fœtus, may be the primary cause, and is, according to Herman, the way in which a considerable number of cases of inversion are brought about. An inert fundus, with a placenta partly separated, and hanging down into the body of the uterus, may be inverted exactly as a chronic inversion is produced by a fibroid polypus. Increase of intra-abdominal pressure, by the patient coughing, sneezing, or "bearing down," encourages inversion to become complete if cupping has been begun, and, according to some observers, this is thought to be sufficient to spontaneously produce the initial partial inversion.

*Evidence and Diagnosis.*—The main symptoms are sudden, severe collapse and hæmorrhage coming on during, or more rarely after, the completion of the third stage of labour. The collapse is caused by strangulation of the uterus and its appendages, and is only relieved by reduction of the displacement.

The condition of the patient resembles that due to rupture of the uterus (see p. 265); but the fact that the uterus cannot be felt over the pubes, and that the collapse has occurred in the third stage of labour instead of the second stage, will suffice to differentiate. If the placenta is adherent the mass will protrude beyond the vulva, and the diagnosis will be obvious.

If the placenta is detached the inverted fundus may, or may not, reach the vulva, and would have to be distinguished from a fibroid polypus. The absence of the uterine body from the hypogastrium will sufficiently exclude the diagnosis of a uterine fibroid, and if the hand be passed into the vagina behind the swelling, and pressure be made by the other hand, the exact condition is easily determined.

*Prognosis.*—The mortality is about 66 per cent. Death may rapidly ensue from shock or hæmorrhage, or may follow in a few days, if the case be untreated, from hæmorrhage or sepsis, or later on from exhaustion.

If the patient survive and the uterus be unreduced, the inversion becomes chronic and involution is greatly impeded, and the patient remains very ill and suffers from much pelvic pain and from continuous hæmorrhages and discharge.

*Prophylaxis.*—Forcible extraction of the child or placenta should be avoided in all cases of secondary uterine inertia. More particularly should expression from above, or traction on an adherent placenta from below, be avoided, except during a definite uterine contraction. If the cord is too short to permit delivery, it must be cut as soon as the passages are sufficiently dilated to admit the rapid delivery of the child by forceps. The relatively short cord wrapped round the child's neck or trunk should be unrolled or, if need be, divided. Good uterine contraction and retraction of the uterus must be ensured after the birth of the child.

*Treatment.*—The uterus should be immediately replaced by manual taxis under anæsthesia. The placenta may be detached if still adherent, and after the bladder has been emptied the inverted uterus should be grasped by the right hand, and steady pressure should be made in the direction of the pelvic veins, while the left hand is steadying the rim of the uterine neck from above. It sometimes hastens reduction to try and reinvert the parts of the uterus nearest the rim of the cervix, instead of pushing on the fundus alone.

Reduction should be very prompt whilst the uterus remains inert. Every hour increases the patient's shock and makes reduction more difficult. A rectal injection of two pints of saline infusion with a little brandy will rapidly relieve the patient after reduction, but does very little good till that is effected. Never give ergot till the uterus is reduced.

If the incident be discovered at once, reduction may be effected without anæsthesia; but if an hour or two have elapsed it will be essential to put the patient under complete anæsthesia. Manual reduction will usually fail after some days have elapsed, and reduction will have to be effected by Aveling's repositor (see "Uterus, Chronic Inversion").

Antiseptic vaginal douches should both precede and follow reduction.

## Labour, Operations

The following operations are here described:—

1. **Induction of Premature Labour** . . . 276
2. **Forceps** . . . . . 281
3. **Vectis** . . . . . 287
4. **Version** . . . . . 287
5. **Embryotomy** . . . . . 290
6. **Symphysiotomy** . . . . . 297
7. **Cæsarean Section** . . . . . 299

### 1. Induction of Premature Labour

History and Indications.—The induction of premature labour is defined as the artificial bringing on of labour before full time, but after the fœtus has reached an age at which it is viable. The induction of abortion implies the emptying of the uterus before the twenty-eighth week of pregnancy, for a fœtus born earlier than that cannot survive.

The idea of intentionally producing premature labour was derived by analogy from parturition occurring accidentally before term. The operation was originally designed for the sake of the child only. It was of British origin about a hundred and fifty years ago, being introduced in Germany some fifty years later, and in France at a still more recent date. Its morality and expediency were long questioned.

"About the year 1756, there was a consultation of the most eminent men in London at that time, to consider the moral rectitude of, and advantages which might be expected from this practice, which met with their general approbation." In 1800, Denman wrote of induction as a recognised and valuable operation in cases where pelvic deformity prevented the birth of a living child at term, and also in cases of habitual death of the fœtus *in utero* at or near the end of pregnancy, an indication which is now very rarely acted upon. At that time induction was not undertaken for the sake of the mother; for Denman wrote: "It ought not to be performed when the patient labours under any hazardous disease." At a later date, however, it became usual to induce labour with a view to checking the progress of any disease

which, while threatening the life of the mother, is due to or is aggravated by pregnancy.

To illustrate the relative frequency of the various conditions in which labour is induced in modern practice, it may be mentioned that in 44 cases reported by S. Korscheban,[1] 11 inductions were for contracted pelvis; 3 for osteomalacia; 14 for lesions of the heart; 7 for nephritis; 3 for pernicious nausea; 5 for tuberculosis; and 1 for perityphlitic abscess. The maternal mortality was 6 per cent, this being entirely in cases where the operation was undertaken on the mother's behalf, and was in no case traceable to the operation; 79·4 per cent of the children were born living. Apart from contracted pelvis, Heyman (see *infra*) has recently induced labour most frequently for threatened eclampsia, lesions of the heart, and tuberculosis.

Methods.—In reviewing the various methods by which labour may be induced, it is not necessary to mention the use of agents such as ergot and quinine, for the action of all drugs is much too unreliable for this purpose. The same applies to mechanical irritation of the nipples, and other devices which have from time to time been suggested for setting up uterine contractions.

The original method of inducing labour was simple puncture of the membranes, and for many years it remained the only one in use. It is sure to be followed by labour sooner or later, but delivery is by no means always ended during the life of the child. The bag of forewaters being absent, the fœtal head has to play the part of dilator, and is exposed to the pressure of the parts dilated during the slow progress of a "dry labour." Owing to its uncertainty as to time, and the undue risk to the child, puncture of the membranes has been given up in cases where induction is undertaken in order to allow of the birth of a living child through a narrow pelvis. In certain cases, however, where the indication is some condition threatening the life of the mother, such as eclampsia or accidental hæmorrhage, rupture of the membranes forms a useful initial step towards emptying the uterus.

Daily repeated digital dilatation of the cervix was the method which displaced puncture of the membranes. The objection to this method is that the practitioner may have to dilate the cervix daily for ten or twelve days before the onset of labour. The results, however, are excellent, and other measures may be adopted at any time if progress is too slow. In the year 1824, Professor Hamilton of Edinburgh was teaching and employing this method. Its value may be judged from the results of Moir,[2] who employed it from the year 1833 until the year 1884. During that time, he induced labour in 26 patients—eight times in one patient, five times in three, four times in one, thrice in ten, twice in three, and once in eight. Of the 72 children (twins once) 59 were born living and healthy, and 13 were still-born. The operation was thus successful as regards the child in 82 per cent of his cases. Moir induced labour 69 times without losing one mother, but the last two patients upon whom he operated died during the puerperium. He used to begin by passing through the cervix a metal rectal bougie, and turning it round so as to separate the membranes near the os. This was repeated daily, until "in the course of three or four days, but sometimes not until six or seven, the condition not only of the os and cervix, but also of the vagina, became entirely changed so as to be soft, relaxed, and dilatable, very much as at the full time." So soon as this occurred dilatation was continued with one and then with two fingers, once or twice daily, until pains began.

Vaginal douching with hot water was introduced as a mode of inducing labour by Kiwisch, in the year 1836. Repeated with sufficient frequency day after day douching will induce uterine contractions, but it is a very uncertain and slow method. The constant flow of water over the part sets up an unhealthy condition of the vagina, and renders it peculiarly liable to become the seat of septic infection by removing the natural discharge and the superficial epithelium. Its employment has been recently advocated by Larwey.[1]

Of late years the most popular method of inducing labour has been that of Krause. A flexible bougie (not a catheter) is introduced through the cervix, and pushed up between the membranes and the uterine wall until its point lies about seven inches above the level of the os internum. The portion of the bougie remaining outside the uterus is then coiled up or cut off short, and a pad of gauze is placed in the vagina in order to keep the bougie in position. When pains begin, which usually happens within a few hours, the bougie is withdrawn and labour is allowed to proceed naturally. There is considerable risk of puncturing the membrane when inserting the bougie, and the placenta may be injured and partly separated should the instrument chance to traverse that part of the uterine wall to which it is attached. These risks are naturally increased when, as is sometimes necessary, a second bougie is inserted after waiting in vain during some hours for the onset of labour. Briggs,[2] of Liverpool, has advised the insertion of three or four bougies together at the beginning, a course which increases the rapidity of the method, but also obviously multiplies its risks. The nature of the results which may be obtained by the use of the bougie

[1] *Monatschrift für Geburtshülfe und Gynäkologie*, Band ii. Heft 1, 1900.
[2] *Obstet. Trans. Edin*, vol. xxiii., 1897-98.

[1] *Archiv fur Gyn.*, Band lx. Heft 3, 1900.
[2] *Trans. North of England Gyn. Soc.*, Nov. 1893.

may be gathered from Heyman's[1] report of 107 cases, out of which 64·3 per cent of the children were born alive. There was no maternal mortality traceable to the operation, though the mothers died in certain cases in which the operation was undertaken owing to serious maternal disease. Leopold's average time for completing labour by this method is 80 hours 16 minutes. Briggs's average is less than half this time when three or four bougies are introduced together; and when the bougie is used, his modification should always be preferred to the original method.

In 1892, Pelzer introduced a method of inducing labour by injecting one or two ounces of glycerine through the cervix into the lower uterine segment between the membranes and the uterine wall. The glycerine is said to act in three ways: (1) as a chemical irritant; (2) by removing some of the liquor amnii by osmosis; and (3) by separating the membranes from that portion of the uterine wall surrounding the os internum.[2] This method has been said to be not only ineffective but dangerous, various inconveniences having followed its employment, namely, slow pulse, high temperature, cyanosis, hæmoglobinuria, and albuminuria. Out of thirty-five cases collected by Helme this method failed to bring on labour in only two instances, and in these two cases one patient was moribund when the glycerine was injected, while the other had already had three bougies in her uterus for several days. Four of the women died, two from eclampsia, and two from nephritis which was known to be pre-existent. Under certain circumstances glycerine is doubtless of use in favouring dilatation, but it is doubtful whether, unaided, it affords a good method of inducing labour. During the last few years very few cases have been reported in which it was used. Spinelli[3] has recently advocated another method of employing glycerine for this purpose. He packs in between the membranes and the uterine wall a strand of gauze soaked in ichthyol and glycerine.

Very many years ago Huter caused uterine contractions by distending the vagina with a bladder smeared with oil of hyoscyamus, and the first india-rubber bags used in obstetric practice were employed in the same way. The action of a bag placed in the vagina being found to be extremely uncertain, fiddle-shaped bags, like those of Barnes, were devised in order that part of the bag might be introduced through the cervix, and might so exert direct dilating force upon the os uteri. These elastic, fiddle-shaped, hydrostatic dilators may be used for the induction of labour after preliminary dilatation of the cervix sufficient to admit of their introduction.

The conical bag designed by Champetier de Ribes[1] has been gaining favour during the last twelve years. It is conical in shape, the tube through which it is distended being attached at the apex, and it has a pelvic curve. Being made of strong silk covered with india-rubber, it is practically inelastic and, when filled with water, preserves its conical form in spite of considerable pressure. When the os has been dilated enough to admit two fingers the folded bag is passed upwards until the whole of its conical portion lies within the uterus. When almost filled with water it fits into the lower uterine segment, and, acting as a large foreign body, it sets up uterine contractions, as a rule, within three or four hours. The bag then acts as a fluid wedge, much as the "fore-waters" do in normal labour, and transmits to the cervix and lower uterine segment the force of the uterine contractions.[2] If these are weak or absent, the attendant may add to or replace them by pulling gently upon the stalk of the bag from time to time. The base of the cone is approximately of the same diameter as a fœtal head, so that when the os is large enough to allow of the bag slipping through it into the vagina, dilatation may be considered to be complete, and delivery may be rapidly ended if necessary. The main defect of Champetier de Ribes' bag lies in the fact that, owing to its large bulk, it is liable to displace the head, and thus tends to favour the causation of malpresentations and untoward positions of the head. The instrument has probably been used more frequently in cases of hæmorrhage and in dry labours than for inducing labour, the purpose for which it was designed. It is, however, most useful in cases in which induction must be rapidly performed, and when simpler means have failed to produce the desired effect.

Horrocks employs for induction small bags of very thin india-rubber, which closely resemble toy balloons. One of these is tied over the end of a No. 6 catheter, which is then passed about two inches through the cervix so that the bag lies within the uterus. It is then filled with fluid by means of a syringe connected with the catheter by a short rubber tube. The bag acts as a foreign body, and after a time sets up uterine contractions; but this result may be delayed for several days. When once established the pains may continue naturally, or it may be necessary to maintain them by the use of Barnes' or de Ribes' bags. These small rubber balloons afford a very perfect method of gradually and slowly inducing labour, which in time may prove superior to the use of the bougie or repeated manual dilatation. They do not in any way

[1] *Archiv für Gyn.*, Band lix. Heft 2, 1899.

[2] The practice of separating the membranes freely by copious injections of water through the cervix was found to be very dangerous, owing to the risk of producing air embolism, and has accordingly fallen into disuse.

[3] *Archiv Ital. di Gin.*, December 31, 1898.

[1] *Annales de gynécologie et d'obstétrique*, 1888.

[2] It follows that this bag is seen to the greatest advantage when the membranes have been ruptured and the waters lost.

compete with other hydrostatic dilators by which force may be directly applied in opening up the os uteri.[1]

Risks.—The special risks which attend the various methods of inducing premature labour have been mentioned above. Those which are incidental to the methods which are now general, or are likely to become so, are not serious, and they affect the child rather than the mother. They should, however, be remembered when choosing the method to be adopted in any case. The risks which attend the operation whatever method is employed, next demand consideration. The child may, of course, be lost if labour is induced too early or too late; in the latter case the mother also is exposed to the risks of a severe instrumental labour. These accidents can only be obviated by careful calculation and measurements, and by frequent examinations made as the time for induction draws near. A lack of patience in waiting for complete dilatation of the soft parts is to blame for the death of many children, and, though obvious, it must be pointed out that for successful delivery by turning a roomy cervix is even more essential than for forceps delivery.

For the mother, the main risk is that of septic infection; for whatever be the method employed, repeated and prolonged vaginal and intra-uterine interference may prove to be necessary before delivery can be brought about. Though the danger of sepsis is indeed the only one which need be seriously considered when contemplating the induction of labour, it is so grave as to forbid the operation except when clearly indicated, and in circumstances when scrupulous cleanliness can be obtained. All fussy attempts to render the vigina aseptic are to be deprecated, and the use of strong antiseptic solutions should be avoided as tending to lower the resistance of the parts to the invasion of pathogenic organisms. Vaginal douching should be limited strictly lest the natural secretions be too completely removed. A mild antiseptic, such as lysol or creolin, should be employed rather than mercurial salts or carbolic acid. All instruments used should be capable of sterilisation by boiling, and should be boiled on every occasion before use. The personal cleanliness of the operator and attendants should be secured with the same care as in major surgical operations. Above all, it is necessary to avoid bruising, tearing, or crushing of the vulva, vagina, and uterus. "A light hand is the best antiseptic," and for this reason, whenever time permits, mild and gentle measures should be adopted in the first instance, and should be given a full trial before more violent and rapid methods are employed. It is essential that all fæcal accumulations be removed from the bowel before beginning induction, and that the rectum be fully emptied every day thereafter.

*Care of Child.*—In arranging to induce labour, preparations for the reception of the child must not be forgotten. If possible, the services of a wet nurse should be secured for a short time, as the maternal supply of milk is usually somewhat delayed, and often fails altogether. An incubator must also be provided in which the child can be kept for a few weeks, namely, until the natural term of pregnancy has expired. Good "thermostatic nurses" can now be obtained in which a temperature of from 85° to 90° F. can be maintained by means of a lamp or gas jet controlled by an automatic regulator. Failing this, a box with a false bottom may be constructed, the lid being of glass, with holes for ventilation. The child, wrapped in cotton wool, with a thermometer, is placed in the upper chamber, while the lower contains a number of hot-water bottles sufficient to raise the temperature to a proper degree. When the thermometer falls, the bottle which has cooled most is withdrawn and replaced by a fresh hot one. The temperature is gradually lowered week by week. The child should be removed from the box only for washing and feeding. If not strong enough to suck, the infant should be fed with a teaspoon, mother's milk being drawn off for the purpose when possible.

Choice of Time and Method.—The cases in which labour may be induced with propriety fall, as above mentioned, into two classes, namely: (1) those in which the operation is done mainly in the interest of the child; and (2) those in which it is undertaken chiefly for the sake of the mother. It is necessary to consider briefly the conditions under which labour should be induced, and the methods which are most appropriate in cases of these two kinds.

In cases of moderate pelvic contraction seen early in pregnancy, we may be aware from experience at previous confinements, or we may conclude from measurements, that a living child cannot be delivered by forceps or turning, while the pelvis is too large to justify Cæsarean section.

If the true conjugate be estimated at from $2\frac{3}{4}$ to $3\frac{1}{4}$ inches (or $3\frac{1}{2}$ if the transverse diameter is small), induction is the proper treatment; for although some authorities prefer symphysiotomy at term to premature induction under these conditions, the cutting operation does not as yet yield results sufficiently good to justify its choice in cases which are seen early enough to permit of induction.

When it is decided to induce labour prematurely, the time at which this can be done with the greatest advantage must be carefully determined. There is little chance of rearing a child born before the thirtieth week of gestation, and in the interest of the child induction should be delayed as long as possible. It must be

[1] Electricity has often been suggested as a means of inducing labour; but any current which is sufficiently strong to set up uterine contractions is so disagreeable to the patient, that this method is without practical value.

performed, however, soon enough to ensure that the fœtal head is smaller and its bones more plastic than at term, and should therefore be done as a rule not later than the thirty-sixth week. The object is to choose a time at which the child can be delivered with forceps without undue difficulty. It is unnecessary to induce labour so soon as to make natural delivery possible. In determining such a date it is necessary to consider the size and shape of the pelvis as ascertained by pelvimetry (*q.v.*).[1] The amount of room available in the transverse diameter is almost as important as the conjugate, and the outlet of the bony pelvis must also be considered with care More valuable than measurement is knowledge gained by experience at previous confinements, and most useful of all is the history of previous inductions of premature labour. If the conjugate measures 4 inches, but a dead child has previously been delivered with forceps, induction at the thirty-eighth week would be justified. The table below gives approximately the times for induction suitable to certain measurements:—

| | | |
|---|---|---|
| Conjugate | $3\frac{1}{2}$ inches . . | thirty-sixth week. |
| | $3\frac{1}{4}$ inches . . | thirty-fourth week. |
| | 3 inches . . | thirty-second week. |
| | $2\frac{3}{4}$ inches . . | thirtieth week. |

When it is decided to induce in a certain week, it is next necessary to ascertain as nearly as possible the date of commencement of pregnancy. It is not wise to attach too much importance to statements as to the date of the last menstruation, and the date of quickening is even more fallacious. It is always desirable to measure the distance from the pubes to the fundus with callipers, and so estimate the stage to which pregnancy has advanced. The average height of the fundus above the pubes in different weeks of pregnancy is given below in inches:—

| Weeks | 16 | 18 | 20 | 22 | 24 | 26 | 28 | 30 | 32 | 34 | 36 | 38 | 40 |
|---|---|---|---|---|---|---|---|---|---|---|---|---|---|
| Inches | 4 | 4·7 | 5·1 | 6 | 6·6 | 7·3 | 7·8 | 8·3 | 8·7 | 9 | 9·3 | 9·6 | 10 |

Two weeks before the proposed date of induction the physician should make a bimanual examination. With one hand on the fundus and two fingers of the other hand in the vagina, he must press down the fœtal ovoid until the head enters the pelvis, which it will do very easily if the calculation is correct. Subsequently the patient must be examined every week, so that induction may be deferred, if possible, or antedated if necessary. As soon as the head fits the brim accurately the process of induction should be begun.

The choice of method must depend upon the experience and judgment of the operator, and upon the circumstances of each case. In premeditated inductions rapidity is not the main object, and it should be the rule to begin with simple measures and employ no unnecessary instrument. The bougie may, of course, be used, in which case Briggs's method is the best; or the small rubber bag of Horrocks. But in most hands, old-fashioned manual dilatation will give the best results. The cervix should first be opened with a graduated series of dilators (no anæsthetic being necessary, as a rule), until a finger can be passed through the cervix. Thereafter dilatation, first with one and later with two fingers, should be made once or twice daily until the cervix gradually softens and labour follows naturally. There is a certain number of cases in which pains do not follow manual dilatation with sufficient rapidity to ensure delivery while the head is still small enough; but simple measures are enough to cause the onset of labour in so large a proportion of cases, that they ought to be tried in every case before resorting to more violent means. The head should be pushed into the pelvis every day whilst dilatation is being carried out, and when it begins to be difficult to make it enter the brim, it is quite soon enough to begin more rapid dilatation. The best course is then to insert a de Ribes' bag, using, if necessary, one or two sizes of Barnes' bags, in order to gain sufficient room for the admission of the larger instrument. If the de Ribes' bag does not set up pains within six or seven hours, traction may be made upon the stalk of the bag. Delivery should never be attempted when pains are completely absent, or before dilatation is complete. It is never wise to apply the forceps until the whole hand can be passed through the cervix. It is impossible to insist too strongly upon the importance of patience and judgment if induction is to be successful. The needs of each individual case must be considered, not only before beginning, but every day and hour until delivery is over.

Apart from moderate pelvic contraction, there are some other conditions in which premeditated induction may be required for the sake of the child. Such are obstruction of the parturient canal by small fibroids in the lower part of the uterus, cancer of the cervix, cicatricial contractions of the vagina, and the like. In these cases time and method must be chosen, as in cases of moderate pelvic contraction. In certain conditions in which induction is undertaken on behalf of the mother there is no special need for speed, and the gentle methods applicable to induction in the interest of the child may be employed. Thus in phthisis (*q.v.*) and in some forms of cardiac disease (*q.v.*) it is sometimes possible to prolong pregnancy until the child is viable, to induce labour gradually, and deliver gently with the help of an anæsthetic,

[1] Numerous attempts have recently been made to obtain by means of radiography an accurate image of the pelvic brim. The methods used have as yet proved very unreliable, and at the present time have no practical value.

in cases in which natural labour at term could not fail to be attended by serious results.

We have referred to the induction of labour in conditions such as persistent vomiting, albuminuria, and jaundice. Regarding these and other symptoms as manifestations of the so-called pre-eclamptic state (hepatic toxæmia of Pinard), we must look upon the induction of labour where thus indicated as a prophylactic against eclampsia, acute yellow atrophy, mania, melancholia, and other serious forms of auto-intoxication (see "Pregnancy, Affections and Complications"). It is not desirable to lose time after it is judged necessary to empty the uterus in such cases. The cervix should, therefore, be dilated at one sitting, first with solid and then with hydrostatic dilators, until a de Ribes' bag can be introduced, an anæsthetic being used if necessary. In this way it is generally possible to end labour safely in from ten to twelve hours.[1] The same method should be employed when inducing labour for chorea, or for mental symptoms. In all the above cases Briggs's method may be preferred by some to the use of bags. Urgent conditions which directly endanger the life of the mother may demand even more active measures for emptying the uterus; such are eclampsia and uterine hæmorrhage due to accidental separation of the placenta and to placenta prævia. The treatment of these conditions is fully described in the articles under their names, to which the reader is accordingly referred,

Induction of Abortion.—The induction of abortion in the later half of pregnancy, but before the seventh month, differs from the induction of premature labour only inasmuch as it is undertaken solely in conditions which threaten the life of the mother. Since the child cannot be saved, one objection to rupturing the membranes is removed, and this is an easy and certain means of ending pregnancy which is sufficient in many cases. If it is desired to empty the uterus rapidly, dilators may be employed in the usual manner. The fifth month is perhaps the most awkward time at which to induce abortion, as at this time the placenta does not separate easily, and if it is retained it is difficult to remove, the uterus being too small to admit a whole hand, and too large for easy exploration with two fingers. In the first half of pregnancy, abortion should be induced by a method which dilates the cervix without rupturing the membranes. For if the ovum is once broken it tends to come away in pieces through an os but partially opened; the emptying of the uterus is then attended with difficulty, and sepsis is very apt to occur. Tents of laminaria used singly, or several side by side, with rigid antiseptic precautions, serve the purpose best.

[1] The author induced labour in this way in eleven cases, the average time from insertion of the bag to delivery being eleven hours.

When sufficient dilatation has been gained the tents should be withdrawn, the cervix and vagina being then packed with gauze to promote uterine action. On removing the gauze six or eight hours later, the ovum may be found complete in the vagina. If part of the ovum has been expelled, the uterus should be completely emptied at once without allowing time for the os to close. If the whole ovum is still *in utero*, the packing may be resumed and more time may be allowed. As soon, however, as any suspicion of putrescence is indicated by the odour of the discharges, by rapidity of pulse, or high temperature, the uterus should be emptied with the fingers or the curette, after any further dilatation which may be needful has been secured by solid and hydrostatic dilators. Abortion may be induced when there is any obstruction due to abnormality of the hard or soft parts which renders the delivery of a viable child impossible, unless the mother prefers delivery by Cæsarean section at term. For this purpose the operation should be done as early as possible. Any disease justifies abortion at any time if it threatens the mother's life and can be checked by the termination of pregnancy (see "Pregnancy, Pathology of, Affections and Complications"). It is necessary to add that abortion should never be induced without a consultation.

## 2. Forceps

Invention . . . . . 281
Modes of Action . . . . . 281
Construction . . . . . 282
Indications for Use . . . . 284
Mode of Employment . . . . 285
Dangers . . . . . 286

The invention of the harmless or safe midwifery forceps has been traced by Dr. Aveling to Peter Chamberlen, who was brought to Southampton when a lad, probably in his teens, by his Huguenot father, William Chamberlen, when the family fled from Paris in 1569, in time to escape the St. Bartholomew massacres. Peter and a younger brother, before their deaths, respectively in 1631 and 1626, acquired a great reputation for their skill in effecting delivery in difficult cases through their possession of an instrument, the nature of which they kept secret and handed down to the son, grandsons, and great-grandsons of the younger brother. Palfyn, a surgeon in Ghent, proposed the use of a rather inadequate variety of forceps in 1720; but the forceps really came into use towards the end of the seventeenth and beginning of the eighteenth centuries, when the nature of the Chamberlen secret began to leak out. In 1773 Chapman wrote a work on *The Improvement of Midwifery*, in which he described and advocated the employment of a forceps modelled more or less on the Chamberlen pattern, and spoke of the instrument as already well known (see Fig. 1).

Modes of Action.—Forceps may be conceived

of as a pair of iron hands passed within the maternal canals so as to embrace the head of the child for its extraction.

Five different modes of action have been attributed to the instrument.

1. *Traction.*—The great and primary use of the forceps is as a tractor, and in the ordinary run of cases it effects the delivery solely by the power it enables the operator to employ of making simple traction.

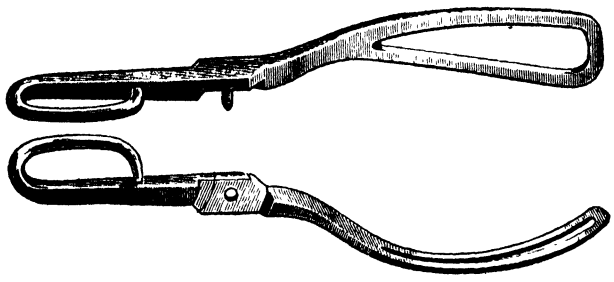


FIG. 1.

2. *Compression.*—The compression influence of the forceps is one that, in most cases, we would willingly dispense with, seeing that diminution of the head in one diameter has to be compensated by increase in another. With most varieties of forceps no traction can be effected without the production of some degree of compression, the pressure on the head amounting to half the force exerted in making traction.

3. *Rotation.*—During the progress of the head under simple traction, the usual forward rotation of the occiput tends to occur, if only the operator is careful not to hinder it. Cases arise where the rotation fails, and can be brought about by a judicious working of the forceps.

4. *Leverage.*—At one time the lever action of the forceps was called into play in cases where the head failed to advance under simple traction. By swaying the handles from blade to blade, the operator could sometimes succeed in dislodging the head that was impacted in the pelvis. This pendulum movement, which is often dangerous, becomes quite unnecessary where an instrument is used that gives full power of traction in the pelvic axis.

5. *Dynamic Action.*—It has been often enough observed in lingering cases, where the parturient powers had become almost inert, that after the head began to be dislodged in the grasp of the forceps the uterine energy revived. This may be due either to the mere presence of the blades within the passages acting as a stimulant, or to the retractile change occurring in the walls of the uterus as its contents are withdrawn, or to the irritation of a new set of nerves as the head moves into a lower plane of the passages. However it may come about, this dynamic action of the instrument must be kept in mind by the operator who has applied it for delivery in a case of uterine inertia.

CONSTRUCTION. — I. *Parts of Forceps.* — The forceps in its simplest form consists of two halves—right and left, in each of which there is a Blade, a Handle, and a Lock or Joint.

1. The *blades* are curved towards each other so as to embrace the head of the child. This curve—called the fœtal, or cranial, or cephalic curve—should have a length from the tip to its proximal end of from 5 to 6 inches, so as to be able to lie along the sides of the fœtal head in its longest diameter, and a width between the most distant part of the curves of the two side blades of $3\frac{1}{2}$ inches, so as to fit the greatest transverse diameter of the average infant head. If the curve be too deep, pressure is exerted during traction only on the part of the head lying towards the tips; if the curve be too shallow, the pressure is confined to a narrow zone in the transverse diameter of the head. There should be a distance of an inch between the tips of the blades, to avert dangerous compression of the neck of the child.

In some forceps the blades are solid: more frequently, and in the best forceps, they are fenestrated. The fenestra save space and allow of a more secure grasp of the head. The blades are left and right, according as they are introduced in relation to the left or the right side of the maternal pelvis.

2. The *handles* are the second essential in the construction of forceps, and serve for the manipulation of the blades. In what may have been the primitive Chamberlen forceps, the proximal extremities of the handles terminated in loops, like the handles of scissors or coal-vase tongs. In the forceps that came first into public use in this country, such as those of Chapman (see Fig. 2) and Giffard, the handles terminated in hooks; and up till a recent period the forceps in common use in France were made entirely of steel, with terminal crotchets through which traction was effected. Smellie had the thin steel strip of the handles covered with wood to give better power of prehension, and such handles were prevalent in the construction of British forceps for more than a century and a half. In Germany the handles were also covered with wood, but had hooks close to the lock for purposes of traction.

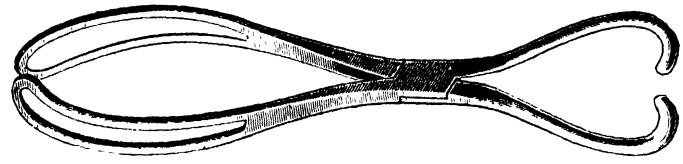


FIG. 2.—Chapman's forceps with handles ending in hooks—the first forceps figured in an English work

3. The two blades are crossed so as to meet at the point where the blade becomes continuous with the handle. This meeting-place forms the *lock* or *joint* of this forceps. In the earliest type the joint is like that of a pair of scissors or coal-vase tongs, except that the pin is riveted only into the left blade, so as to allow of its separate introduction. After the right blade is passed, the orifice at its joint fits over the pin projecting at the joint on the left, and thus the two sides are locked. The pin is sometimes made to work by being screwed into the left side, after the opening or slot on the right has been fitted over it. This form of lock is commonly found variously modified in French forceps. In Germany, the Brunninghäusen lock is preferred; in this a button and stem are found on the left blade, and a notch on the right fits on the stem and forms an excellent joint. In Great Britain, forceps are usually made with the Smellie lock, in which the blades mutually fit into grooves on the opposite handles at their crossing, so as to allow of their easy locking (see Fig. 3).

II. *Curves of Forceps.*—Forceps so constructed are straight from tips of blades to handle-ends

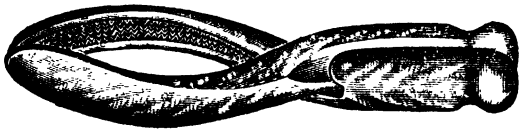


FIG. 3.—Smellie's wooden forceps, showing the "Smellie lock."

when viewed from the side. They have only one curve to allow of the blades adapting themselves to the rounded head of the child.

1. *The Cephalic Curve.*—This is found in every forceps, and enables the practitioner to lay hold of the head of a child lying low in the parturient canal for its extraction. For cases where the head is arrested farther up in the passages, attempts were made to get access to it by lengthening the distance between blades and handles. But the forceps were still straight, and however long the shanks or stems might be, it was impossible to get a satisfactory grasp of the body that had to travel through a curved canal. The head was apt to be seized only by the part lying close to the back wall of the pelvis, and when traction was attempted the forceps slipped. If the practitioner tried to carry the points of the blades forward towards the symphysis to get a fuller grasp of the head, then the shanks or the lock of the instrument were found to be crushing the perineum. It became evident that to grasp and guide the head through the curved canal the forceps must themselves be curved.

2. *The Pelvic Curve.*—Pugh and Smellie in this country both independently recognised the importance of having the blades curved, with their tips turning forwards for adaptation to the pelvis, and nearly simultaneously proposed the employment, at least in special cases, of forceps having a pelvic curve. But it was Levret, in Paris, who first published a memoir describing forceps with a "new curve," which made it possible to seize the head at the pelvic brim or at any lower level, and to extract it with the greatest safety to the mother in the proper line of descent through the maternal passages. There may have been others besides who improved forceps in this direction, but it was the clear demonstration of the value of the pelvic curve made by Levret that led to the general introduction of the double-curved instrument.

Forceps by Pugh, Wallace Johnston, Mulder, and others were contrived with what was called a "perineal curve." In these the shanks were slightly recurved just above the lock, with the view chiefly of avoiding the danger of pressure on the perineum. But they were not widely used; and even after the value of the pelvic curve had been demonstrated, practitioners continued to make use mainly of straight forceps, which were most easily and often secretly applied, and were usually effective in the extraction of the head lodged on the pelvic floor. Some obstetricians, even after they had recognised the necessity of using the double-curved instrument for what were regarded as "high" or "long" forceps cases, would still use a short straight pair for their "low" or "short" forceps cases. After anæsthesia came in to give the operator more freedom in the application of forceps, the smaller straight instrument, that was only applicable to a head at the outlet, gradually gave place to the longer curved instrument that could grasp the head at any level in or above the pelvis, and that could pull the head through the outlet with less danger to the perineum. The more it was used, however, the more clear it became that an instrument with a pelvic curve had two drawbacks when traction was made through straight handles. In the first place, some of the expended traction power was lost, because the pull was not exerted directly in the line of the chord of the blade-curve, which represents the line of direction for the descent of the head; and in the second place, some of the force was misdirected, tending to press the head against the symphysis pubis, and thus favouring a slipping of the blades off the infant's head (see Fig. 4).

*The Compensation Curve.*—In 1860 Professor Hubert, of Louvain, who very clearly demonstrated the disadvantages of the double-curved forceps, proposed to remedy their defect by bending back the free extremities of the handles at a right angle in order to correct the line of traction. In 1868 Dr. Aveling, of London, had forceps constructed with what he called a "handle curve," in which the rounded compensation curve of the handles gave the instrument, on side view, a sigmoid aspect. Such an instrument, although somewhat more difficult

of application than the straight-handled forceps, was better adapted for allowing the normal descent of the head when traction was made at the ends of the recurved handles. But they

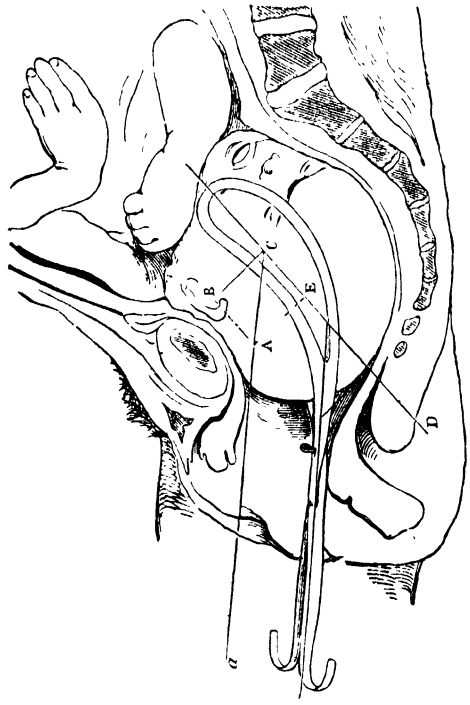



FIG. 4.

never came into general use, and their value was superseded when, in 1877, Professor Tarnier of Paris showed that the correct traction could be most certainly attained through curved rods, jointed to the heels of the blades and pulled on by a traction bar attached to their free extremities (see Fig. 5). Tarnier demonstrated that when traction was made through jointed rods with the necessary compensation curve,

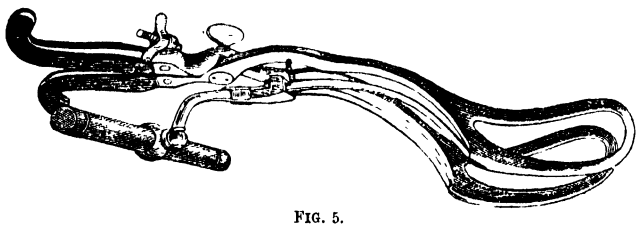


FIG. 5.

there was not only no loss of force and no misdirection of force, but further, that as the head changed its direction during its transit through the curved canal, this change in its direction would be indicated by a forward movement of the application handles. For these application handles, keeping the head fixed in the grasp of the blades by means of a screw placed close below the lock, were free to change their direction with the changing movements of the head, and so gave a guide to the direction in which at any moment traction should be made. Forceps constructed on this principle are found to be possessed of other advantages. The operator expends no energy

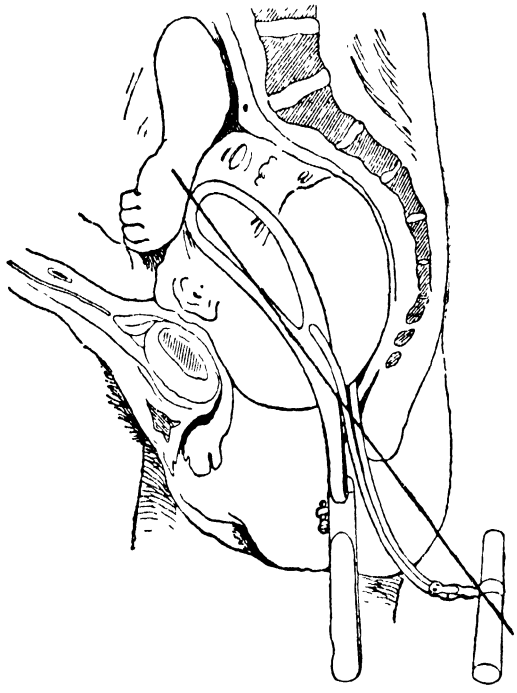


FIG. 6.

in squeezing together the forceps handles; and as he pulls simply on the traction bar, his muscular sensibility remains unimpaired, and he is better able to judge of the amount of force he is expending. There is no undue compression of the head in one direction, with tendency to elongation in another, so that the changes in configuration of the head more nearly resemble those seen in a head expelled by natural efforts. As the head is drawn down in the axis of the canal there is no unnecessary injurious pressure on the maternal structures, and as the head is not dragged against the symphysis pubis it is not so liable to elude the grasp of the blades. Rotation of the head in occipito-posterior cases will usually occur spontaneously under steady traction with the traction bar; where rotation fails, it can be aided by grasping the application handles (see Fig. 6).

INDICATIONS FOR USE.—Indications for the use of the forceps arise either in delayed labours or in labours more immediately dangerous.

A. DELAYED LABOURS.—There is a fault in one or more of the factors of labour; and when two or even all the three factors are seen to be faulty, it is always right to seek to determine in which of them the fault originated and is most pronounced—whether (1) in the Powers, (2) in the Passages, or (3) in the Passenger.

1. *Fault in the parturient Powers, primary or secondary, or both.*

(1) *In the Uterine Power.*—The uterine muscular fibres may be in a condition of atony or of spasmodic irregular contraction, or their force may be misdirected from some obliquity of the organ.

(2) *In the Accessory Powers.*—There are cases of weak muscularity in the abdominal muscles, or paralysis, or exhaustion, that may render the patient unable to further the involuntary action of the uterus by her straining efforts. More frequently some thoracic mischief renders such efforts undesirable, and the practitioner has to supply the lack of parturient power by traction on the head of the child with forceps.

2. *Fault in the parturient Passages.*

(1) *In the Soft Canals.*—The forceps is not a good means of hastening delivery in cases of rigidity of the *cervix* uteri, though it has been thus employed. When the head is pulled through the imperfectly dilated os uteri the lips are very apt to be lacerated. But the instrument is often serviceable when the *vaginal walls* are narrowed from any cause, and again in many cases where there is delay from the rigidity of the *perineum*, or narrowness of the pudendal aperture.

(2) *In the Hard Canals.*—Pelvic contractions form a large contingent of the cases where the practitioner effects delivery with forceps. The range of contraction in the brim conjugate where the forceps is available is from 4 inches to 4¼ inches, or, where the head is not too large, to 3 inches. If the pelvis is of the justo-minor type, the dip of the hind-head, indicative of a marked degree of head-flexion, and the position of the head with its long diameter in the right oblique of the pelvis, give opportunity for the perfectly safe application of the blades along the two sides of the head. Where the pelvis is flattened and the head is placed in the transverse diameter of the brim, care has to be taken lest the blades be applied in the line of the occipito-frontal diameter and a dangerous degree of depression be produced. In cases of coccygeal anchylosis, of funnel-shaped pelvis, or where the space is encroached on by tumours to a moderate degree, forceps can frequently be used to bring the head past the contracted plane.

3. *Faults in the Passenger.*

(1) *Large Size of Head.*—If we meet with a patient who has had one or two labours that were strictly natural, requiring the use of the forceps in a subsequent confinement, the cause of the difficulty is likely to be found in the greater bulk of the body passing, and notably in the increased size of the fœtal head. The increase in bulk may be simply the larger size of the male over the female head, or of an unusually large child of either sex, as is seen in some cases of protracted gestation. Or the head may present such a degree of ossification of the bones as to interfere with the moulding which allows of the expulsion even of a tight-fitting head under natural efforts. In these cases the head comes down with the occiput and triangular fontanelle markedly depressed—the large head finding its way through a normal pelvis, as the normal head makes its way through an undersized pelvis, with an exaggerated degree of flexion. Such enlargements of the head form a very fair group of cases for extraction with the forceps; but the instrument becomes dangerous in its application to cases of morbid enlargement, as in hydrocephalus. It is not easy to define the degree of enlargement at which danger begins; but in any case where the operator has satisfied himself that he has not taken an antero-posterior grasp of the head, and yet finds that the handles are not easily approximated, he may be sure that to persevere with the use of the instrument will be dangerous both to child and mother.

(2) *Malposition of the Head.*—Where the head in one of the posterior positions fails to make progress, forceps will often serve to bring about its descent and rotation; and where the occiput has rotated backwards it often becomes necessary to extract the head artificially.

(3) *Malpresentation of the Head.*—When we meet with a brow or face presentation at the commencement of labour, and have reason to distrust the natural powers, we may give mother and child the best chance by turning as soon as the cervix is fully dilated, or as soon as possible after the membranes have broken. But where the head so presenting has come into or through the brim and is delayed in the pelvis, we must have recourse to forceps. Care has to be taken in these face cases to apply the blades to the sides of the child's head, otherwise the points of the instrument may easily do serious damage to the structures in the infant's neck.

B. Dangerous Labours.—Special danger may arise calling for immediate delivery, and the forceps gives us the control of the labour.

1. *Maternal Complications.*—In many cases of labour complicated with utero-placental hæmorrhage, with rupture of the uterus, with convulsions, chest disease, syncope, etc., the artificial delivery that is urgently indicated can be most safely effected, both for mother and child, with forceps.

2. *Fœtal Complications.*—Again the instrument is invaluable in cases of irreducible prolapsus funis, when the waters have escaped for some time and the head is entering the pelvis, as well as in some cases of malformation of the infant.

Mode of Employment.—1. *Preliminary Measures.*—A practitioner having made up his mind that forceps delivery is necessary in the interest of the mother or child or both, (1) should intimate to the responsible attendants his intention to employ the instrument; and

whilst he is warranted in explaining the safety of the operation as regards the mother, he (2) should be cautious in his prognosis as to the condition of the infant. (3) His instrument should be sterilised, warmed, and greased on the outer surface of the blades with an antiseptic ointment. (4) He should ascertain that the bladder and bowel of the patient are emptied, and with the patient anæsthetised, (5) he should have her placed across the bed, on her left side, with the knees bent up and the nates brought to the edge of the bed. The delivery may be completed with the patient in this position, though sometimes he will find it easier to extract the head if the patient be placed supine. (6) Before proceeding with the application of the blades, he should make a further vaginal examination with the four fingers of the right hand, so as to assure himself of the precise position and relations of the head, and feel where there is most space for the entrance of the instrument.

2. *Introduction of the Forceps.*—(1) The left blade should be introduced first, and the practitioner who would save himself the uncertainty that sometimes comes on a man at this moment as to which is left and which is right, ought to provide himself with a pair of forceps with "Left—First" stamped on the left blade. (2) The fingers of the right hand, which have been examining the relation of the head to the passages, are used to guide the tip of the left blade, which is held in the left hand and applied to the left side of the pelvis. (3) The introduction should be begun immediately after the cessation of a pain. The practitioner may succeed in applying both blades before the recurrence of another contraction. If a pain comes on, he must suspend the attempt to push on the blade during the continuance of the pain. (4) The instrument is to be insinuated gently, and if it meets with resistance it must be withdrawn and its direction altered. (5) The tip of the blade is to be kept in contact with the fœtal head rather than with the vaginal wall. (6) Each blade is to be entered and applied in the proper axis of the pelvis. (7) The concavity of the blade is to be adapted to the convexity of the fœtal head. (8) When the first blade is fully introduced it is to be kept *in situ* with the thumb and last two fingers of the left hand, while the index and medius of the same hand are applied to the head to guide the right blade into the passages. (9) The right blade, with the traction-rod swung forward, is held in the right hand and introduced at right angles to the left, pointed at first towards the hollow of the sacrum, and carried round the head until it comes into complete antagonism with its fellow. (10) The handle of each blade is grasped by its homonymous hand to effect the locking, the right rod is swung back, and the fixation screw adapted but not tightened.

3. *Extraction of the Head.*—(1) The application handles are grasped with one hand, and the screw is fixed with the other at the point where safe and sufficient compression of the head is secured. (2) Traction is made with the traction handle during pains; or, if no pains are present, at intervals. (3) In making traction the traction rods are kept constantly parallel with the shanks, thus ensuring progress of the head in the proper axis of the canals. (4) After each tractile effort the screw is slackened, but not unshipped, and during the interval the operator ascertains what progress the head has made. (5) In occipito-posterior cases, when the forward rotation of the occiput fails to occur under the simple traction, the rectification is aided by grasping the forceps by the application handles. (6) At the outlet, while traction is made with the right hand, the perineum may be supported with the left. (7) The head is made to distend and pass through the pudendal orifice very slowly, the uterus being allowed to complete as often as possible the expulsion of the head and always of the body of the infant. (8) Immediately after the birth of the head the screw must be slackened, the right traction rod freed from the locking plate, and the right and left blades removed successively.

Dangers of Forceps.—Although the forceps is safe in principle, both to mother and child, there are dangers to both attendant on its use which are reduced to a minimum with a properly constructed instrument properly applied and worked. The practitioner who has these risks clearly in view is most likely to avoid them.

I. *To the Mother.*—The vaginal walls or roof, or the cervix uteri, may be torn with the tip of the blades during their introduction. The vaginal walls may be bruised at the sides during rotation of the occipito-posteriorly placed head. The nerves of the pelvis have sometimes been damaged, and the ligaments strained at the various articulations. The most frequent mischief is produced when the head is extracted too rapidly or in a wrong direction through the pudendal aperture, when the perineum is more or less deeply lacerated, or the labia pudendi are bruised and torn. But greater than all the risks attendant on the use of forceps is the risk incurred by delay in their application in cases where the head is lodged for hours in the pelvic cavity.

II. *To the Child.*—The greatest danger to the infant lies in such a grasp of the head as involves undue compression, when bruising of the soft parts and even fractures of the skull may be produced. Sometimes the neck of the child is squeezed between the tips of the blades, and where the cord is coiled round the neck one of the blades may be found to have produced a fatal compression of it. Not infrequently the face of the child is seen to be

the subject of a Bell's paralysis resulting from compression of the facial nerve, and usually passing off within ten days or a fortnight.

SPONDYLOTOMY.—See "Embryotomy."

### 3. Vectis

When the midwifery forceps was coming into general use in the end of the seventeenth and beginning of the eighteenth centuries, Roonhuysen, Ruyschl, and the other chiefs of the Amsterdam Academy, were known to be making use of an implement for promoting labour, the nature of which they kept secret. They were enabled to obtain an enactment that prohibited anyone from practising midwifery in Holland who had not purchased their secret, which was a source of revenue to them until it was made public, in 1753, by Visscher van der Poll. The instrument they employed, with various modifications, speedily came into general use under the designation of the vectis or lever, and for more than a century competed with the forceps as a means of promoting the advance of the head in cases of tedious and difficult labour. It was used sometimes as a lever of the first order with the mother's pelvis, or, better, with the operator's left hand as a fulcrum; sometimes as an extractor worked as a lever of the third order. It is in the latter fashion that its employment has recently been advocated by Dr. Bartlett of Chicago, the instrument being made with a handle flattened and so elongated as to rest on the forearm as a fulcrum near the elbow. Sometimes it has been used as a single blade of forceps, one of the operator's hands taking the place of the second blade. It has been claimed for it that it is easier of application than forceps and less likely to do harm in extracting, whilst it is often efficacious in aiding rotation in occipito-posterior cases. It has been applied chiefly over the occiput, but may be adapted to the sides of the head or to the brow or face. In some cases its place of application is moved from one part of the head to another. It is, of course, only available when the uterus is in action; and because of its tendency to slip, and the ease and safety with which in most cases delivery can be effected with forceps, the vectis has fallen almost completely into desuetude. The "levier-prehenseur-mensurateur" proposed by Faraboeuf is in reality a variety of forceps which owes what efficacy it may possess to its second blade.

### 4. Version


VARIETIES . . . . . 287
INDICATIONS . . . . . 287
METHODS—
*External* . . . . 287
*Bipolar* . . . . . 288
*Internal* . . . . 289
PRACTICAL RULES . . . . . 290


The operation of version, or turning, consists in the artificial substitution of one presenting part by another. This comprises either the substitution of one pole of the fœtus by another, or the conversion of oblique presentations into the presentation of one or other pole so that the long axis of the fœtus and uterine cavity correspond.

VARIETIES.—From the definition of the operation it must be apparent that there are two varieties: (1) Cephalic version, in which the head of the fœtus is made to present; (2) Podalic version, in which the breech or feet are made to present.

GENERAL INDICATIONS.—The operation is indicated:—

1. In all shoulder and oblique presentations.
2. In cases of placenta prævia, where a large area of placenta presents.
3. In prolapsus funis during the first stage, when reposition of the cord cannot be accomplished.
4. In mento-posterior face and brow cases, when rotation backwards of the chin is feared.
5. When rapid delivery is required, and can be most expeditiously performed by changing the presentation.
6. It used to be extensively employed before the introduction of axis traction forceps, and is still recommended by some, in flat pelves with a conjugate vera over three inches.

METHODS.—The operation may be performed by three methods, known respectively as the external, bipolar, and internal.

*External Method.*—To perform this operation the patient is placed in the dorsal position with

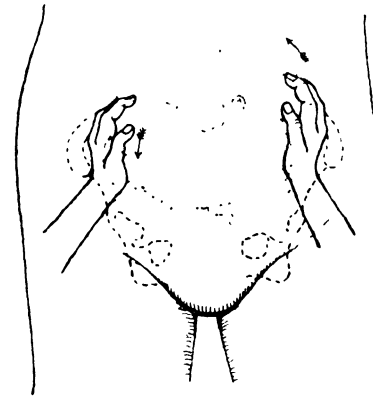


FIG. 1.—External version (Schaffe).

the shoulders raised and knees flexed. The hands of the operator are placed externally over the bare abdomen, one on each pole of the fœtal ovoid. By a series of moderate impulses with the respective hands in opposite directions during the intervals of uterine contractions, the fœtus may be made to revolve on its transverse axis till one or other pole is brought to present. The presentation having been altered, is is well

to maintain it in its newly-acquired situation by means of a binder. Unfortunately, this method of version, which is practically without risk, can only be performed under favourable conditions which are rarely met with when required. Not only must the liquor amnii be wholly present and in considerable quantity, but it is rarely possible after labour has commenced and the presenting part thus fixed in the pelvis.

Should shoulder or pelvic presentations be noticed before labour has commenced, an attempt at their conversion into cephalic presentations should unquestionably be made by this method; but after the onset of labour the fixation of the presenting part prevents its ready substitution in this manner, and the direct employment of the bipolar method should be undertaken.

*Bipolar Method.*—This method of version, as introduced by Braxton Hicks, was undoubtedly

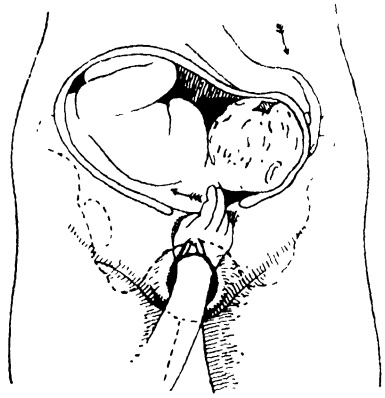


FIG. 2.—Bipolar cephalic version (Schaffer modified.)

one of the greatest advances in obstetric science during the last century. It enables the accoucheur to perform version early in labour when, as in shoulder presentations, it is easy and safe; but perhaps still more important is this method in the early stages of placenta prævia, before great separation of the placenta and consequent hæmorrhage has occurred.

It is carried out by both hands, one externally and the other internally, acting simultaneously on different poles of the fœtus, and has therefore been named by some authors the "Combined method." For its performance it is necessary that there be considerable mobility of the fœtus *in utero*; practically, in the majority of instances, intact membranes and consequent entire liquor amnii.

Before operating, the patient should be anæsthetised, as this is beneficial in controlling the force of the uterine contractions and permitting the painless introduction of the whole hand into the vagina.

The patient may be placed in the dorsal or left lateral position. One hand, preferably the left, having been carefully disinfected and lubricated on the extensor aspect, is in a cone-shaped manner slowly introduced into the vagina by a rotatory movement—any pressure necessary being directed backwards on the distensible perineum. On the cervix being reached two or three fingers are introduced to act on the presenting part as required, while the other hand is placed externally over the abdomen ready for coincident manipulation. The two hands can then move the extremities of the child in different directions. These manipula-

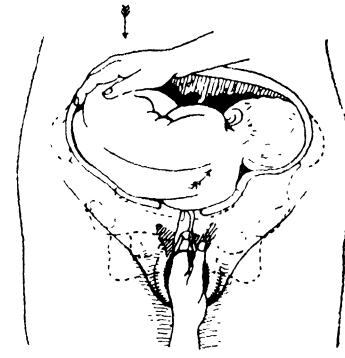


FIG. 3.—Bipolar podalic version, 1st stage. (After Schaffer.)

tions are to be conducted only during the intervals of pains. Thus, in the conversion of a vertex presentation into a breech the internal hand will be made to push the presenting part upwards to free it of the pelvic brim, and then in the direction of the occiput, while at the same time the external hand will depress the breech in the direction of the feet. Rotation of the fœtus is thus made to occur through the smallest arc. In like manner, in shoulder

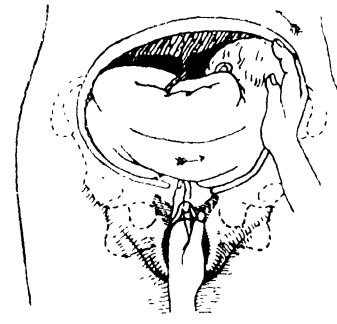


FIG. 4.—Bipolar podalic version, 2nd stage. (Schaffer modified.)

cases the presenting shoulder may be pushed in either direction, according to whether a cephalic or podalic version is desired, the external hand acting on the breech or head as is considered more efficacious.

The advantage of the "combined" over the "external" method chiefly rests with the readiness by which the fixed presenting part can be freed from the pelvic brim after labour has commenced. As a mode of delivery, it is invaluable in placenta prævia when the cervix is but partially dilated. Here podalic version

can be readily performed, and a foot dragged through the cervix to act as a plug against further hæmorrhage. It is also of incalculable value in the easy or early rectification of shoulder presentations. Unfortunately, it cannot be performed after the liquor amnii has drained away, and now the only method that is applicable is the—

*Internal Method.*—From a teaching point of view the term "internal," in contradistinction to the "combined" method of turning, is unfortunate, as it conveys the idea that manipulation is wholly intra-uterine, when in reality the external hand placed over the exterior plays as important a rôle as in both of the preceding methods described.

The operation essentially consists in the introduction of the entire hand into the uterine cavity, by which means one or other pole of the fœtus is grasped and made to present. From the difficulty in seizing the fœtal head internal version is usually podalic, although the cephalic is now more frequently attempted than formerly. This method of operating is of great antiquity, and, until the recent perfecting of forceps, was by far the most common mode of artificial delivery. It is the only method of version applicable after the liquor amnii has drained away.

*Method of Operating.*—The patient should be anæsthetised, so that uterine relaxation may be as complete as possible, and at the same time the absence of any movement on the part of the patient facilitates the ready accomplishment of the operation and minimises risk. Having placed the patient in the dorsal or left lateral position, according to the operator's fancy, he then, after having thoroughly disinfected his hands, proceeds to introduce one slowly into the uterus.

The choice of hand has been, and is, a much controverted subject, and must be left very much to the judgment of each individual operator without laying down any binding rule; but generally it may be said that in dorso-anterior positions the left hand, and in dorso-posterior positions the right hand, seem to be more readily applicable than *vice versâ*. The introduction of the hand into the vagina is to be done by a rotatory movement, the fingers previously having been arranged to form a cone. All attempts at introduction or manipulation whatsoever must be confined to the intervals of pains; during uterine contractions the hand must remain quiescent, and be flattened out as much as possible.

Having reached the os externum, if it be large enough to admit the passage of the hand, the movement upwards is continued in a rotatory manner until the uterine cavity is entered. Should the membranes be unruptured the fingers must be bored through them at the most dependent part, and the hand at once pushed into the uterus to block the entire escape of the liquor amnii. The hand should now be more or less flattened out and carried along the ventral aspect of the fœtus until the part desired to catch is reached. During the introduction of the internal hand the other hand must be placed over the uterus externally to form a counter-pressure to any forcible internal manipulation, so that overstretching of the vaginal attachments of the uterus may be prevented, and at the same time assist in bringing the fœtal parts nearer the inside hand.

In podalic version a much debated point exists as to which foot or knee is to be grasped. In dorso-anterior cases it is almost universally agreed that either which is first met with should be seized, but in dorso-posterior positions there is much difference of opinion among authorities. In the majority of instances it is found that the part which is grasped will rotate anteriorly when pulled on, and thus, by grasping the upper or farther knee, the entire fœtus will be made to rotate on its long axis, which may assist greatly in the dislodgment of the presenting part should it be already fixed (Figs. 5 and 6). Having seized the part desired, slow, gentle, and steady traction must now be made while the opposite pole is pressed in the opposite direction by the external hand. During pains, all manipulations are to be avoided. After version has been completed and the foot dragged into the vagina, the expulsion should be left to nature and treated as an ordinary breech case, unless special indications are present for immediate delivery.

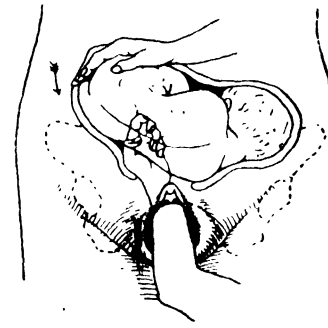


FIG. 5.—Internal version, 1st stage. (After Schaffer.)

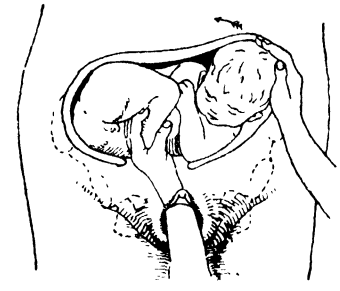


FIG. 6.—Internal version, 2nd stage. (After Schaffer.)

With the membranes immediately previously intact, or but recently ruptured, version is usually an extremely simple operation; but in cases where the liquor amnii has been for long drained away and the uterus firmly retracted on the child, the difficulties and risks are very great and sometimes insuperable. Here the lower lateral segment will be found extremely

thinned, and any forcible manipulation must result in rupture. It need hardly be stated that in these cases the greatest care and gentleness must be exercised in the manœuvre. When the shoulder is impacted in the pelvis turning is much expedited by the application of a noose of tape round the ankle, by which traction may be made, while at the same time the shoulder should be gently pushed in an upward direction (Fig. 7).

Should, from thinning of the uterus, rupture

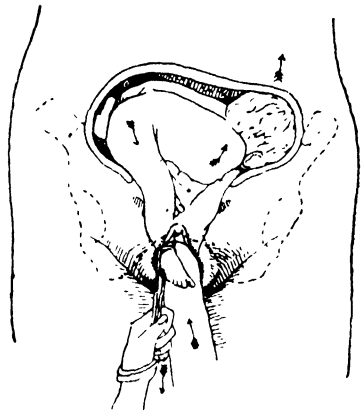


FIG. 7.—Internal version in impacted case assisted by loop. (After Schaffer.)

be feared, decapitation of the child is to be preferred. This, fortunately, however, is rarely necessary.

The dangers of version are mainly sepsis and rupture of the uterus, both of which are avoidable; the first by careful attention to antisepsis and asepsis, and the second by the recognition of existing conditions, which must lead every intelligent practitioner, from his knowledge of general principles, to exercise the necessary amount of caution which is required in dealing with a mechanical problem where the conditions must vary in each individual case.

### PRACTICAL RULES FOR TURNING

1. If labour not yet begun, try external method.
2. If labour commenced, membranes unruptured, and os uteri slightly dilated, try external method.
3. If membranes unruptured and os uteri sufficiently open to admit three fingers, try either external or bipolar.
4. Bipolar method specially indicated in placenta prævia, with os uteri partially open.
5. If membranes unruptured and os dilated sufficiently to let hand through, try bipolar method first in all cases, if no hurry; if hurry necessary, perform internal version at once.

If membranes ruptured, internal method is alone available.

## 5. Embryotomy

SYNONYMS . . . . . 290
IN HEAD PRESENTATIONS—
INDICATIONS . . . . . 290
STAGES OF OPERATION . . . 291
*Perforation* . . . . . 291
*Comminution* . . . . . 292
*Extraction* . . . . . 293
IN TRUNK PRESENTATIONS
INDICATIONS . . . . . 294
PROCEDURES . . . . . 294
*Evisceration* . . . . . 294
*Decapitation* . . . . . 294
*Spondylotomy* . . . . . 294

*Embryotomy* (Gr. Ἐμβρυοτομια) and *embryulcia* (Gr. Ἐμβρυουλκια) are terms alternatively applied from ancient times to delivery brought about through mutilation of the infant. The former term is expressive of the procedures that imply the cutting to pieces (Gr. τήμνο) of the embryo or fœtus, whilst embryulcia expresses the dragging out (Gr. ἕλκω) of the mangled remains. Cases of embryotomy are sometimes described as cases of craniotomy, cephalotripsy, etc., according to the method adopted for securing the delivery. For ages, physicians and surgeons who were called in to see women in difficult labours knew no other way to save the mother but to sacrifice the life of the child, hacking it to pieces, fracturing its bones, and extracting it with hooks or pincers. Version, the Forceps, Induction of premature labour, Symphysiotomy, and the Cæsarean sections, have limited the range of application of embryotomy, so that some obstetricians have expressed the opinion that improvements in these other operations should enable the practitioner to dispense altogether with a process that necessitates the death of the infant. We have not yet, however, attained to this desirable ideal, and the practitioner still meets with cases both of head and of trunk presentation where he finds himself warranted and even compelled to have recourse to embryotomy.

### A. IN HEAD PRESENTATIONS

INDICATIONS.—The conditions that justify or require the sacrifice of the life of the child are usually to be found in such disproportion between the passages and the passenger that the latter cannot be passed through unless it be diminished in bulk.

I. *Fault in the Passages.*—There are different forms of contraction of the *Bony Pelvis*, such as the rickety, where the contraction is mainly at the brim; the kyphotic, where the outlet is narrowed; the osteomalacic, where the whole canal is narrowed. In any of these the practitioner may find himself shut out from delivery by the alternative operations, and obliged to have recourse to perforation of the

head. The range of brim contraction in the conjugate diameter would be between 3¼ and 2 inches. If the contraction is in the transverse diameter there would be corresponding degrees at the outlet. Sometimes the obstacle is due to a morbid condition of the *Soft Canals*, as when there is cancer of the cervix or cicatricial contraction of the vagina, or the space is encroached on by a uterine or ovarian tumour. In a maternity hospital such cases would preferably be treated by some of the Cæsarean methods; but the general practitioner may give his patient the best chance of recovery by lessening the size of the child's head.

II. *Fault in the Passenger.*—Undue size of the fœtal head, or a brow or face presentation in a slightly narrowed pelvis, or locked twins, may necessitate the operation.

III. *Complex Labours.*—In some cases of convulsions or hæmorrhage or rupture of uterus, where the os is still imperfectly dilated, the mother may have a chance of survival if the fœtus can be diminished in size and extracted with less distension of her parts. Where the infant is already dead, as in cases of neglected prolapsus funis in a pelvis measuring something under 4 inches in the brim conjugate, the dangers to the mother will be lessened by reducing the bulk of the head.

As a rule, head-perforation is unwarranted in a woman who has previously given birth to a living child by the natural efforts, or who has been delivered by the simpler operations of turning or the forceps. But malacosteon may cause an increasing difficulty, or tumours may develop; or the head of the later child may be of larger size or present less favourably; or a complication may arise requiring the graver interference.

Stages of the Operation.—There are three distinct stages or steps in the procedures involved in the complete artificial delivery of a child that has the head reduced in size.

I. *Perforation.*—The operator must first make an opening in the presenting part of the cranial vault.

(1) *Instruments to be used.*—1st: In a case of hydrocephalus with wide sutures it will usually be best to puncture simply with a fine aspiratory trocar. 2nd: In the more frequent conditions the bones may be pierced with a Knife, Bistoury, or Spike, of which many varieties have been contrived, sometimes curved, sometimes straight, sometimes sheathed. 3rd: More effectively the opening may be made with a pair of long, strong Scissors, provided with shoulders, which prevent them from sinking too far through the skull. When the handles are separated the points are pulled apart to enlarge the opening. Of this variety Smellie's scissors (see Fig. 1) may serve as a type. 4th: Better still are the Perforators that have the cutting edge of the blades turned outwards, and the blades not crossing at the joint, but hinged, so that when the handles are compressed the points of the blades are separated. Sir J. Y. Simpson's perforator (see Fig. 2) is the most widely employed instrument of this construction in this country, and Blot's in France.

Fig. 1.—Smellie's scissors for perforating the skull.

5th: In Germany wide use has been made of Trephines, which are alleged to have the advantage of producing an opening with clean-cut edges, and so of diminishing the dangers that arise from the presence of protruding fragments and loose spiculæ of bone. 6th: The Basilyst possesses the advantages of both. If it

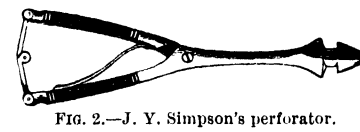


Fig. 2.—J. Y. Simpson's perforator.

be screwed into the vault as far as the shoulder and then withdrawn, a round aperture is left with smooth edges, through which the cranial contents may escape as after the use of a trephine. But in most instances a wider aperture is demanded, and this is produced with the basilyst when the handles are compressed and the points of the blades forced apart.

(2) *Preliminary Measures.*—Before proceeding to operate, the practitioner has some points to consider:—1st: He will have the patient anæsthetised. 2nd: He will have the patient placed in the left lateral position with her knees bent up and her hips at the edge of the bed; it is sometimes more convenient to have the patient in the supine posture when she is asleep with chloroform. 3rd: Bowels and bladder must be empty. 4th: Provision should be made for receiving and covering up fragments of bone and the cranial contents. 5th: The operator must make one more careful examination to make out clearly the promontory of the sacrum, the margins of the os uteri, and the exact position of the head. 6th: An assistant keeps the head fixed at the brim by pressure from above through the abdominal walls.

(3) *Rules for Perforating.*—1st: The Perforator, held in the right hand, is guided to the head by the fingers of the left, which are touching the part selected for perforation. 2nd: The point of the instrument is directed against a bone. In a flat pelvis this will usually be a parietal bone; in the justo-minor it may be the occipital; in brow and face presentations the frontal bones will be struck. It is important to pierce a bone and not merely to go through a suture or fontanelle, else the edges of the bones may come together after the instrument is withdrawn, and little collapse of the skull be produced. 3rd: The perforator should be directed against the bone at a right angle to avoid the

risk of its slipping on the surface. This rule is more needful to be observed with the older perforators than with the basilyst, the point of which, with its double-threaded screw, lays hold at once of the scalp and cannot slide off. 4th: Screw in the instrument up to the shoulder and open it widely and crucially. To obtain a wide gap in the vault it may be necessary to turn it in different directions and expand it. 5th: Advance the instrument into the brain and tear and break down as thoroughly as possible all the cerebrum, cerebellum, tentoria, and the ganglia at the base, otherwise the child may be extracted a living, breathing horror. When quite dead, a softening of its textures immediately ensues, which facilitates its delivery. 6th: Wash out the cranial cavity and the maternal canals with an antiseptic lotion. 7th: Remove with the fingers or a pair of pincers any loose fragments of bone from the edges of the opening in the skull. In some cases the head may be found so diminished in bulk as to be capable of expulsion by the parturient powers, aided, perhaps, by traction from below with the finger of the practitioner passed through the opening, or by the pressure of the assistant's hands above. Usually, however, it becomes necessary to have recourse to further reduction of the size of the head.

II. *Head Comminution.*—Many methods have been adopted for breaking down the bones of the head, and many instruments have been contrived for the purpose. They may all be grouped in five categories.

(1) *Craniotomy.*—The earliest method consisted in laying hold of the plates of bone round the edges of the gap made in the vault and breaking off and removing the fragments morsel by morsel. The archaic writers on obstetrics describe pincers used for the purpose; and the Lyon's forceps and countless other modern varieties are only modifications of the ancient

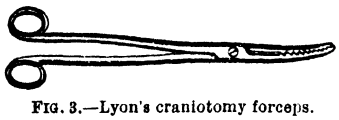


FIG. 3.—Lyon's craniotomy forceps.

implements. At the best an operator can only succeed with them in picking away the bones of the roof and sides of the skull. After his most tedious efforts the bones at the base remain unbroken (see Fig. 3).

(2) *Cephalotripsy.*—It was an important advance that was made by Auguste Baudelocque when he contrived the cephalotribe. This is in essence a pair of powerful forceps provided with a screwing apparatus at the handles, by means of which the blades that embrace the head are brought together so closely as to crush and compress the whole skull. Variously modified, it was for many years the favourite implement all over the Continent for the head-reduction of the infant. But its use was attended with some serious drawbacks. The introduction of the thick blades into the cavity of the uterus carried with it the risk of the entrance of septic germs into the bruised maternal tissues. Whilst it effectually crushed the bones in flattening the head in one diameter, it lengthened it in another. It proved so unsatisfactory as an extractor that some obstetricians used it only to crush the head by repeated applications if need were, and left the expulsion to the natural efforts, or had recourse to turning.

(3) *Cephalotomy.*—The proposals to displace the cephalotribe by instruments that would cut the head in slices did not approve themselves to the general profession. The Labitom of von Ritgen was never used outside Giessen. The Saw-forceps of van Huevel made with a single chain-saw had for a time some vogue in Belgium, and stimulated Tarnier to construct and use one with a double chain that could cut the head into three sections; but they have both fallen into desuetude. The Wire-écraseur proposed for use by Robert Barnes is not easily applied.

(4) *Cranioclasm.*—In 1858 Sir J. Y. Simpson, examining the head of a child that he had extracted with unusual facility after fracturing the bones with a Murphy's craniotomy forceps, found that the occiput had been broken close to the foramen magnum. He recognised the necessity for facilitating the process in every case by fracturing the base of the skull, and contrived the Cranioclast for the purpose. Modified by Carl Braun von Fernwald, the cranioclast has almost everywhere superseded the cephalotribe (Fig. 4). In using it the solid blade is introduced into the opening made in the vault; the fenestrated blade is applied outside the head, preferably over the occiput, the

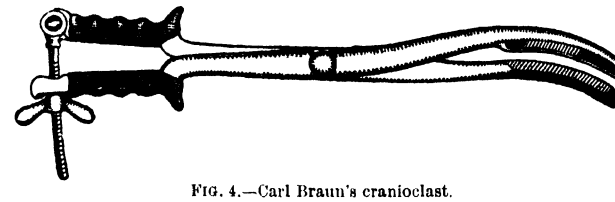


FIG. 4.—Carl Braun's cranioclast.

point of it carried as far as possible to the neck of the child. With a twisting movement the bones in the grasp of the blades are broken, and with the hold that has been obtained the operator makes an effort at extraction. If the head does not offer to descend easily he may relax the grasp and apply the outer blade to the side of the head or over the frontal bones, so as to fracture the skull more effectually.

(5) *Basilysis.*—When it is remembered that the firm, unyielding base of the skull is the greatest obstacle to the escape of the head, it

becomes obvious that the ideal operation must be directed towards the breaking-up and loosening of the basic bones. With this view, Fabbri of Bologna proposed the use of a pair of bone-pliers fashioned like a parrot's beak, which he described as a tenaglia, and which is intended to gouge out fragments of the sphenoid and neighbouring bones. Hubert of Louvain followed a procedure which he described at first as Sphenotresia, and afterwards Transforation. It was effected by means of an instrument consisting of a long rod with a bulbous point that could be screwed through the vault and on into the base of the skull, and of a blade, resembling a solid lever, to be passed round the head. The blade and rod can be fixed close to their handles, and are so constructed that the point of the screw, when it has pierced the floor of the skull, is received into a depression close to the tip of the blade. The Basiotribe of Tarnier is of somewhat similar construction, but is provided with two blades, so that if the compression from one side prove insufficient, the second blade can be applied to crush the head from the opposite direction. The Basilyst, which we have seen to produce most satisfactorily the necessary aperture in the cranial vault, was contrived especially to be used for tearing up and dissolving the basis cranii. When the point has been screwed into any part of the base, and the handles compressed, the bones are not merely perforated, but torn apart. The perforation and dilaceration may be applied, if need be, to several of the bones — the ethmoid, the sphenoid, the petrous portion of the temporal or basilar portions of the occiput—and such a complete dissolution of the base effected as to allow of easy collapse and compression of all the skull. Further, crushing the head is brought about, when it becomes necessary, by applying the tractor blade for the purpose of immediate delivery. This fits to the perforating part of the instrument as the fenestrated blade of the cranioclast fits its solid blade, and gives to the basilyst all the additional value of the cranioclast as an extractor (see Figs. 5 and 6).

III. *Extraction.*—If the head is effectively comminuted the expulsion may be left for a time to the natural efforts. The soft parts between head and pelvis, being relieved of pressure, recover their circulation, and allow of the more easy transit. The uterus, after retracting, resumes its contractions more vigorously, and if it succeeds in expelling its contents, as it is now free to do, there is less risk of trouble in the third stage and subsequently.

In most instances the practitioner finds it desirable or necessary to proceed to the extraction of the mutilated infant. At one time hooks, blunt or sharp, single or double, were applied inside the skull or outside the scalp, to get purchase for traction. The hooking out of the fœtus is now almost obsolete. The cephalotribe, powerful as a crusher, fails as an extractor, for it is apt to slip, and it is difficult with it to adapt the head to the pelvic diameters. The cranioclast or the basilyst-tractor has the advantage of introducing only the one (fenestrated) blade into the cavity of the uterus; the other blade plays its part entirely within the cranium. They allow of the adaptation of the head to the configuration of the pelvis in all its planes, and in dragging on the occiput tend to elongate the head in sugar-loaf fashion. In extracting, the operator (1) will see that he has taken a secure hold of the head, and it will be more satisfactorily done if the outer blade be placed over the hind-head. (2) He will see that

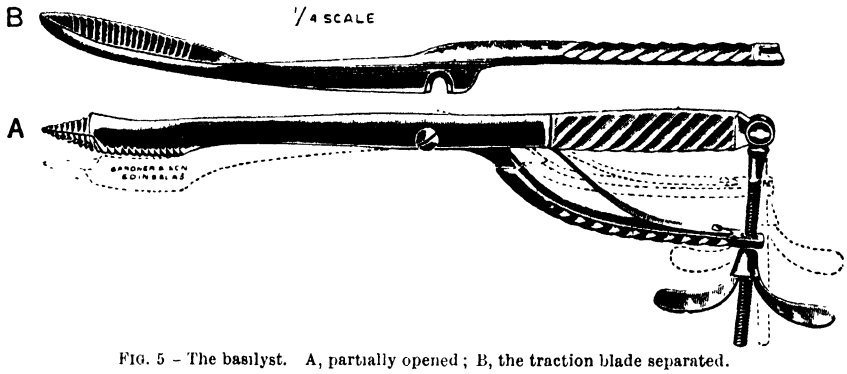



FIG. 5 – The basilyst. A, partially opened; B, the traction blade separated.

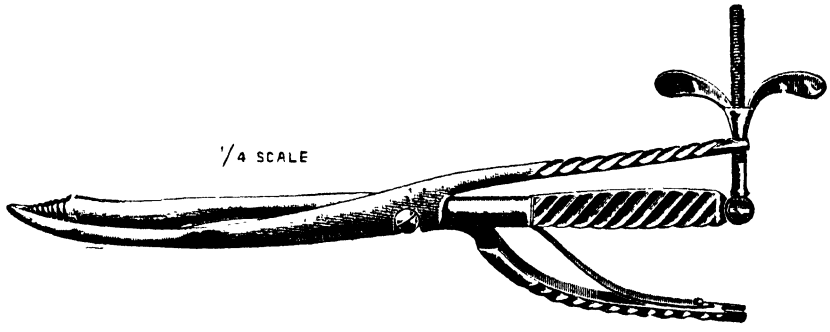



FIG. 6.—The basilyst with traction blade adapted.

the jagged edges of the bone are covered with the scalp, keeping the fingers of his left hand in contact with the head whilst he pulls with the right hand. (3) He will make traction during the pains, and make sure that the uterus is following and aids in the progress of the fœtus. (4) He will pull in the proper axes of the different parturient planes. (5) He must be prepared in some cases to lessen the bulk of the trunk, as by cleidotomy. (6) He should in all cases make careful examination of the maternal canals after the labour is over to see if there be any spicula of bone left or any laceration that may need repair. It goes without saying that the whole procedure must be conducted on aseptic principles, and that after such an operation it will be desirable to wash out the uterine cavity with an antiseptic douche, or at least with hot sterilised water.

### B. In Trunk Presentations

Indications. — There are a few cases of narrow pelvis where, after the head has been crushed, the thorax fails to enter the brim, and it becomes necessary to lessen the trunk also. But it is usually in cases of transverse presentation that the necessity arises for breaking up the trunk. A shoulder is presenting, possibly the arm has fallen to the vulva; the waters have all escaped; the uterus is closing firmly round the fœtus, which becomes impacted at the brim and cannot be driven through; the lower uterine segment, the cervix, and the vaginal roof are all becoming stretched and thinned, and in danger of rupture. The child is very likely already dead or dying, so that there is no hope of rescuing it by turning, which would now be dangerous to the mother. Her only chance lies in delivery by embryotomy.

Procedures.—There are different procedures that the practitioner may adopt in seeking to effect a diminution in the size of the passenger. Some have begun by removing the arm, cutting it off at the shoulder-joint, or better, passing a finger or hook or forceps through the wound in the axilla and bringing away the scapula, and perhaps the clavicle, along with the prolapsed extremity. Such mutilation, however, is usually futile. It does not reduce the bulk sufficiently to make version safe or to allow of the expulsion of the infant; and the operator has deprived himself of the safest medium for traction on the trunk if he removes the arm and then finds that he has to eviscerate the cavities or break through the spinal column. Cleidotomy (division of the clavicle), which allows of some degree of collapse of the upper part of the trunk, and may facilitate extraction through a pelvis that has required head comminution, is of little or no avail in transverse presentations.

I. *Evisceration.* — Delivery has often been effected by opening the thorax and abdomen with some variety of perforator and disembowelling the fœtus with the forceps or fingers. This Evisceration or Exenteration, as it has sometimes been called, does not usually secure sufficient diminution of the bulk of the infant to allow of its extraction. It only lessens the soft structures and leaves untouched the spinal column, the resistance of which constitutes the great difficulty to the passage of the trunk.

II. *Decapitation.*—If the spinal column can be severed in the cervical region by Decapitation of the fœtus, the difficulty is overcome. Different methods have been adopted for separating the head from the trunk. (1) The neck has been divided from below upwards with knives or scissors, a method dangerous both to the maternal tissues and the fingers of the operator. (2) Ramsbotham's sharp hook carried round the neck was at one time the approved implement for cutting through from above. But its cutting edge on the concavity of the hook was also not free from danger to mother or operator. (3) The chain or wire of an écraseur has been used, but the obstetrician may have difficulty in getting the chain or the wire adapted to the neck. (4) Pajot had a special hook constructed for carrying a loop of whip-cord or strong twine round the neck. A stout copper wire bent double can be curved so as to go round the neck, and as it is withdrawn it can carry back the cord which is to be used for sawing through the neck. (5) Braun's decapitator, which is a long blunt hook like a boot-hook, is the simplest and safest of all the decapitating implements. The curve of the hook is small, so that it is relatively easy of application; a half twist usually suffices to break the vertebral column, and an additional twist or two tears through the soft parts as well.

Delivery by this method implies, first, the Decapitation itself; second, Extraction of the Trunk (this will usually be brought about by pulling on the protruded arm); third, Birth of the detached Head. This may be effected by pressing upon the uterus from above; but sometimes the head requires to be laid hold of with forceps, or some kind of hook, or to be perforated and extracted with a cranioclast or basilyst.

III. *Spondylotomy or Spondylolysis.*—In some cases the neck is not accessible. The trunk of the fœtus has got jammed into the pelvis with the head above the brim, so that decapitation is impossible. In such cases Sir J. Y. Simpson proposed the use of the Spondylotome for cutting through the spinal column in its most accessible part, usually in the dorsal region. The spondylotome is a pair of strong scissors, one blade of which is sharp-pointed for perforating the thorax, whilst the other probe-pointed blade is passed outside over the vertebral column, so as to cut through the spine. A pair of bone-pliers may be used for the purpose. When the vertebral column has been thus divided the

trunk can be doubled together, and may be expelled by the natural efforts aided by pressure from above or traction from below with a hook or cranioclast. On the dead fœtus in a phantom it has been found that the basilyst can be made to perforate and tear up the spinal column even more thoroughly than the cutting instruments, and its use as a Spondylolyst is attended with this advantage, that after the vertebræ have been broken the tractor-blade can be at once applied outside the trunk so as to facilitate the extraction of the infant.

### 5*a*. Cleidotomy

DEFINITION AND HISTORY . . . 295
INDICATIONS . . . . . 295
TECHNIQUE . . . . . 296
ADVANTAGES . . . . . 296

*Definition and History.*—Cleidotomy, or division of the clavicles (Ger. *Schlüsselbeinschnitt*), is an operation accessory to craniotomy or basilysis, and has for its object the diminution of the width of the shoulders of the infant in cases of difficult labour. It is possible to imagine cases in which it might be performed as the sole operative procedure, *e.g.* in broad-shouldered anencephalic fœtuses, but I do not know of an instance of this mode of use.

No doubt obstetricians in the past, when confronted with such an emergency as impaction of the shoulders after birth of the head (either intact or craniotomised), adopted some plan of procedure for the reduction of the width of the bisacromial diameter; but they do not appear to have specialised the manœuvre into a definite operation, nor to have restricted the embryulcia to the clavicles. Dr. H. R. Spencer, writing in the *British Medical Journal* (April 13, 1895, p. 808), introduced the operation of cleidotomy, although he did not give it that or any other specific denomination. He said: "It may be necessary to reduce the width of the child's shoulders. With this object, I have found it a useful plan to snip through the clavicles with scissors; then, if necessary, to pass a blunt hook into the axilla, and then to bring down the arms." He went on to say that he had met with several cases where craniotomy was not sufficient to extract; so it may be concluded that he practised this snipping through of the clavicles on several occasions — "in about five or six cases." Soon after the publication of Spencer's article (which was entitled "On Delivery in Certain Cases of Impaction of the Trunk of the Fœtus"), there appeared a contribution from the pen of Professor N. N. Phänomenoff, of Kasan in Russia, named "Zur Frage über Embryotomie: Über die Durchschneidung des Schlüsselbeins (Cleidotomia)." Phänomenoff's article was in the *Centralblatt für Gynäkologie*, xix., p. 585, June 1, 1895. He was led to perform the operation on account of disproportion between the fœtus and the pelvic canals, due rather to great size of the former than to marked contraction of the latter, and more particularly to great size affecting the shoulders. The patient upon whom he operated had a true conjugate diameter of just under four inches. The head of the infant was without much difficulty brought down to the perineum by means of forceps, but no further progress could be accomplished; the infant meanwhile died, and Phänomenoff, suspecting wide shoulders impacted in the slightly narrowed maternal pelvis, passed up a pair of scissors above the brim and divided with some short snips ("durchschneidet mit einigen Kurzen Schnitten") both clavicles near their inner end; and strong traction on the head was now sufficient to bring one of the axillæ within reach, and a Braun's hook fixed therein sufficed to complete the delivery. The infant weighed 13 lbs., and the circumference of the shoulders was 41 cms.

In the same year (1895) Knorr showed to the Berlin Obstetrical Society an infant upon whom craniotomy, followed immediately by cleidotomy, had been performed (*Ztschr. f. Geburtsh. u. Gynak.*, xxxiv. 105, 1896). The patient was moribund from rupture of the uterus; craniotomy was carried out, and the head brought down with a cranioclast; then there being delay in the delivery of the trunk, both clavicles were divided with Siebold's scissors—the result was most astonishing ("der effect war sehr eclatant"), for a slight pull now delivered the trunk. The infant was very large. Strassman published three cases of cleidotomy (two unilateral, one bilateral) in 1897 (*Arch. f. Gynaek.*, liii. 135, 1897), in all of which the operation was preceded by that of craniotomy for large-sized fœtus, contracted vagina, and generally contracted pelvis. H. A. v. Guérard (*Centralbl. f. Gynäk.*, xxii. 71, 1898) performed cleidotomy after craniotomy for impaction of the head and then of the shoulders at the pelvic outlet, the distance between the tubera ischii being only 5·2 cm. (2 in.). Other contributions to the subject of cleidotomy have been made by E. Bonnaire (*Presse médicale*, No. 21, p. 125, 1900), by G. Kallinowsky (*Diss. inaug.*, Berlin, 1898), by Rieznikoff (*Yuzhno-russk. med. gaz.*, Odessa, v. 425, 1896), by Perondi (*Clin. ostet.*, Roma, ii. 133, 1900), and by myself (*Trans. Edin. Obstet. Soc.*, xxvi. p. 24, Nov. 1900).

*Indications.*—The indications for the operation of cleidotomy can hardly be said to be as yet quite clearly defined. It has not yet been performed, so far as I know, upon the living infant, although there are circumstances in which it might be justifiable to do so, *e.g.* in the cases of monstrosities such as anencephalus, double terata, etc. It is generally to be carried out as a sequel to the operation of craniotomy or basilysis, but it may be required apart from these other procedures. The indications may be roughly

grouped under five headings. 1st: It is indicated in the case of the broad-shouldered fœtus, where the head also is above the average size (and where, therefore, craniotomy will doubtless have been carried out), or where the head is of normal size or deformed (anencephalus or hydrocephalus), when cleidotomy may be the only operative procedure rendered necessary. 2nd: It is indicated in cases of contracted maternal pelvis, justo-minor, flat rachitic, or kyphotic, in which the fœtus may be normal in size. In such cases craniotomy is generally performed first and cleidotomy later; in the first and second types of contraction the division of the clavicles will have to be done at the brim of the pelvis, and in the third at the outlet. 3rd: There is the group of cases in which the fœtus is large and the pelvis moderately contracted; in these instances, it is the association of two difficulties neither of which is in itself sufficient to cause great delay, which renders cleidotomy necessary. 4th: Cleidotomy may be advisable in cases of breech presentation in which the shoulders are impacted. 5th: In all cases in which rapid delivery is called for (as in eclampsia, threatened uterine rupture, accidental hæmorrhage), and in which the fœtus is either dead or its chances of survival very small.

*Technique of the Operation.*—The operation can be carried out very easily, and requires little or no special skill. The only instrument necessary is a long and strong pair of blunt-pointed scissors. Craniotomy, or craniotomy followed by basilysis has been performed, and the comminuted head has been drawn down to the perineum by the cranioclast or the basilyst tractor, or by a crotchet. Delay now occurs due to the impaction of the shoulders in the contracted brim of the pelvis. By continued traction the difficulty may possibly be overcome at the risk of injury to the maternal structures, and with the loss of valuable time and the expenditure of much force. In order to prevent this delay and these dangers, division of one or both clavicles is enough. If the hand be lying with the occiput to the front, it is drawn well forward; the operator then slips his left hand up the vagina over the face of the child till he feels the clavicles projecting from the thorax at the level of the pelvic brim; then he insinuates the scissors, held in the right hand, between his left hand and the child, until he is able to grasp the clavicle of one side between their points; and then with a strong snip he divides the bone near its inner end. The process may be repeated on the clavicle of the opposite side. The immediate result is an astonishing collapse of the shoulders, and the extraction of the infant's trunk with very little further effort, or, at the most, with the help of traction with a blunt hook upon the axilla. I have done this operation four times as an accessory procedure after basilysis, and on all the occasions I found it quite simple and immediately satisfactory. In one case alone, in which the infant was very large and the maternal pelvis moderately contracted, I experienced some difficulty in dividing the clavicles, which showed an unusual degree of ossification.

If the contraction of the pelvis be at the outlet the procedure is not materially altered—indeed, it is simplified; for craniotomy permits the extraction of the head from the vulva, and the clavicles are brought within the range of vision and can be divided with complete ease. The same remark applies to the performance of cleidotomy in cases of the birth of an anencephalic fœtus. The small size of the deformed fœtal head permits the hand of the operator easily to reach the shoulders of the fœtus impacted in the brim of the pelvis, or within the pelvic cavity, and so the division of the bones with strong scissors can be easily carried out. The skin wound is a small one, and can be closed with one or two sutures for appearance' sake.

*Advantages.*—If we compare cleidotomy with the only other procedures possible under the circumstances, it will become at once apparent that it excels them all. It must, for instance, be better than *simple traction* on the craniotomised head of the infant, with the loss of time, the risk of injury to the mother's part, and the danger of separation of the head from the trunk of the child, which all accompany that method of trying to overcome the delay. *Cleidotripsy*, or the breaking up of the clavicles and of the tissues of the shoulders with a perforator or basilyst, is an awkward procedure, not easy of accomplishment; it is quite unnecessary also, for simple division of the clavicle is all that is needed. *Cleidorrhexis* is a sort of bruising or breaking of the shoulder-girdle carried out of set purpose, and performed by some obstetricians (*e.g.* A. Muller, *Monatsschr. f. Geburtsh.*, viii. 477, 1898). It is used in cases in which the body of the child is born and in which the shoulders and head are still at or above the brim; and it consists in carrying the trunk first forward and then backward, until the shoulder-girdle gives way and moulding occurs. *Supra-acromiotomy* is another operative procedure, and it has been described by Bonnaire (*loc. cit.*). It consists in a localised embryotomy by which the skin and muscles covering the projection of the shoulder are divided widely and deeply; but it is not an exact operation, although it may conceivably be useful in cases in which the clavicles cannot be reached.

It is quite evident that cleidotomy has advantages over all these competitive procedures. The division of the clavicles at once, and very materially, reduces the bisacromial diameter by allowing the shoulders to collapse; indeed, the clavicles alone keep the shoulders apart. It has been found by experiment that

there is a diminution of the bisacromial diameter by 2 and 3 cm. in unilateral and bilateral cleidotomy respectively. This narrowing of the shoulders either permits them to enter the pelvic cavity, or gives room for the passage of a blunt hook upwards and for the fixing of it in the axilla in order to make traction. Division of the clavicles is the one thing necessary, and cleidotomy alone of all the proposed plans does this and no more. It is simpler, more rapid, much more elegant, and it is quite as effective as any of the other operative procedures; it is, further, founded upon the correct scientific principle of attacking the parts which are concerned in maintaining the breadth of the shoulders. The cause of delay in labour in these cases is the width of the shoulders in the bisacromial diameter; by cleidotomy this diameter is quickly diminished, and with practically no risk to the mother.

### 6. Symphysiotomy

An extremely clear and judicious estimate of the relative position of this to other obstetric operations has recently been given by Professor Bar in his *Leçons de pathologie obstétricale.* Combining his own results in twenty-three cases with those of Pinard, Zweifel, and Küstner, he finds that the present maternal mortality in symphysiotomy is 7·45 per cent, and the infantile mortality 9·3 per cent.

Indications.—Symphysiotomy came on the field in the interests of the mothers whose lives were sacrificed in Cæsarean operations, and whilst it is still chosen in certain cases as a safe alternative to these procedures, its employment is more urgently advocated in the interests of the infants whose lives are sacrificed in embryotomy operations. It is intended to widen the canals with less danger to the mother than was implied in opening the peritoneal cavity and the uterus, and it allows of the passage of the undiminished head of the infant in cases of pelvic contraction. The form of pelvic contraction to which it is best adapted is the pelvis *æquabiliter justo minor*, but it is had recourse to also in the rickety and osteomalacic pelves. In the Naegelé pelvis, as Farabœuf has shown, section of the symphysis should be avoided, and, instead, the innominate bone on the side of the synostotic sacro-iliac joint should be sawn through the ischio-pubic rami, so as to allow of expansion of the bony girdle at the double hinge of the symphysis pubis and of the healthy sacro-iliac joint. Symphysiotomy has been performed in women with pelves measuring as much as 9 cm. (3¾ ins.) in the conjugate of the brim; but we expect in pelves with this space to witness the birth of the child under the natural efforts or with the aid of forceps or turning. On the other hand, where the conjugate is reduced to 2½ ins. (6·3 cm.), it is hardly possible that sufficient expansion would occur for the extraction of a living child, unless, according to the suggestion of Novi, symphysiotomy be conjoined with induction of premature labour. Morisani's minimum contraction of the conjugate of 6·7 cm. (2⅝ ins.) should be maintained as the utmost limit in this direction for symphysiotomy interference, and 9 cm. (3½ ins.) in the other, and the operation should, as a rule, be reserved for multiparous women, in whom the dilatation which the soft parts have undergone during their previous labours will lessen the dangers both to mother and child. The operator must, of course, make sure that the infant is alive and vigorous.

*Methods of Operating.*—Consideration has to be given to:—

1. *The First Stage of the Labour.*—In some cases the labour is already so far advanced that the more strictly surgical interference is at once demanded. In these patients too often there have been preliminary attempts at delivery with forceps or by turning, that may have seriously impaired the prognosis for mother and child.

With a pelvis at the higher limit of contraction—say from 8 to 9 cm. (3 to 3½ ins.)—it may be permissible to make cautious use of these alternative operations in cases where yet the operator fears he may have to divide the symphysis; but where the contraction approaches the lower limit such interference would be futile and dangerous. Only in some cases of multiparous women, where the operator has made up his mind eventually to deliver by turning and extraction on the limbs, he may bring about the preliminary version by external manipulation, or by the bi-polar method whilst the membranes are still unbroken, so as to facilitate the seizure of the leg when the moment for extraction shall have come. It is to be remembered that in women with contracted pelves the first stage of labour is apt to be prolonged, and it may sometimes be of advantage to promote the dilatation of the cervix and of the vagina by means of douches and hydrostatic dilators. The operator has to take note further of any complication that may be present, such as prolapsus funis, and prepares to intervene only when the first stage is completed and the cervical canal is fully dilated.

II. *The Division of the Symphysis.*—It goes without saying, that as all obstetric procedures should be carried out under as aseptic conditions as possible, so especially in this surgical intervention operators are careful to have the whole lower abdominal and pudendal region shaved and cleansed, and the parts and passages disinfected. Three different methods have been adopted in cutting through the symphysis.

1. *The Neapolitan Method.*—Morisani and his school usually make an incision of 1½ inches to 2 inches in length through the skin and

subcutaneous structures in the hypogastrium and mons veneris. The lower extremity of the incision terminates just above the upper margin of the symphysis pubis, and the deep part is widened by dissecting some of the muscular fibres from the pubic bone. An assistant pulls the urethra and bladder backwards with a metallic catheter, and the operator, pushing his finger down behind the symphysis, detaches the loose retro-pubic connective tissue, and guides a Galbiati knife down below the lower margin of the symphysis. With this knife, probe-pointed and sickle-shaped, with the cutting edge in its concavity, he divides the interosseous cartilage and ligaments from below upwards and from behind forwards, until a creaking sensation tells him the section is complete, and he feels the bodies of the pubic bones spring apart. This symphysiotomy is thus almost a subcutaneous operation.

2. *The Parisian Method.*—M. Pinard and his confrères divide the structures of the mons veneris in front of the symphysis in their entire length from above the pelvic brim to below the anterior commissure of the vulva, close to the clitoris, by an incision from 3 to 4 inches in length. When the pubic joint is exposed the recti in the upper part of the wound are separated to allow of the entrance of the finger into the pre-vesical cavity; this guides the bistoury which cuts through the symphysis from above downwards, and before backwards.

3. *The Leipzig Method.*—Professor Zweifel makes a transverse incision through the structures in front of the symphysis, a finger's breadth or a little more below its upper margin. The fascia over the joint and for some distance above towards the umbilicus is laid bare, and the lips of the wound pushed up and down with the finger-nail till the lower margin of the subpubic ligament is exposed. The fascia between the recti is now divided, and a finger being pushed down into the recto-pubic space, the cartilage is cut through with a probe-pointed knife from above and in front downwards and backwards.

Some operators have had difficulty in hitting on the joint tissues, and have had recourse to the use of a saw or chisel to divide the osseous structures. This adds greatly to the dangers of the operation, and where patients survive the convalescence is prolonged and perhaps imperfect. Dr. Sandstein, in his graduation thesis, points out that in 50 per cent only is the symphysis median; in 33⅓ per cent it is to the left, and in 16⅔ per cent to the right of the middle line, and that, further, it frequently slopes towards one side instead of being vertical. He finds a guide to its upper margin in a projection that can always be felt between the pubic bones at their upper margin, and that is continued as a ridge on the posterior aspect of the symphysis. When the symphysial tissues are severed, the bodies of the pubic bones spring apart spontaneously to a distance of 2 to 4 cm. (¾ in. to 1½ ins.). If the patient is in the Walcher position, with the legs hanging down over the end of the table, the separation is greater. Zweifel avails himself of this tendency of the dependency of the limbs to strain the symphysial structures at their upper margin to facilitate their section. Where a greater separation of the bones is demanded, pressure is made on the inside of the limbs flexed upwards on the abdomen. They have sometimes been separated as much as 7·5 cm. (3 ins.); but a separation of from 5 to 6 cm. (2 ins. to 2⅜ ins.), or fully two fingers' breadth, gives the full advantage of the expansion without endangering the sacro-iliac ligaments. That one side of the pelvis is moving more than the other can be recognised, according to Sandstein, by noting that the upper margin of the less mobile innominate remains at a higher level, and then it becomes necessary to make pressure on the inner side of the corresponding thigh, to stretch the sacro-iliac joint, and to prevent the sacro-iliac ligaments of the mobile side from being unduly strained and torn. Sometimes there is free bleeding from the incised soft parts that has to be checked with pressure forceps, or by the pressure of the fingers of the assistants who are controlling on the two sides the movements of the limbs. The wound is packed with iodoform gauze, or otherwise kept from the chance of any germ-invasion, whilst attention is given to—

III. *The Birth of the Child.*—The dilatation of the pelvis may be sufficient to allow of the expulsion of the infant by the natural powers. During the transit of the head the pubic bones may be further separated and a fresh access of hæmorrhage set up; hence it is necessary to have an intelligent assistant at each side who will keep up such pressure on the trochanters as will prevent undue and dangerous distension. Where the parturient powers are insufficient for the expulsion, the operator must have recourse to the extraction of the infant, either by means of version and traction on its leg, or by application of forceps to its head, according to the conditions that emerge in each individual case. Whether the child be born naturally or extracted artificially, it has to be borne in mind that the anterior vaginal wall and the soft parts in relation with it are deprived of their usual bony protection, and may easily be stretched and torn to a degree that will endanger the patient's life or render her recovery imperfect. After the birth of the infant the obstetrician sees to the complete evacuation of the uterus and its usual post-partum contraction, before he turns again to complete the surgical intervention necessary to secure the union of the severed surfaces.

IV. *Closure of the Wound.*—The Neapolitan procedure at this stage simply requires the care-

ful suturing of the suprapubic incision, and the keeping together of the bodies of the pubic bones so as to secure the reunion of the symphysis by mean of carefully adjusted bandages. Operators who have laid bare the pubic bones before dividing the symphysis have in some instances drilled the bones so as to allow of their being kept together by a couple of metallic sutures. For the most part it has been found sufficient to pass silk sutures through the aponeurotic tissues, and after the soft parts of the wound have been duly sutured, to apply a bandage or keep the pelvis immobile in a hammock or in a special framework. Care must be observed in bringing together the separated bones that none of the soft parts that lie behind get caught between them, else damage may ensue to the bladder or urethra.

Results.—In 1892 Spinelli gave a résumé of twenty-four cases of symphysiotomy that had been carried out by Morisani and his colleagues in Naples during the preceding fifteen years, which showed that all the mothers had recovered, and only one child had been lost. In four of the instances the operation was performed twice in the same patient. One woman had her second symphysiotomy fourteen months after her first. Another, who had been able to be out of bed by the eighth day, was operated on seventeen months later, and got up after the second symphysiotomy on the seventh day. These results, and the statistics of maternal and infantile mortality already quoted from Bar, give, however, an unduly favourable impression of the life-chances attendant on this mode of delivery. The danger to the life of the mother arises mainly from the double possibility of septic absorption through the long-exposed pubic wound, and through the bruised and fissured parts of the genital canal, but also from the hæmorrhages, primary or secondary, from the wound or from the uterus. It has to be kept in view, moreover, that there may be protracted convalescence because of inflammations set up at the damaged joints, pubic or sacro-iliac, or among the areolar tissues of the pelvis, or in the bladder and urethra. Whilst, as a rule, the patient makes a complete recovery, she sometimes suffers afterwards from impaired power of progression when the reunion of the joint-ligaments has not been complete, from loss of retentive power in the bladder, or from prolapsus uteri.

### 7. Cæsarean Section and its Modifications

Cæsarean section is an operation whereby an opening is made in the abdominal wall, and another in the uterus, through which the fœtus is extracted.

According to Pliny, it is named Cæsarean because the first of the Cæsars was so extracted from his mother's womb as she was dying. According to another version, it is named from the operation itself, *cæso matris utero.*

This operation was at first made upon dead women at a more or less advanced state of pregnancy. It is attributed to Numa Pompilius, one of the first kings of Rome, who enacted (*lex regia*) that a pregnant woman deceased could not be interred until the fœtus was extracted. This law remained in operation throughout all countries under Roman rule, and was approved by the Church, as well as adopted as a civil law by northern states, more especially Germany. For many years they dared not perform the operation upon a living woman, and in this way encouraged the performance of craniotomy, as the passage of the fœtus through the pelvis in cases of deformity was impossible without mutilation.

Levret and Mauriceau deny that this operation was known to the ancients, but Dionis and Gardien refer to Pliny's *Natural History.* Dr. Mansfield published a work "On the Antiquity of Gastrotomy and Hysterotomy on the Living" (*Weber, das Alter des Bauch- und Gebärmutterschnitts am Lebenden zu Braunschweig,* 1824).

He states that in an earlier work named *Mischnajoth,* written about 1400, there is this passage: "In a twin birth, neither the first child which by section of the belly is brought into the world, nor the one coming after, can attain the rights of heirship or priestly office."

Nicolai Falconiis recorded a case at Venice in 1491. The case of Jacob Niefer, the Swiss peasant who performed it upon his own wife, is frequently quoted; but most authorities are agreed that it was much later before it was generally attempted upon the living woman. In fact, we need only refer to the action of Mauriceau in the case treated by himself and Chamberlen, where the operation was delayed till after death, although Mauriceau was in actual attendance for several days. He wrote: "The child has been dead to all appearance about four days, and I told all the assistants that she could not be delivered. They asked me to perform Cæsarean section, which I did not wish to do, knowing that it was always certain death to the mother." This poor woman died with her infant *in utero* twenty-four hours afterwards.

Rousset, physician to Catherine de Medicis, and contemporary of Paré, published a work upon the subject in 1581. This book was translated into Latin about ten years later. The author attempted to prove the possibility of saving the mother and child by means of this operation, but his views were opposed by Paré, Guillemeau, and others. In the middle of last century the subject divided operators into two sections, the Symphyseans and Cæsareans, or those who advocated division of the symphysis pubis and those who advocated Cæsarean section.

It may be taken as a recognised rule in midwifery that no woman should be allowed to die undelivered without some attempt being made

to save her and her offspring, or at least to save her at the expense of her child.

Concerning the latter point, whether we are justified in destroying the infant when alive, there has been, and still exists, difference of opinion, due in some measure to religious belief, and likewise to the personal feeling of the husband, who felt that very little hope was held out to him that his wife could be saved by section. Amongst such men we had Napoleon, who, when appealed to by Dubois, said: "Treat the Empress as you would a shopkeeper's wife in the Rue St. Martin; but, if one life must be lost, by all means save the mother." In marked contrast to him we had Henry VIII., who, when thus questioned before the birth of his son Edward, exclaimed: "Save the child by all means, for other wives can be easily found." At the present time such men might be put down as either a good husband but a bad father, or a good father but a bad husband.

The doctrine of the Roman Catholic Church has been that, if you could not extract the child without killing it, you could not, without mortal sin, do so; and likewise, until lately, it was held that the infant could not be baptised in the uterus, as it should be *natus* before it could be *renatus* by baptism.

Of late years the happy results following Cæsarean section and Porro's operation have done much to efface the dreadful feeling that we have got in such cases to decide whether the life of the mother or that of the child is to have our preference, seeing it is now quite possible to save both.

Barnes wrote: "Cæsarean section is resorted to with a feeling akin to despair. Embryotomy stands first, and must be adopted in every case where it can be carried out without injuring the mother. Cæsarean section comes last, and must be resorted to in those cases where embryotomy is either impracticable, or cannot be carried out without injuring the mother. There is, therefore, no election. The law is defined and clear. Cæsarean section is the last refuge of stern necessity."

As against this statement, Dr. Barnes has recently said: "It is no longer permitted to us, without ample proof of clear necessity, to sacrifice the child in order to save the mother. The cases in which the two lives are supposed to stand in antagonism are vanishing before the light of modern science and skill."

If anything is needed to sicken one at the revolting practice of craniotomy, I might be allowed to relate the obstetric history of a rachitic woman, who during her three last confinements was under my personal care:—

| | |
|---|---|
| 1st—1862, . . | Embryotomy. |
| 2nd—1863, . . | Embryotomy (labour induced). |
| 3rd—1864, . . | Embryotomy. |
| 4th—1865, . . | Induced labour at half term. |
| 5th— — , . . | Embryotomy (Birmingham, L.I. Hosp.). |
| 6th—1868, . . | Induced labour at half term. |
| 7th—1870, . . | Embryotomy. |
| 8th—1871, . . | Embryotomy (eighth month). |
| 9th—1873, . . | Embryotomy. |
| 10th—1874, . . | Embryotomy. |
| 11th—1875, . . | Induced labour at half term. |

We must never forget that we have a sacred trust, and I hold we have no right to sacrifice a child, however unequal its life may be in some cases to that of the mother. In advocating the preference of section as against craniotomy in the living child, I do so only after very mature consideration, and with a feeling that to do otherwise would be to sacrifice a life which I was bound to preserve. I think the time has come when the lives of the mother and child may alike be saved, and prefer to think that an infant come to maturity is destined for something greater than to have its glimmering life extinguished by an accoucheur skilled in the use of a dreadful perforator. Let our motto be, "We live to save and not to destroy."

In another case where the obstetric history was like the preceding one, Cæsarean section was performed, and the mother now attained her long-wished-for desire, a living child.

Burns in twenty-four cases gave twenty-two deaths, whilst others gave the death-rate as from 50 per cent. to 100 per cent.

With such results, it is not to be wondered at that so many opposed the operation. In England, for example, accoucheurs condemned it absolutely. In Paris, during half a century, there was not a successful case, although it had been performed about sixty times. In the large Maternity Hospitals of Paris and Vienna, with from four to eight thousand confinements in the year, not a single successful case of Cæsarean section has been recorded. No doubt now exists that the great fatality was due to the fact that the operation was only resorted to after other measures had failed.

*Indications for the Operation.*—As regards the general indications for the operation, of course they vary in the hands of different operators, as some, still looking upon Cæsarean section as a last resource, divide the indications into absolute and relative. The absolute is where the deformity of the pelvis is so pronounced that the passage of even a mutilated fœtus is impossible, whilst the relative is where they may remove a mutilated fœtus by the natural passage with as good or a better result for the mother. It is here that difference of opinion exists. Baudelocque admitted Cæsarean section in cases with a conjugate diameter under $2\frac{1}{2}$ inches; Cazeaux, 2 inches; Tarnier, 2 inches; and Depaul from $1\frac{1}{2}$ to $2\frac{1}{4}$ inches where the child was alive, and under $1\frac{1}{2}$ inches when the fœtus

was dead. Stolz advocated Cæsarean section whenever the child was alive and could not be brought through the natural passage.

| | |
|---|---|
| Scanzoni . . . . | under 3 in. |
| Naegelé and Spiegelberg . | „ 2 „ |
| Barnes, Playfair, and Leishman | „ 1½ „ |

Of late years the good results following Cæsarean section in the hands of Cameron, Leopold, Sänger, and other operators have materially changed the views of many authors, who now favour Cæsarean section more than they have done in the past.

Lusk, at the International Congress held at Washington in 1887, declared that Cæsarean section was preferable to embryotomy even with a conjugate diameter from 2½ to 3 inches when the child was alive.

It can well be urged that:—

(1) Embryotomy in a very contracted pelvis is as dangerous to the mother as Cæsarean section.

(2) Embryotomy always compromises the life of the child, whilst Cæsarean section gives a living child.

(3) No one has any right to sacrifice a child where he can save it without exposing the mother to any additional risk.

For these reasons the operation should be one of election when the child is alive, and it should be performed before the patient is exhausted—in fact, early after labour has commenced, or even at full term before labour sets in, especially in multiparæ. In all cases it should be done before rupture of the membranes, and, if possible, the patient should be placed under the care of an experienced operator.

Little difficulty is experienced in obtaining the consent of the patient and her friends; and it is better to have her under observation previous to the operation, so as to regulate her diet, and have her prepared for operation beforehand.

A very important point in favour of Cæsarean section is that the Fallopian tubes can be tied and divided, so as to prevent subsequent conception, whereas embryotomy may require to be performed ten or a dozen times.

Besides deformity of the pelvis, other conditions, such as tumours or cancer of the cervix uteri, may exist which would demand either Cæsarean section or some modification of it.

If the child be dead and the conjugate diameter not over 1½ inches, Cæsarean section should be done.

Rousset, the earliest writer upon this subject, recognised the indications, the one furnished by the fœtus, and the other by the mother. Under the first category he placed excessive size of the fœtus, monstrosities, and faulty positions; under the second he placed marked contractions from whatever cause. Some operators would include placenta prævia and puerperal convulsions. Cæsarean section might be advisable in some cases of eclampsia, but a skilful obstetrician would never think of such procedure in the case of placenta prævia. In fact, the operators who advocate this step are surgeons who have little or no experience in obstetric practice.

Our decision for operation should be based upon the degree of contraction of the pelvis, the size of the child's head, and its reducibility, unless the obstruction is due to some other cause, such as cancer or the presence of a tumour in the pelvic cavity.

Every practitioner should be able to form a fair estimate of the amount of contraction, as it is easier to measure a contracted pelvis than a normal one; and it does not require a highly skilled obstetrician to say before labour has commenced, or during the early stage of the process, that the diameter of the pelvis is, or is not less than 3 inches; and, as a matter of fact, such a pronouncement should be within the skill of the ordinary practitioner, who should be more than a generally useful person, otherwise he will sink to the level of an ignorant midwife. Not only must he be able to form an estimate of the amount of contraction, but, by patient study of normal cases, qualify himself to form an opinion as to whether it will be impossible for a living child to pass, and also whether, under the difficult circumstances in which he may be placed, it would not be better to send the patient where Cæsarean section could be safely performed than to extract a mutilated fœtus through a minimum diameter.

With a diameter under 2½ inches, where engagement of the head is impossible, no one should hesitate to advise Cæsarean section, although there will always remain cases, such as where the child is dead or a subject of hydrocephalus, in which craniotomy may be resorted to.

Experience alone will enable one to avoid extreme measures in cases with a conjugate diameter measuring more than 3 inches, and where the skilled practitioner will weigh the chances between premature induction of labour and symphysiotomy.

There can be no questioning that Cæsarean section is a highly dangerous operation; but the danger, it should be remembered, depends for the most part on delay, and death most frequently results not so much from the operation, as from previous operative abuse, which is the just term for all injudicious attempts to extract the fœtus through a deformed natural passage.

Success depends upon prompt interference before the patient is exhausted, as then there is less danger from hæmorrhage, delayed shock, or peritonitis.

When Cæsarean section has been resolved upon, another question presents itself, namely, whether Cæsarean section or Porro's operation is preferable. If the former, there still remains

to be decided whether the operation will be accompanied or followed by a removal of the ovaries, or the patient be sterilised by the simple expedient of tying and dividing the Fallopian tubes. This we have done in about fifty cases, and no harm has resulted, although theorists would have us believe that such a procedure would be surely followed by hæmatocele. Where there is a choice of operation Cæsarean section is to be preferred, as it can be completed much sooner, and is free from the danger of shock and peritonitis which might complicate Porro's operation.

The preparation of the patient will depend upon the urgency of the case. When she is under observation it is better to confine her to bed for a couple of days beforehand, and the bowels should be moved by an enema and a slight laxative. The abdomen is washed and gently scrubbed, and the parts shaved, whilst the vagina is cleaned and rendered aseptic. The preparation, in fact, is the same as in any other abdominal section. The operator and his assistants who have to do with the case must be exceptionally careful in cleansing and disinfecting their hands, whilst the chief nurse should see that the instruments and sponges are sterilised and counted.

Very few instruments are necessary, and should comprise two straight scalpels and one blunt-pointed bistoury, pressure forceps, dissecting forceps, scissors, director, twenty straight $2\frac{1}{2}$-inch Hagedorn needles, compression pessary, aseptic silk, silk-worm gut, adhesive plaster, and dressings.

The catheter should always be passed into the bladder shortly before operation. The needles should be threaded in pairs beforehand, with No. 3 Chinese twist silk ligatures, about 20 inches long, and placed in a towel wrung out of 1-30 carbolic solution ready for use.

Palpation will reveal the position of the fœtus, and this is all the more important, as from this you will know the attachment or site of the placenta.

Briefly, my experience in Cæsarean section has shown me that in dorso-posterior positions the placenta is attached upon the anterior wall, whilst in dorso-anterior positions the placenta is upon the posterior wall.

(*a*) Thus, in the first cranial position, or O. L. A., the placenta will be found upon the posterior wall, and somewhat to the right side.

(*b*) In the second cranial position, or O. D. A., the placenta will be upon the posterior wall, and somewhat to the left side.

(*c*) In the third cranial position, or O. D. P., the placenta will be upon the anterior wall, and somewhat to the left side.

(*d*) In the fourth cranial position, or O. L. P., the placenta will be upon the anterior wall, and somewhat to the right side.

The fœtus and placenta will be found in the same relation in the various pelvic positions.

From this knowledge you know when your uterine incision is likely to cut down upon the placenta, and you also form an idea as to how you will extract the fœtus. The abdominal incision should be made in the median line, as in ovariotomy, and it will vary in situation according to the distension of the abdominal wall.

Thus, if the abdomen takes the form as seen in Fig. 1, an incision of from 5 to 6 inches may be got without extending beyond the umbilicus; but when it is pendulous as in Fig. 2, the incision must of necessity extend more or less above the umbilicus.

Before opening the uterus the operator should satisfy himself that the uterus is not only in the median line, but that it is not twisted upon its axis. This is found by locating the position of the Fallopian tubes by means of the fingers. He will frequently find the left tube more or less in front, as the uterus is usually rotated to the right. This displacement must be corrected, and, if necessary, an assistant can easily keep the uterus in position by pressing with his hand on the right side.

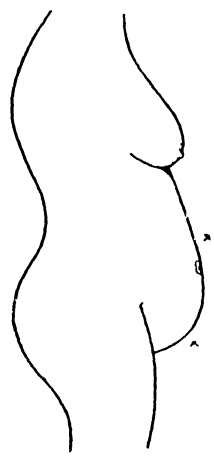


FIG. 1.

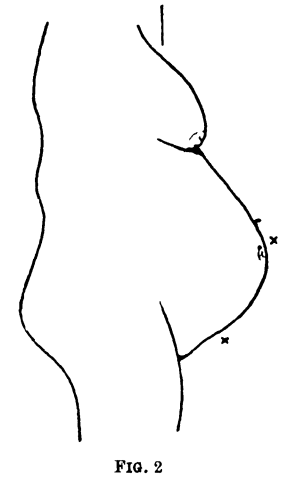


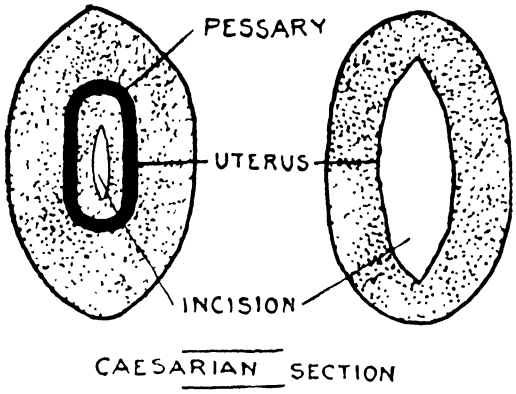
FIG. 2

When the placenta has its attachment upon the anterior wall the site is seen to bulge, and upon palpation has a fluctuant feeling akin to a large pointing abscess.

The next point is to open the uterus with as little loss of blood as possible, and this can be done by placing a flat vulcanite pessary upon the uterine wall around the point to be incised, as in Fig. 3.

The operator with the fingers of his left hand applies pressure upon the pessary, whilst his assistant does the same on the opposite side. The incision is then made with two or three strokes of the scalpel, and the blood sponged away by the assistant with his right hand.

After this has been done no more bleeding takes place unless the placenta is attached in front, as the pressure with the pessary thoroughly prevents even oozing. Care should be taken




Figs 3, 4.

not to puncture the membranes, which will soon be observed and recognised by their pearly colour. If the placenta intervenes, this method of pressure is beneficial not only in preventing bleeding, but also in permitting us to observe its tissue, which is recognised by its darker colour.

Whenever the membranes are reached a director is placed within the opening, which is then enlarged with a blunt-pointed bistoury, upwards and downwards as far as the pessary will admit. At this stage the compression pessary is removed, and the incision extended upwards and downwards sufficiently to permit the passage of the fœtus. The extension of the incision downwards should be limited, as it is likely to interfere with proper contraction of the uterus. Should the placenta intervene, it must be dealt with as a placenta prævia after completing the incision—that is, either separated upon one side, or, if central, pierced by the hand. There must be no hesitation in extending the incision, which is made upwards and downwards from within outwards in each direction with a blunt-pointed bistoury, so as to make an incision of about 5 or 6 inches, as in Fig. 4. The left hand is inserted without rupturing the membranes till the head is being turned out, or the feet grasped, and then the child should be extracted without delay. On no account should the hand be withdrawn after its insertion, unless during extraction of the fœtus, as the uterus speedily contracts. If the shoulder presents, a hand should be placed upon it to prevent its expulsion, as it adds very much to the difficulty when any portion of the child's body is allowed to protrude.

The child having been extracted, the assistant places a large flat sponge over the upper angle of the abdominal incision, to prevent the bowels from escaping, and then with both hands grasps the uterus, so as to prevent bleeding.

The cord having been tied and divided, the placenta is immediately removed with the left hand, great care being taken to secure the removal of all membranes and to prevent the entrance of blood into the peritoneal cavity. The assistant now everts the uterus from the cavity, and pushes a flat sponge behind it. The lips of the uterine wound are next everted, the assistant grasping the upper angle and wall with his right hand, and the lower angle and wall with the left, as in Fig. 5.

The operator immediately inserts the silk ligatures, beginning at the middle, each suture grasping the entire wall, with the exception of the mucosa. From seven to ten sutures should suffice, as with the contraction of the uterus the incision is greatly diminished.

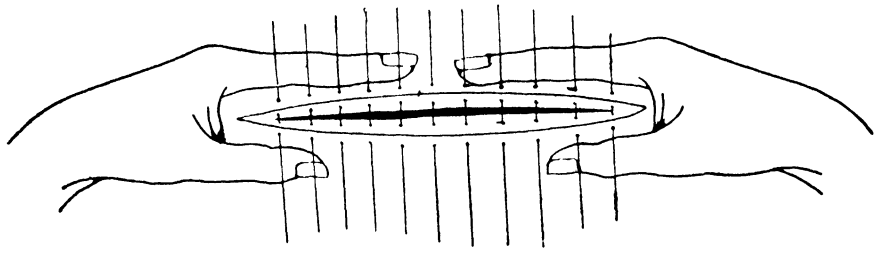


Fig. 5.

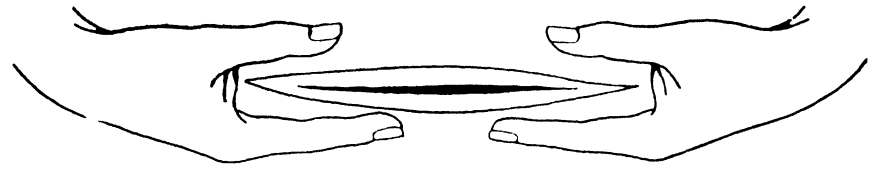


Fig. 6.

This accomplished, the sutures are gathered up and a large flat sponge laid over the anterior wall, and another behind. Firm compression or kneading is then made through the sponges, with the result that the uterus contracts firmly. The assistant should again seize the uterus as before, whilst the operator ties the sutures. When this has been accomplished the whole organ is enveloped in a large, warm, flat sponge, and firm compression again made so as to ensure thorough contraction. Should any oozing appear at the needle punctures, a second warm

sponge should be applied, and very slight compression will suffice to overcome any tendency to relaxation. Should the peritoneal edges gape at any points, a few superficial fine sutures should be inserted to bring the margins together.

The performance of hysterectomy for bleeding is bad treatment, and indicates that the operator has lost his nerve, as pressure with a warm sponge with both hands never fails to secure thorough contraction.

Several operators advise the introduction of a drainage tube through the cervix and vagina, and the leaving it there to act as a drain. Nothing could be worse. Of course, it is the procedure of a surgeon; but every one who has practised midwifery knows that the presence even of a clot in the uterus may lead to serious hæmorrhage. Such a body as a tube, if not expelled, would induce hæmorrhage, distension of the uterus, and bursting of the incision, with speedy death of the patient. This is no mere theory, but is what has actually taken place where drainage has been resorted to. On no condition should the uterine cavity be washed out or medicated in any way; the less the parts are interfered with the better.

After the ligatures have been cut short, the next step is to ligature and divide the Fallopian tubes with aseptic silk in order to prevent future conception. Of course, the consent of the patient for this procedure should have been obtained beforehand. Two ligatures are tied upon each tube, which is then divided between those points; this method is effective, and leads to no complications nor bad results, nor is menstruation interfered with. Next, the cavity is cleaned by the removal of all clot, etc., and the uterus replaced. The external wound in the parietes is closed in the usual way with silk-worm sutures. The vagina should now be cleansed of all clot and sponged out, after which an antiseptic pad should be applied to the vulva.

The wound should be dusted with iodoform, and a few layers of gauze placed over the wound. This should be secured with plaster, both to prevent slipping of the dressing and strain on the sutures, in case of sickness or cough. A sheet of Gamgee is next applied, and then the bandage.

The after-treatment consists of sips of warm water, say a tea-spoonful every fifteen minutes for twelve or twenty-four hours, after which milk and soda may be given in increasing quantities. For a few nights a half-grain morphine suppository is given. The urine should be drawn off every six hours for two or three days, care being taken to thoroughly cleanse the parts before doing so.

On the fourth day two tea-spoonfuls of glycerine in a couple of ounces of soapy water is administered as an enema, and, if necessary, some slight aperient by the mouth. The bowels having been moved, the patient is allowed chicken soup, fish, eggs, beef-tea, etc. If the child is to be nursed, it may be put to the breast on the second or third day.

The abdominal sutures may be removed in from ten to fourteen days, and the patient allowed to rise at the end of four weeks. She should always wear an abdominal belt, and should be warned against kneeling when scrubbing floors, as this is apt to induce hernia from pressure and stretching of the cicatrix.

In review, it may be explained that rupture of the membranes, either intentionally or by labour, means a contraction of the uterine wall, and as a consequence a greater wounding of the uterine tissue in order to secure a sufficient opening to extract the child. Some operators, instead of using manual or pessary compression to prevent bleeding when opening the uterus, make use of an elastic ligature. The uterus is first everted, and the elastic ligature then passed round the cervix. This not only necessitates a much larger abdominal incision, but also induces asphyxia of the fœtus, and causes inertia of the uterus, as the organ does not so readily respond to kneading. Its use is, therefore, conducive to hæmorrhage. Veit, Doleris, and Pajot have blamed it for causing death from hæmorrhage, and Zweifel, Sänger, and Lusk have also noticed this complication.

Caruso advised the early removal of the ligature.

Another way of dealing with the uterine incision is by Sanger's method. In this procedure the muscular wall of the uterus is closed with from ten to fifteen sutures which approximate to, but do not include, the mucosa; and between each suture two superficial sutures are inserted to unite peritoneum to peritoneum. Previously the peritoneum was separated from the muscularis, and a wedge-shaped piece of muscularis removed from each side, the base of the wedge being outermost. This done, the peritoneal flaps were folded into the wound and secured by the superficial stitches. Such a detailed process is quite unnecessary, as the sutures, as recommended by Cameron, secure perfect apposition, not only of the muscular tissue, but also of the peritoneum. In fact, most operators now make use only of eight or ten deep sutures, and reserve superficial sutures to secure contact where there is any gaping between the stitches. Such unevenness can be readily avoided by beginning in the middle and working towards each end, and by taking care to keep the sutures at regular intervals.

Porro's Modification.—The fatal results following the early Cæsarean section led to a modification of the operation. It had been found by experiment that the uterus in pregnant rabbits could be removed with better results

than by simple section, and therefore it was concluded that similar results would follow in the case of women.

Blundell, in writing upon this subject, said such a method might prove an eminent and valuable improvement; but he also wrote, in speaking of deaths from peritonitis after Cæsarean section, that experience sometimes contradicts our most favourite opinions, and that something of the kind would be found to occur in the cases under consideration, as he had no doubt the risk of diffused peritonitis had been greatly exaggerated. How his surmise has proved true is seen in the present-day position of abdominal surgery.

Acting on the lines suggested, Storer of Boston, in 1868, first practised amputation of the uterus after section. The case was one of pregnancy complicated with a fibroid of the uterus. He was interrupted by such an alarming hæmorrhage that he had to remove the body and fundus with the ovaries; but his patient died three days afterwards. This was an operation of necessity.

Porro first performed the operation as a matter of choice, as he considered it impossible to secure the uterine incision in Cæsarean section, so as to prevent the flow of blood and septic fluid into the peritoneal cavity. The results got under antiseptics in other abdominal operations encouraged him to make the attempt, and in 1876 he did so with happy results. Others took up the operation, and very quickly the old Cæsarean section was superseded by it; but only for a few years, as now Cæsarean section can be performed without the slightest danger from bleeding, peritonitis, septicæmia, or other dangers that Porro's operation sought to avert.

At the present day Porro's operation is an operation of exception, that is only necessary in some conditions, such as serious rupture of the uterus, or where labour is obstructed by a large fibroid. As regards the steps of the operation, it is to begin with similar to Cæsarean section. It is only after the uterus has been emptied that it varies, as at this point the uterus is everted and an elastic ligature applied round it, just above the os internum.

The uterine tissues are then compressed until the bleeding has ceased. The uterus is then removed, and the stump secured outside the abdominal wound, and maintained in position by needles and a serrenœud.

Porro, upon emptying the uterus, transfixed it with a trocar and cannula at the union of the body and cervix. He then withdrew the trocar, and passed two silver wires through the cannula, which was also withdrawn and the wires tied, one upon the right and the other upon the left side, including in their grasp the ovaries and tubes. This done, the uterus and appendages above the wires were cut away, whilst the stump was secured outside. The method has been improved by transfixing with needles and ligating with a serrenœud instead of with separate wires.

The stump is dusted with iodoform and dressed with gauze all round. The needles should be raised to allow of proper packing. A layer of sublimated Gamgee should be placed over all. It may require to be dressed daily; and the ligated portion usually separates about the tenth day, but the raw cavity requires regular dressing till perfectly healed.

It was urged as an important factor that Porro's operation prevented future conceptions; but this end is gained in Cæsarean section by the more simple method of tying and dividing the tubes.

**La Bourboule.**—A mineral water containing iron and arsenious acid.

**Labridæ.**—A family of fish, dangerous for eating. *See* SNAKE-BITES AND POISONOUS FISHES.

**Labrum.**—The upper lip in mammals, as opposed to *labium*, the under lip.

**Labyrinth.** *See* AUDITORY NERVE AND LABYRINTH (*Physiology, Diagnosis, Diseases, etc.*); EAR, ACUTE INFLAMMATION OF MIDDLE; EAR, MIDDLE, CHRONIC NON-SUPPURATIVE; NOSE, POST-NASAL ADENOID GROWTHS (*Labyrinthine Deafness*); SYPHILIS, TERTIARY (*Labyrinthine Disease*); VERTIGO (*Aural, Ménière's Disease*).

**Labyrinthitis.**—Inflammation of the labyrinth. *See* AUDITORY NERVE AND LABYRINTH (*Labyrinthitis*); DEAF-MUTISM (*Morbid Anatomy*).

**Lac.**—(1) A resinous incrustation produced in the case of certain trees by the puncture of an insect (*Coccus lacca*); it is astringent, but is now little used in medicine; according to the state in which it is offered for sale it is called stick-lac, seed-lac, or shell-lac (*Shellac*). (2) milk or a milky fluid, *e.g.* lac sulphuris or milk of sulphur.

**Lace-Maker's Disease.** — Lead poisoning.

**Lacerated Wounds.** *See* MEDICINE, FORENSIC (*Kinds of Wounds, Lacerated*); WOUNDS.

**Lacerations.** *See* LABOUR, INJURIES DURING (*Cervix, Perineum, Uterus, etc.*); VAGINA, DISORDERS OF (*Injuries*).

**Lachrymal.** *See* LACRIMAL.

**Lacing-Liver.**—The deformity of the liver produced by tight-lacing, consisting in the downward prolongation of the right lobe so as to constitute practically a new lobe or tongue, and in the presence of a marked transverse groove. *See* LIVER, DISEASES OF (*Anatomical Abnormalities*); PREGNANCY, MANAGEMENT (*Clothing*).

**Laconicum.** *See* BALNEOLOGY (*Historical, Hot-air Bath*).

## Lacrimal Apparatus, Diseases of.


ANATOMY AND PHYSIOLOGY . . . . 306
DISEASES OF THE GLAND . . . . 307
*Inflammation* . . . . . 307
*New Growths* . . . . . 308
DISEASES OF THE EXCRETORY APPARATUS . 308
*Epiphora* . . . . . 308
*Foreign Bodies* . . . . . 309
*Calculi* . . . . . 309
*Polypi* . . . . . 309
*Wound, Abscess in* . . . . . 309
DISEASES OF LACRIMAL SAC AND NASAL DUCT . . . . . 309
*Inflammation* . . . . . 309
*Stenosis of Duct* . . . . . 311


*See also* ALCOHOLISM (*Chronic, Visceral Variations, Lachrymal*); CONJUNCTIVA, DISEASES OF (*Conjunctivitis, Complications*); ELECTRICITY (*in stricture of the lachrymal duct*); FIFTH NERVE, AFFECTIONS OF; MUMPS (*Affections of Eye*); NERVES, NEURALGIA (*Symptoms, Lachrymation*); SYPHILIS (*Tertiary, Lachrymal Apparatus*).

THE lacrimal apparatus comprises the tear gland, with its excretory ducts, the puncta, the canaliculi, the tear sac, and the nasal duct. The lacrimal gland and its ducts constitute the secretory portion, the other structures named the excretory portion of the lacrimal apparatus, and our subject will be discussed under these two headings.

Diseases of the lacrimal apparatus are about thrice as common in women as in men, perhaps in consequence of the more zealous use that the former make of the function of lacrimation.

ANATOMY AND PHYSIOLOGY. — The *lacrimal gland* is a compound tubulo-racemose gland, resembling the serous salivary glands; it is about the size and shape of an almond, measuring rather more from before backwards than from side to side, but its size varies if one may judge from measurements of accredited observers. It consists of two portions: the larger, called the superior lacrimal gland, lies in a depression in the roof of the orbit, just within the upper and outer orbital margin. This portion is in contact by its upper convex surface with the periosteum of the orbital roof, to which it is attached by fibrous bands; the anterior edge corresponds with, but does not project beyond the margin of the orbit; the posterior border reaches to the junction of the first and second fourth of the roof of the orbit; the lower concave surface is in apposition with the superior and external recti muscles. The lower portion, also called the palpebral portion or accessory gland, separated by tendinous strands from the main gland, is less constant in size and shape, and is sometimes absent; it consists of one or two small lobules, which lie just beneath the mucous membrane of the superior conjunctival fornix, and may sometimes be brought into view by eversion of the lid and strong, downward rotation of the eyeball. The efferent ducts from both portions of the gland, some eight to twelve in number, open by a row of apertures into the conjunctival sac at the upper and outer part.

The glands of Krause, similar in structure and formation to the lacrimal gland, are small, rounded bodies, situated chiefly in the upper, but also met with in the inferior cul-de-sac, and suffice to moisten the eye even if the lacrimal gland is destroyed. The lacrimal gland in its connection with the conjunctival sac may be well compared to the salivary glands and the cavity of the mouth. The secretory nerve of the lacrimal gland is generally reckoned to be the lacrimal branch of the fifth, but Goldzieher and Jendrassich have recently declared that it belongs to the facial nerve. According to Kirchstein the gland is very small and rudimentary in the new-born child, which accounts for the absence of tears at that period of life.

Under usual conditions the tears are secreted only in sufficient quantity to moisten the conjunctiva and cornea, the greasy edges of the eyelids also preventing overflow; the surplus is disposed of by evaporation and by escaping into the nose through the naso-lacrimal canal. An overflow takes place by direct irritation of the lacrimal nerve, by reflex irritation of the conjunctiva or nasal mucous membrane, by strong light acting on the retina, or by painful emotion. The tears escaping from the gland are at once dispersed over the surface of the eye, just like fluids between a cover-glass and microscopical slide, aided by winking movements of the eyelids.

The *puncta lacrimalia* are two pin-point apertures situated near the posterior edge of the eyelids about 5 to 6 mm. from its nasal commissure. They are not quite opposite one another, the lower one being about 1 mm. farther from the commissure than the upper. They lie against the conjunctiva of the bulb, so that they are visible only when one causes slight eversion of the eyelids. The upper canaliculus runs vertically upwards from its punctum for a distance of 2 mm., then makes a sudden bend inwards and downwards; the lower canaliculus runs vertically downwards for a still shorter distance, and then takes a horizontal direction. It is of practical importance to bear this in mind when probing or slitting the canaliculi.

The canaliculi enter the tear sac separately, as a rule, but sometimes by a common duct; the openings are situated at the outer side of the sac, but also on its anterior aspect. The canaliculi are lined by squamous epithelium, differing, as we shall see, from the lining of the sac and nasal duct.

The lacrimal sac and its continuation, the

nasal duct, which opens into the nose beneath the inferior turbinate bone, in structure, character of secretion, and pathological relations, is to be properly regarded as an accessory part of the nose rather than of the eye.

Both sac and duct are formed of a fibro-elastic material, with a well-developed mucous membrane lined by an imperfectly ciliated columnar epithelium. The lower part of its duct has numerous glands similar to those in the meatus of the nose. The sac lies in a deep groove formed by the superior maxilla and ethmoid bone; the bony canal lodging the nasal duct is completed by the inferior turbinate bone.

The internal palpebral ligament, which can be felt as a hard cord running inwards from the nasal commissure of the lids, is the best guide to the sac; an abscess in the latter always points just below this ligament, but the blind, dilated end of the sac reaches a little distance above it.

The direction of the naso-lacrimal passage is downwards, backwards, and a little outwards, which it is of importance to recollect when passing a probe. The diameter of the sac is 5 to 6 mm., that of the nasal duct 3 to 4 mm., and the narrowest part is usually at the junction of the sac and duct.

An empyema of the frontal sinus may burst into the sac, and an empyema of the maxillary sinus into the nasal duct.

Diseases of the Gland.—The lacrimal gland possesses almost complete immunity in inflammatory or other affections of the eye; even in gonorrhœal and other severe conjunctival inflammations the gland escapes. The accessory part, however, does occasionally become enlarged in phlyctenular ophthalmia, acute trachoma, panophthalmitis, and some other conditions, and is recognised as a small, very soft swelling at the upper and outer part of the superior lid, even when one cannot make it out by touch.

Diseases, and still more injuries of the main mass of the gland are of rare occurrence, from its protected position and its multiple system of ducts. Stabs of the upper lid may reach and wound the gland, and if healing is delayed, and especially if suppuration occurs, a fistulous opening may be left from which tears escape.

True lacrimal fistula has also been met with as a congenital condition, and is recognised as an opening in the upper lid so minute as to be easily overlooked, the tears exuding in very small quantities, and evaporating almost as soon as they escape. A cure is best effected by passing a needle armed with a silk suture through the opening in the skin, and bringing it out in the upper and outer part of the conjunctival sac; the other end of the suture hanging from the opening in the skin is then threaded on another needle, passed through the lid near the fistula, and brought out in the conjunctival sac near the other. The two ends are tied together and allowed to cut their way through. The effect of this is to make the tears find their way into the conjunctival sac instead of through the opening in the skin, which now closes of itself, or will do so if the edges be freshened and brought together by a suture.

*Dislocation of the gland* is very rare, but has been met with as the direct result of a blow, from a cicatrising wound of the upper lid, and still more rarely as a spontaneous condition. The presence in the outer and upper part of the eyelid of a movable subcutaneous lobulated swelling about the size of an almond, renders the diagnosis easy. Replacement is sometimes possible and should be tried, for it is occasionally followed by cure; but excision through an incision in the skin is mostly required.

Chalky concretions, called *dacryoliths*, are sometimes found blocking one or more of the excretory ducts, and must be removed through the conjunctiva as they give rise to a good deal of pain.

*Inflammation of the lacrimal gland* occurs in an acute and in a chronic form, the former leading to suppuration, the latter to hypertrophy.

The acute form is almost always confined to one side, and is usually met with in delicate children as the result of a blow or from exposure to cold.

It is ushered in by a general febrile condition; there is a feeling of tension, and sometimes very severe pain, shooting to the brow, temple, and even to the upper jaw. The lymphatic glands of the neck are enlarged, sometimes to such an extent as to cause cyanosis from pressure on the jugular vein. There is a painful, tender, dusky red swelling of the outer part of the upper lid, which hangs down and covers the cornea. The upper lid is greatly thickened and enlarged, causing obliteration of the natural folds; the veins are engorged and tortuous, and even some of the arteries may be seen and felt to pulsate.

The ocular conjunctiva shows all grades of inflammation, even to most severe serous chemosis protruding from between the lids.

The globe is somewhat protruded and displaced, not directly forwards, but towards the lower and nasal side, and its movements are always restricted upwards and outwards, although they may be unimpaired in other directions. In tenonitis the displacement is straightforward, and the movements of the globe are restricted in all directions. The gland itself cannot be felt (compare with the chronic form), on account of the swollen and infiltrated condition of the lid and tissues underneath, and probably also because the gland is already in a state of suppuration, and forms no distant tumour.

Hot fomentations followed by an incision when fluctuation occurs, or even before we can be certain of this, is the best course; it is useless to try abortive treatment in acute cases. It will be noticed that the escape of pus is

followed by clear fluid, the tears, which are characteristic of this affection. The probe sometimes comes upon exposed bone, which has led some observers to regard these cases, not as suppurative adenitis, but as a localised inflammation of the bone, a view which I cannot subscribe to. If the abscess is allowed to burst of itself the pus makes its way through the skin or into the conjunctival sac, and in the former case a fistula may result, and require operation as before described.

*Chronic adenitis* is met with as the result of syphilis, tubercle, leukæmia, and as an accompaniment of mumps. It may affect both orbits. The first symptom noticed by the patient is ptosis or drooping of the upper lid, followed in a day or two by protrusion and displacement of the eyeball with consequent diplopia. A blunt, often lobulated and elongated swelling is felt under the outer part of the brow. Sometimes, but not always, there is dryness of the eye from want of the lacrimal secretion, and this may exceptionally be so marked that desiccation of the corneal epithelium may take place. Dryness of the mouth has also been observed from a coincident affection of the salivary glands, which may or may not be enlarged.

Numbness of the brow on the same side may be present, from pressure on the frontal branch of the first division of the fifth nerve; this soon disappears, but the power of lifting the lid and rotating the eye upwards persists, the sensory nerve recovering from the effects of the pressure sooner than the motor nerves, which is in accordance with observations of the kind elsewhere.

The treatment consists in inunction of mercurial ointment over the swelling and the brow and temple, with iodide of potassium internally in syphilitic cases, cod-liver oil with creasote in strumous cases, or arsenic, in the form of Fowler's solution, when neither syphilis nor tubercle is present. If the indurated swelling does not disappear we must excise it by an operation, which will be later described.

Various new growths occur in the gland, and as the symptoms are similar to those of chronic adenitis we are led to suspect the presence of some growth if the swelling does not disappear or diminish under treatment, or still more if it increases in size. It is often impossible before excising and examining this swelling to say if it is due to simple hypertrophy or new growth, or whether it is benign or malignant in nature. In children we meet with sarcoma; in adults with carcinoma, cylindrina, chloroma, also with gumma, hydatid cyst, enchondroma, and some other rarer varieties of growth.

*Excision of the gland* for hypertrophy or new growth is done in the following manner:—The patient is anæsthetised, the brow is shaved, and the parts thoroughly cleansed. A curved incision is made down to the periosteum along the outer third of the brow, and, if necessary, it may be prolonged for some distance beyond the external commissure. The edges of the wound are kept apart by hooks, and the tarso-orbital fascia is divided, exposing the gland, which is drawn forward by a hook and removed in its capsule if possible. The fascia is united to the periosteum by a catgut suture, a drain is inserted, and the skin wound closed with silk sutures.

With strict antiseptic precautions the operation is usually free from risk, but a fatal case has been recorded.

The operation very often leads to permanent cure even when done for growths, as they are often completely encapsuled, and the changes are confined to the centre of the gland, where cystic spaces often occur.

It is much more often successful than in growths from any other part of the orbit.

A rare form of cystic growth called *dacryops*, due to obstruction of one or more of the efferent ducts of the gland, was first described in 1814 by A. Schmidt. It appears as a bluish, thin-walled, translucent cyst with fluid contents, which springs into view beneath the upper lid. In some of the cases, from imperfect closure of duct, its contents, which become much more tense on crying, may escape gradually or be from time to time pressed out by the patient. A well-recorded case has been described by Arnold Lawson in vol. xvii. *Trans. Ophth. Soc.*, and an interesting paper by Hulke in vol. i. *Royal London Ophth. Hosp. Reports* should be read. An attempt should be made to excise the cyst entire, as was successfully done in Lawson's case; but even if only a large piece of the wall is removed, a cure is often effected.

### Diseases of the Excretory Apparatus

Diseases under this heading are much more common than diseases of the lacrimal gland, and are hence of more practical importance. They are met with more often in adults than in children, but some are congenital in origin. Heredity from the mother's side plays an important part in their causation.

*Puncta and Canaliculi.*—A constant symptom in all diseases of the excretory apparatus is *epiphora* or overflow of tears down the cheek, and when the puncta and canaliculi are the parts involved, this may be the only symptom. Epiphora is always aggravated by cold wind, dust, or smoke, and necessitates the constant use of the pocket-handkerchief. Although weeping is always present in diseases of the lacrimal passages, its presence does not always prove the existence of anything amiss with those parts, for it may be caused by inflammation of the conjunctiva, cornea, or iris; and some people, quite free from ophthalmic disease, are liable to a temporary epiphora on coming into the fresh air. It is frequently associated with morbid

blushing, and also occurs in the early stage of Graves' disease. Practically, however, in all cases coming under treatment it is the drainage apparatus that is at fault. Epiphora is by no means always due to stricture of the canaliculi, the slightest displacement of the puncta, particularly the lower, which is functionally the more important, will produce it; and hence we have it in ectropion of the eyelids, especially of the lower, also in entropion, where the inturned lashes also set up irritation and excessive secretion of tears. Epiphora may also be due to the presence of a Meibomian cyst or other tumour of the inner part of the lower lid, and is seen in old people from relaxation of the lid and in paralysis of the facial nerve—conditions all inimical to the proper approximation of eyelid and globe.

In old-standing neglected blepharitis the edge of the lid becomes rounded and slightly everted, the secretion of the Meibomian glands is diminished, and the tears readily overflow. The tears, containing as they do a large proportion of salt, set up irritation and inflammation of the skin, accentuating the ectropion and giving rise to constant blepharospasm and discomfort.

The treatment consists in slitting up the lower canaliculus by means of a Weber's probe-pointed knife, thus converting the little tunnel into an open rill, which must be prevented from closing by passing a probe along it for some days after the operation. This operation will be mentioned more fully later on.

The same procedure is to be adopted when the epiphora is due, not to displacement, but to stenosis or abnormal narrowing of the canaliculi or puncta.

In these cases the puncta become so minute as to require the aid of a magnifying lens to make them out; and we must first dilate the passage by a conical sound so as to admit the beak of the Weber's knife.

Small *foreign bodies*, such as an eyelash, the wing of an insect, a wheat bristle, etc., may be carried by the flow of tears from the conjunctival sac into the canaliculi, almost always the lower, and protruding from the punctum, will rub against the eye and give rise to some pain, irritation, and overflow of tears. The treatment consists in the removal of the foreign body by means of forceps.

*Calculi* composed of carbonate of lime and leptothrix threads sometimes block the canaliculus, a condition recognised by the presence of a little fusiform swelling, and requiring slitting up of the passage for its removal.

*Polypi* in this position are still rarer, and, if luxuriant, may protrude from the punctum.

In cases of *wound* of the lower lid dividing the canaliculus, the permeability of the passage must be ensured by slitting up both distal and proximal parts before suturing the edges of the wound. *Congenital absence* of one or both puncta is occasionally met with; but is not usually associated with epiphora, probably on account of a compensatory imperfection or absence of the lacrimal gland. An accessory punctum and caniculus may very rarely occur, mostly in the lower lid. It may end blindly or open into the tear sac, or it may open into the canaliculus, in which case it may be regarded as a congenital fistula of that passage.

### Diseases of the Lacrimal Sac and Nasal Duct

The mucous membrane of the sac is subject, like the conjunctiva, to catarrhal and purulent inflammation, the starting-point being almost always in the nose and not in the conjunctiva. It is astonishing but no less true that purulent conjunctivitis practically never extends to the lacrimal sac; perhaps the thick layer of pavement epithelium lining the canaliculi to some extent accounts for this. In trachoma, however, the lining membrane of the sac has in some instances been found affected with a like disease, the two regions being probably simultaneously attacked. In thirty-eight cases of disease of the sac, Gruhn, in all but two, found nasal disease as simple chronic coryza, atrophic or hypertrophic rhinitis with or without ozæna, etc., with obvious signs of the nasal disease being of much older standing than that of the tear sac. Michel thinks that the lacrimation which snuffers of tobacco suffer from or (!) enjoy, is due not only to reflex stimulation of the lacrimal gland, but also to the chronic inflammation of the nasal mucous membrane extending up the nasal duct and narrowing it. There is no doubt whatever that if one takes the trouble to make inquiries in cases of lacrimal trouble, one will get a history of repeated colds in the head, if not direct evidence of intra-nasal disease.

Malformation of the nose, in the form of flat-nose and deviation of the septum, is a predisposing cause in many cases.

The secretion in the sac, especially if purulent, contains the staphylococcus pyogenes aureus and albus and streptococcus pyogenes. The bacilli of tubercle may also be present, and should be looked for as an aid to diagnosis.

Vegetations in the nasal fossa may block the end of the duct, as may also syphilitic or tubercular ulcers. Lupus of the nasal cavity is a very common cause of stoppage of the canal.

*Catarrh of the sac* comes on very insidiously, and at first gives rise only to epiphora most marked in the morning or in cold wind.

This symptom soon becomes constant, and is associated with ciliary blepharitis, redness, swelling, and discharge from the caruncle and adjacent conjunctiva, a condition called conjunctivitis angularis or lacrimalis, which should always lead us to suspect inflammation in the sac, especially if only in one eye.

The diagnosis is made certain by gently opening the lids with the fingers of one hand, while pressure is made on the sac with the tip of a finger of the other hand. Catarrh of the sac is made known by the escape from one or both puncta of a few drops or a tiny stream of turbid fluid.

In time the sac becomes distended, forming a characteristic rounded swelling at the corner of the eye, and the patient very soon learns the trick of emptying this by pressure. In some cases pressure empties the contents, not into the conjunctival sac, but into the nose.

This condition of slight distension of the sac may remain unchanged for an indefinite time if the patient regularly empties it and keeps the eye clean; but from a fresh coryza stagnation and decomposition of the contents takes place, giving rise to *purulent cystitis*.

Here the symptoms are for the most part similar to, but more intense than in the catarrhal form. The eyelids in the morning are glued together by profuse muco-purulent secretion, the lashes are covered by crusts, the conjunctiva is swollen, red, and even chemotic. The expressed contents of the sac are markedly purulent, and there is a painful feeling of distension in the region of the sac.

A third stage called *phlegmonous dacryocystitis* may supervene from extension of the inflammation to the parts outside the sac. The onset is rapid, and accompanied by general febrile symptoms and intense pain, the patient in some cases becoming almost maniacal.

The neighbouring soft parts are infiltrated so that the limitations of the sac can no longer be made out, but are merged in the general swelling, which is hard, brawny, and shining.

The puncta are with difficulty brought into view, and the ocular conjunctiva is chemotic. The condition might be mistaken for erysipelas, but the extreme tenderness just over the sac and the history of a preceding lacrimal discharge ought to prevent such a mistake.

The pain is so excessive that, as a rule, we are not enabled to help our diagnosis by pressing out pus from the puncta as in the catarrhal stage.

The abscess points just below the tendo-oculi in most cases; but it sometimes burrows under the orbicular muscle, and escapes at some distance below the lower eyelid. The symptoms rapidly abate on the escape of the pus, and the swelling subsides so that the limits of the distended sac are again made out. A fistulous opening is very apt to be left. In some few cases the pus escapes from the puncta, and it has been known to make its escape by perforation of the lacrimal bone; but this is exceedingly rare.

The swelling and infiltration of the mucous membrane is alone sufficient to cause retention of tears and pus, and actual stricture is by no means usually present—in fact it is distinctly exceptional, and when present is to be regarded, as in stricture of the urethra, not as the cause, but as the effect of the inflammation in the canal. Stenosis can be diagnosed only after surgical treatment by probing, and we will have more to say on this subject under the heading of Treatment.

Disease of the bone forming the walls of the canal is often met with in syphilitic and tubercular cases; it is probably always the cause and not the effect of the purulent cystitis, but rough treatment in probing may give rise to it.

In some cases of long-standing obstruction the sac becomes greatly distended, forming a tumour almost the size of the first joint of the thumb. The overlying skin is much attenuated, semi-translucent, and bluish, and the condition might be mistaken for a varix. The swelling is incompressible, and it is usually not possible to empty it either into the nose or into the conjunctival sac. This condition is called a *mucocele* or *hydrops*, and is due to distension of the sac with glairy fluid.

Dermoid tumours, although, of course, almost situated at the outer part of the brow, do sometimes occur in the region of the sac, and might then be mistaken for mucocele, as has occurred more than once in my own experience.

A mucocele of the frontal sinus, with or without infiltration of the anterior ethmoidal cells, might be confounded with a hydrops of the sac; but the former condition soon gives rise to proptosis and lateral displacement of the eye with consequent diplopia, which is never the case with dilatation of the sac, however great.

Of late years a good deal has been written concerning the so-called *congenital blennorrhœa* of the sac, which is really due to retention of the normal mucus from a membranous obstruction at the lower end of the nasal duct, a condition which Vlacovich found four times in the examination of fourteen bodies of new-born infants. This affection is nearly always confined to one eye, and the characteristic symptom noticed by the parents a day or two after birth is the presence within the lower eyelid of a quantity of glairy mucus. If the secretion be gently wiped away and pressure then made on the sac, the source of the discharge can be proved by the escape of some from the puncta. It is only seldom that any distension of the sac can be made out. The secretion may rarely, in older standing cases, become slightly purulent. I have known the condition to be mistaken for ophthalmia neonatorum by those not overburdened with ophthalmic knowledge.

It has been noted by Heddäus, and confirmed by others, that the secretion, like all physiological secretions, is in abeyance during sleep, so that if the eye be cleansed last thing at

night, it will be free from the mucus for some hours after the child awakes in the morning.

These cases, unlike real dacryocystitis in later life, show a tendency to become cured rapidly and spontaneously from giving way of the membranous obstruction. It is therefore advisable at first to content ourselves with keeping the eye clean by frequent use of a soft rag, and making pressure over the region of the sac. If a cure is not soon effected by these means, the case must be treated as an ordinary dacryocysto-blennorrhœa.

*Treatment.*—The best and most rapid cures are obtained in cases which are not accompanied by strictures, disease of the bone, or great distension of the sac; but even in the presence of these complications good results may be obtained by patience.

As soon as the diagnosis is established by the mucous or muco-purulent discharge from the puncta, we may make up our mind that an operation is required. Delay can do no good. Purulent or even phlegmonous inflammation may supervene at any time, and any slight abrasion or ulcer of the cornea may give rise to hypopyon keratitis and loss of the eye. If any surgical operation on the eye, especially cataract extraction, be contemplated, it is, of course, absolutely necessary to cure any lacrimal trouble first, or we should be practically certain to lose the eye from suppuration — an unfortunate result not infrequently due to overlooking a slight discharge from the sac.

We have to do with a catarrhal or purulent process taking place in what is practically a closed sac, and our aim is to get free access to the cavity, empty its contents, prevent their reaccumulation, and bring the mucous lining into a healthy condition. Entrance into the sac used to be gained by an incision in its anterior wall, but since Bowman's time this is done by his method of splitting the canaliculus and freely incising all the tissues at the neck of the sac. This operation is the only great advance that has been made in the treatment of lacrimal disease since quite ancient times. The patient is laid on his back, and a few drops of a 4 per cent solution of cocaine having been repeatedly instilled into the corner of his eye, the skin of the lower lid is kept on the stretch by means of outward traction with the thumb of the left hand, and (the right eye is here supposed to be under operation) the surgeon standing behind the patient's head inserts the beak of the Weber's knife vertically into the lower punctum, turns the cutting edge upwards and a little backwards, and, keeping the handle almost parallel to the lid margin, thrusts it slowly and steadily inwards till the probe point impinges on the lacrimal bone. By raising the handle like a lever, while still keeping the point against the lacrimal bone, the canaliculus may be slit along its entire length. The knife should now be felt to lie quite freely and movable in the sac; but if this is not the case, slight sawing movements will bring it about. In operating on the left eye the surgeon, standing as before, keeps the lid on the stretch with the thumb of his right hand, and uses the knife with his left; or if he finds it easier, he may stand on the patient's left side, facing him, and so get the advantage of using the knife with his right hand. Some prefer to have the patient seated on a chair with the head, covered by a towel, resting on the chest of the operator who stands behind.

The surgeon now passes a probe about 2-3 mm. in diameter along the divided canaliculus till it is arrested by the lacrimal bone, and raising it to a vertical position against the brow, thrusts it steadily downwards, backwards, and a little outwards till it is arrested on the floor of the nose. To do this satisfactorily requires some confidence, which comes by practice. If the upper end of the probe, after being pushed down, stands forwards away from the brow, we have probably made a false passage, which is more likely to take place in using the smaller probes; hence it is a good rule to use as large a probe as will easily pass. I have known the probe to make its appearance in rather unexpected places—for instance, in the cavity of the mouth behind the soft palate; and on another occasion, from too vigorous use, it has gone through the roof of the mouth. Some skilled surgeons pass the knife itself down into the nose, which certainly ensures an easy passage for the largest probe; but this had better not be done by those of little experience, as with such a fragile instrument the blade is apt to break off and be left in the nasal duct, and false passages are more easily made than by the probe. A more than usually prominent brow renders the use of a straight probe difficult or impossible, and generally the curved probes of Couper are the best to use.

It will generally be found that $1\frac{1}{2}$ to $1\frac{3}{4}$ inches of the probe are concealed when thrust home. Its lower end may often be felt by means of a second probe passed along the floor of the nose for a distance of $1\frac{1}{8}$ inch from the posterior edge of the nostril. This cannot be done in every case, as the opening of the duct is often on the outer wall, and not in the roof of the inferior nasal fossa, and is then protected by a flap-like arrangement of the mucous membrane.

The probe is passed twice a week till the discharge ceases, or at least loses its purulent appearance; but we must be prepared for relapses in many cases.

Strictures are diagnosed and localised best by the olive-pointed probes of Couper, and occur most frequently at the junction of the sac and nasal duct, but also at the nasal end of the duct and also at the neck of the sac.

Great patience both on the part of the patient and the surgeons is called for. If a small probe can be passed, we may hope for amelioration by the use of gradual dilatation; but if the stricture is osseous in nature, it is probably incurable. The use of a dental drill has been recommended in such cases.

In phlegmon of the sac we must first make a deep vertical incision over the sac, and, after escape of the pus, lightly pack the cavity with iodoform gauze, which is renewed each day till the swelling of the tissues has been dispersed, when Bowman's operation is then done. The opening in the skin will gradually close if probes are passed by the slit canaliculus. In disease of the bone the pus forms burrows in various directions, and these must be freely laid open from end to end and well scraped with a sharp spoon. Injection of iodoform emulsion into the sac may also be done. In strumous and syphilitic cases the appropriate constitutional treatment must not be neglected.

In very obstinate cases, where, in spite of probing, the discharge remains purulent and very profuse, injections by means of a hollow perforated probe and syphon arrangement is good practice. For this purpose we may use 0·6 per cent sulphate of zinc, 1 per cent acetate of lead, 0·02 per cent corrosive sublimate, 2 to 5 per cent nitrate of silver, or, best of all, 10 per cent protargol. If this does not suffice to dry up the discharge, Fick recommends the injection into the sac of a few drops of a 10 per cent solution of chloride of zinc, first protecting the cornea by a thick layer of vaseline. Severe reaction takes place, but a good result is obtained.

In cases of chronic distension of the sac, and in cases of incurable stricture, the best thing to do is probably to destroy the sac. This operation, I am informed by my colleague Dr. Little, used to be frequently done by Mr. T. Windsor and himself with good and permanent results, and why we should have discarded it during the last twenty-five years he cannot say. An incision was made commencing below the tendo-oculi at about 4 mm. from the inner commissure. To lay open the whole length of the sac, as is necessary, we prolong the incision upwards to include the fundus. The bleeding was stopped and the cavity stuffed daily for a few days, when a strong paste containing 20 per cent zinc chloride was introduced on strips of lint, the final result being a firm and by no means unsightly scar. Excision of the sac is a very difficult operation, and is more often commenced than completed, resolving itself into a rather haphazard cutting and scraping away of the tissues.

**Lacrimation or Lachrymation.**—A profuse flow of tears. *See* LACRIMAL APPARATUS, DISEASES OF.

**Lacs.**—The obstetrical fillet, formerly much used in the management of delayed labour cases (Fr. *lacs*, a string).

**Lactagol.**—The powdered extract of the seed of the cotton plant. It has been used to increase the milk secretion in nursing women, for it is said to contain the galactagogue principle of the plant; it is insoluble in water. *Dose*—1 to 2 oz. daily.

**Lactalbumin.**—One of the proteids of milk, the other being casein.

**Lactams.**—*Lactones* are produced from certain hydroxyacids by loss of water (*e.g.* santonin); "aromatic compounds containing $NH_2$ in the ortho position, and losing water by the oxidation and removal of one or two atoms of that hydrogen, furnish bodies which may be distinguished as *lactams* and *lactims*" (*Attfield and Dobbin*).

**Lactase.**—A zymin splitting up milk-sugar into glucose and galactose, and found in the intestine of animals taking milk.

**Lactate.**—A salt of lactic acid.

**Lactation.** *See* BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Occlusion of Cerebral Vessels, Arterial Occlusion, Causes*); INFANT FEEDING; INSANITY, ETIOLOGY OF (*Direct Causes, Lactation*); LYMPHATIC SYSTEM (*Enlargement of Axillary Glands*); MILK; PUERPERIUM, PHYSIOLOGY (*Lactation*); PUERPERIUM, PATHOLOGY (*Conditions interfering with Suckling*); TYPHOID FEVER (*Association with other Conditions, Lactation*); UTERUS, INFLAMMATIONS OF (*Superinvolution, Causes*).

**Lactational Insanity.** *See* PUERPERIUM, PATHOLOGY (*Insanity of Lactation*).

**Lacteals.**—As a noun the *lacteals* are the chyliferous vessels; as an adjective *lacteal* simply means milky or consisting of milk (the *lacteal secretion* is milk). *See* ASCITES (*Characters of Fluid, Chylous Fluid*); INFANT FEEDING; etc.

**Lactic Acid.**—The acid (hydroxypropionic) which is formed when milk turns sour ($C_3H_6O_3$), by the conversion of milk-sugar (lactose) under the influence of the bacillus acidi lactici; it has been used as a solvent for diphtheritic false membranes and in tuberculous disease of the larynx, and somewhat extensively in the form of lactophosphates. *See also* CALCIUM AND ITS SALTS; DIGESTION AND METABOLISM (*Gastric Contents*); MICRO-ORGANISMS (*Lactic Fermentation*); RICKETS (*Etiology and Pathology*); TEETH (*Dental Caries, Lactic Acid in Saliva*).

**Lactifuge.**—A drug stopping or diminishing the secretion of milk; an antigalactagogue.

**Lactometer.** *See* MILK (*Examination, Lactometers*).

**Lactophenin.**—An analgesic and anodyne, produced by the action of lactic acid on phenetidin. *See* ANALGESICS AND ANODYNES (*Anilin Series*).

**Lactophosphates.** *See* CALCIUM AND ITS SALTS (*Calcii Phosphas*).

**Lactoscope.**—An instrument for determining the amount of fat in milk; the distance at which a candle flame can be seen through the milk is ascertained, and its opacity (and, consequently, the amount of fat contained in it) is thus discovered.

**Lactose.**—Milk-sugar, a disaccharide composed of a molecule of dextrose united to a molecule of galactose with dehydration; $C_{12}H_{22}O_{11} + H_2O$. *See* INVALID FEEDING (*Milk and its Products*); PANCREAS, PHYSIOLOGY OF; PHYSIOLOGY, FOOD AND DIGESTION (*Carbohydrates*); PUERPERIUM, PHYSIOLOGY (*Excretory System*).

**Lactoserum.**—Whey. *See* MILK.

**Lactoserve.**—A white powder obtained from pasteurised milk which has been subjected to a certain degree of lactic acid fermentation, evaporated to dryness, and mixed with sugar, flour, and vegetable albumin; it is used in the form of an emulsion—in dyspepsia, in atrophic states, and in constipation in children, under similar circumstances to those in which buttermilk has been employed (*Merck*).

**Lactosomatose.**—A food consisting of desiccated milk and somatose.

**Lactosuria.**—The presence of milk-sugar in the urine, especially in the case of nursing women. *See* DIABETES MELLITUS (*Varieties*); GLYCOSURIA (*Puerperal Lactosuria*); URINE, PATHOLOGICAL CHANGES IN (*Sugars in Urine*).

**Lactuca.**—The lettuce; the common garden lettuce (*lactuca sativa*) yields a concrete juice, the *lactucarium* of the United States Pharmacopœia, which may be used as the *syrupus lactucarii* or the *extractum lactucarii fluidum*; it contains a bitter principle *lactucin* ($C_{22}H_{18}O_7$), a crystalline body *lactucerin*, and *lactucic acid* ($C_{80}H_{64}O_6$).

**Lacuna.**—A small cavity, crypt, or space, *e.g.* the lacunæ of bone or *Howship's lacunæ*, *Marie's lacunæ* (small areas of softening in the grey matter of the brain), *Morgagni's lacunæ* (recesses in the mucous membrane of the male urethra), and the *lacunæ of de Graaf* (Graafian follicles of the ovary).

**Læhme.**—The disease known as "foot-halt" of lambs; a form of rickets met with in the young of animals kept in captivity, and regarded as due to deficiency of lime salts in the food.

**Lænnec.** *See* MEDICINE, HISTORY OF.

**Lævulose or Levulose.**—A sugar which rotates the plane of polarised light to the left instead of to the right (*dextrose*), but in other particulars behaves as dextrose. *See* INULIN; PANCREAS, PHYSIOLOGY OF; PHYSIOLOGY, FOOD AND DIGESTION (*Carbohydrates*).

**Lævulosuria.**—The presence of lævulose in the urine.

**Lagneia or Lagnesis.**—Excessive venereal appetite (Gr. λαγνος, lustful); satyriasis, nymphomania, lagneia furor, or lagnosis.

**Lagophthalmos.**—The state of incomplete closure of the eyelids during sleep, due to various conditions, such as ectropion or shortness of the lids, paralysis or paresis of the orbicularis muscle, and large size of the eyeball; the state of being "hare-eyed" (Gr. λαγώς, a hare, and ὀφθαλμός, the eye), because hares were supposed to be unable completely to close their eyes. *See* EYELIDS, AFFECTIONS OF (*Defects in Position*).

**Lagostoma.**—Hare-lip.

**La Grippe.** *See* INFUENZA.

**Lahs' Theory.** *See* LABOUR, MECHANISM (*Flexion*).—The theory that flexion of the fœtal head is due to its wedge shape, the steeper or occipital end of the wedge descending first.

**Laking of Blood.**—The effect produced upon blood when the red corpuscles are acted on by some solvent (*e.g.* ether, acids, etc.), and the hæmoglobin diffused.

**Lalling.**—Imperfect pronunciation or "baby-talk," especially the articulation of *r* as if it were *l*.

**Laloo.**—The name of an individual who had a small fœtus attached to the anterior aspect of his thorax; an instance of the double monstrosity known as thoraco-parasitus.

**Lalopathy.**—Disordered speech, or, sometimes, aphasia.

**Laloplegia.**—Speech-paralysis from any cause.

**Lamalou.** *See* BALNEOLOGY (*France, Chalybeate Waters*).

**Lambdacism.**—An abnormal state of pronunciation, in which there is difficulty in the articulation of *l* or lambda.

**Lambdoidal.**—Shaped like the Greek *l* (lambda, λ), *e.g.* the lambdoidal suture between the supra-occiput and the margins of the parietal bones contiguous to it.

**Lamblia Intestinalis.**—A protozoan parasite, furnished with flagellæ, which infests the small intestine of man and of many of the lower animals; it was described by Lambl. *See* Parasites (*Protozoa*).

**Lamella.**—A thin plate, especially of bone, but also of nerve substance and of embryonic tissues.

**Lamellæ.**—Thin discs made of gelatin and glycerin, used as a means of applying certain drugs (atropin, cocaine, etc.) to the conjunctival surface, the disc being dropped into the eye; there are four official lamellæ in the British Pharmacopœia, those of atropin, of cocaine, of homatropin, and of physostigmine. *See* Prescribing.

**Lameness, Intermittent.**—A malady due probably to arterio-sclerosis; intermittent claudication; angiosclerotic dysbasia (*Erb*); crural angina (*Walton*).

**Lamina.**—A thin layer or lamella.

**Laminaria Tents.**—Dried sea-tangle (*laminaria digitata*) has been used in the form of tents for the dilatation of the cervix uteri. *See* Gynæcology, Diagnosis in (*Cervical Dilators*).

**Laminectomy.**—The surgical procedure by which one or several of the spinal laminæ are removed to relieve pressure on the spinal cord in cases of fracture or fracture-dislocation of the spine, or of spinal caries. *See* Spine, Surgical Affections of (*Fractures, Spinal Caries*).

**Lancet.**—A lance-shaped knife, thin with a double edge; used for opening abscesses, making incisions, scarifying the gums (when a special form is employed), and for performing venesection.

**Lancinating.**—Darting, shooting, or cutting, used specially in reference to the pains accompanying certain disorders of the nervous system. *See* Tabes Dorsalis.

**Landeck.** *See* Balneology (*Germany, Thermal Waters*).

**Landouzy-Déjerine's Myopathy.**—The facio-scapulo-humeral type of idiopathic muscular atrophy. *See* Muscles, Diseases of (*Idiopathic Muscular Atrophy or Hypertrophy*).

**Landry's Paralysis.**—Acute ascending paralysis. *See* Paralysis (*Flaccid Paralysis, Landry's Paralysis*).

**Langenbeck's Operation.** *See* Palate (*Cleft Palate, Operation*).

**Langerhans' Cells or Islets.**—A layer of epithelial cells lying internal to the glandular epithelium proper of the pancreas; centro-acinar cells. *See* Pancreas, Physiology of; Physiology, Food and Digestion (*Pancreas*); Physiology, Internal Secretions (*Pancreas*).

**Langhans' Layer.**—A layer of polyhedral cells lying under the syncytium covering the villi of the chorion of the young placenta; similar cells are found in the chorionepithelioma or deciduoma malignum. *See* Pregnancy, Ovum and Decidua (*Epithelioma of the Chorion*); Puerperium, Pathology (*Sarcomo-Deciduo-Cellulare or Deciduoma Malignum*).

**Language.** *See* Physiology, Respiration (*Voice*).

**Lanoform.**—An antiseptic ointment; lanoline containing formaldehyde.

**Lanoline.**—*Adeps Lanæ Hydrosus* of the B.P., Hydrous wool fat. A fat derived from the wool of the common sheep. It is very stable, difficult of saponification, and never becomes rancid; it possesses remarkable penetrating powers when applied to the skin, and is therefore employed as a basis for ointments containing mercury or other drug which we wish to be quickly absorbed. Its disagreeable stickiness is avoided by the admixture of vaseline.

**Lanugo.**—The hair or down developed upon the fœtus *in utero* and shed to a large extent before birth.

**Laparo-.**—In compound words *laparo-* (Gr. λαπάρα, flank) means relating to the abdominal walls, *e.g. laparohysterectomy*, the removal of the uterus by abdominal section; *laparocolotomy*, abdominal colotomy; etc.

**Laparocele.**—Lumbar hernia.

**Laparoelytrotomy.**—An operation formerly used as a substitute for Cæsarean section; the abdominal wall is divided by an incision over Poupart's ligament, the parts are dissected up till the roof of the vagina is reached, it is opened into, the cervix uteri is dilated, and the child extracted through it.

**Laparomyomectomy.**—The removal of a fibroid tumour of the uterus by abdominal section.

**Laparonephrectomy.**—Nephrectomy carried out by means of an abdominal incision.

**Laparotomy.**—Abdominal section, cœliotomy or celiotomy. *See* Abdomen, Injuries of (*Penetrating Wounds, Treatment*); Appendix Vermiformis (*Appendicitis, Operation*); Bladder, Injuries and Diseases of (*Rupture, Treatment*); Intestines, Surgical Affections of (*Obstruc-*

*tion, Treatment*); LABOUR, INJURIES TO THE GENERATIVE ORGANS (*Rupture of Uterus, Treatment*); LABOUR, OPERATIONS (*Cæsarean Section*); OVARIES, DISEASES OF (*Ovarian Cyst, Ovariotomy*); PELVIS, HÆMATOCELE AND HÆMATOMA (*Treatment*); PERITONEUM, ACUTE PERITONITIS (*Treatment*); PERITONEUM, TUBERCULOUS PERITONITIS (*Treatment*); PUERPERIUM, PATHOLOGY (*Peritonitis, Treatment*); STOMACH AND DUODENUM, DISEASES OF (*Cancer of Stomach, Treatment*); TYPHOID FEVER (*Perforation of Intestine, Treatment*); UTERUS, NON-MALIGNANT TUMOURS OF (*Abdominal Operations for Fibroids*); UTERUS, MALIGNANT TUMOURS OF (*Hysterectomy, Abdominal*).

**Lapis.**—A stone, especially in such combinations as *lapis divinus* (sulphate of copper, 3 oz.; nitrate of potash, 3 oz.; alum, 3 oz.; and camphor, 60 grs.), *lapis lunaris* (nitrate of silver), and *lapis causticus* (caustic potash).

**Lappa.**—The root of the burdock (*Arctium lappa*), official in the U.S. Pharmacopœia, has a diuretic, diaphoretic, and alterative action, and is used in syphilis, scorbutus, and scrofula.

**Lard.** *See* ADEPS.

**Lardacein.** *See* LARDACEOUS DEGENERATION.

## Lardaceous Degeneration.


DEFINITION . . . . . 315
GENERAL CHARACTERS . . . . . 315
ETIOLOGY . . . . . 315
CHEMISTRY . . . . . 316
PATHOLOGY . . . . . 316
CLINICAL CHARACTERS . . . . . 317
DIAGNOSIS . . . . . 318
PROGNOSIS . . . . . 318
TREATMENT . . . . . 319


SYNONYM: *Amyloid, Waxy, or Albuminoid degeneration.*

*See also* HEART, AFFECTIONS OF MYOCARDIUM AND ENDOCARDIUM (*Morbid Processes, Lardaceous Infiltration*); LIVER, DISEASES OF (*Lardaceous Liver*); NEPHRITIS (*Etiology*).

DEFINITION.—Amyloid degeneration is a peculiar change, affecting certain organs, by which the whole or certain parts of them are converted into a structureless homogeneous substance called amyloid or lardacein.

The organs most frequently affected are the liver, spleen, kidneys, and intestines; but lymphatic glands, the stomach and alimentary canal, the bladder, prostate, generative organs, serous membranes and muscles may at times undergo this change. The same substance also occurs locally in tumours, thrombi, and scars, especially those resulting from syphilis.

GENERAL CHARACTERS.—Organs affected with amyloid degeneration are generally pale in colour, firm in consistency, and much enlarged: the capsule appears stretched and tense, and the edges are somewhat rounded, though the general shape of the organ is maintained. On section, the cut surface presents a peculiar smooth glistening appearance, owing to which the name "lardaceous" was applied to this condition. The organ is bloodless in consequence of the pressure exercised by the newly-formed material upon the blood-vessels of the part, and the diminution in their calibre produced by the amyloid change in their walls; and to the same cause is due the fatty change in the active cells of the organs, which generally accompanies advanced stages of the process. Microscopically, in the early stages, the affection is frequently limited to the subendothelial layer of the intima and to the middle coat of the smaller blood-vessels, the arterioles and capillaries being affected before the veins; later, it spreads to the connective tissue of the organ. Owing to pressure and diminished blood-supply, the essential cells of the organ may be found to have largely disappeared by fatty degeneration and absorption.

Amyloid substance may be recognised, when invisible to the naked eye, by its peculiar staining reactions. If on the freshly-cut, washed surface there is poured a solution of iodine, the amyloid substance at once takes on a rich brown (mahogany) colour, while the unaffected tissue is only stained a faint yellow. The same staining may be used for microscopic sections, but fades rapidly, and is therefore useless for permanent preparations. Further, if to sections thus coloured there be added a 10 per cent solution of sulphuric acid, a peculiar greenish colour is produced in the diseased portions. The best stain for microscopic purposes is afforded by methyl or gentian violet. In sections stained with these dyes and subsequently treated for a few minutes with weak acetic acid, the amyloid substance is stained a bright magenta colour, the surrounding tissue appearing blue. These colour-reactions are not absolutely constant—that with iodine and sulphuric acid being apparently seen only in very advanced amyloid degeneration, and the simple iodine-staining sometimes failing to appear in tissues which have been long preserved. The violet reaction is the most constant and reliable.

ETIOLOGY. — Amyloid disease is stated to occur more frequently in males than females, and originates almost invariably in persons below the age of thirty. It occasionally seems to occur as a primary disease, but many of such cases are probably due to causes of which all trace has disappeared.

In the great majority of instances it is induced by long-continued suppuration, such as that existing in connection with chronic bone-disease, or tuberculosis of lungs, joints, or kidneys. It is also found in tertiary syphilis, and rarely in the cachexia of malignant disease

or malaria. It can be produced experimentally in animals by inducing and maintaining suppuration by means of cultures of the Staphylococcus pyogenes aureus. Some observers have stated that the same result follows on suppuration produced by the Bacillus pyocyaneus, or even by turpentine; but these results have not been confirmed.

Chemical Nature.—Amyloid substance presents a marked resistance to the action of the gastric juice; by the action of this ferment it may be obtained practically pure. It has been shown by Krawkow to consist of an organic acid (chondroitin-sulphuric acid) combined with some form of albumen. This latter portion of the compound is probably not constant in composition, and it is possible thus to account for the varying behaviour of different specimens in respect of staining. Amyloid substance is very closely allied to "hyaline," which is considered by some authorities to be either identical with it, or, at least, its forerunner. A substance giving the same reactions as amyloid is found in the coats of the aorta and arteries under normal conditions, so that the pathological product appears to have a physiological prototype.

Pathology. — Since amyloid degeneration secondary to suppuration is limited almost entirely to cases presenting ill-drained cavities and sinuses, it seems probable that it is the result of the absorption of some poisonous product formed by the bacteria to which the suppuration is due. This theory is confirmed by the results of the experiments on animals quoted above. Possibly the toxin injuriously affects the metabolism of the cells—the digestion by the cells of the circulating albumens of the lymphs—and thus are formed unusual derivations of albumen, which are deposited in or around the cells. Authorities appear to incline towards calling amyloid change an "infiltration" rather than a "degeneration" proper, but it is doubtful whether any rigid distinction between these two processes is maintainable. Some recent observers have endeavoured to trace a connection between amyloid substance and hæmoglobin. Thus Petrone attributes the degeneration to soaking of the tissues with dissolved blood-pigment, many corpuscles being broken up in the course of wasting diseases such as syphilis, tuberculosis, or chronic suppuration; while Obrzut considers that the masses of amyloid occurring in the spleen are composed of conglomerated hæmocytes which have undergone a peculiar transformation. It seems difficult, on either of these hypotheses, to account for the great swelling of the affected organs and the amount of pressure apparently exercised by the new product upon the surrounding cells.

### Morbid Anatomy

Amyloid Liver.—The liver is much enlarged and may fill the greater part of the abdominal cavity. It is pale in colour, and the surface is smooth and regular, the normal shape of the organ being preserved. The specific gravity of the tissue is increased, and it is firm and resistant in consistency. On section, little blood is found in the organ; the cut surface is smooth and glistening, of a greyish-red or dirty yellow colour. In early stages the outlines of the individual lobules may be distinguishable, but later on all trace of structure is lost. If a portion is stained with iodine a very characteristic appearance is produced, each lobule being marked out as a thick, dark brown ring, with a pale centre, separated by pale substance from neighbouring rings. This appearance is due to the fact that the amyloid change occurs principally in the middle zone of each lobule, in the area of distribution of the hepatic artery. The periphery of the lobule is the seat of fatty degeneration.

Amyloid Spleen.—Two varieties of the degeneration in this organ are distinguished:—(1) The change may be limited to the Malpighian corpuscles, which present the appearance of grains of boiled sago set in the substance of the organ, from which the condition is known as *sago spleen*. (2) The connective tissue forming the trabeculæ may be affected throughout, the cells also appearing to undergo degeneration, while the Malpighian corpuscles escape. This is known as the *diffused* form. A *mixed* form is also described, in which both of the above changes occur simultaneously. The organ is enlarged, pale, firm, and heavy, the greatest enlargement occurring in the diffuse and mixed varieties.

Alimentary Canal.—Here the change appears late, and never occurs without the simultaneous affection of other organs. The intestine is little altered in appearance to the naked eye, but may look unduly pale and translucent, and feel thickened and rigid. On pouring a solution of iodine on to the mucous surface, the whole appears stippled with closely-set brown dots, which correspond to the villi, the central arteries of which are the main seat of the change. Owing to increased rigidity thus produced the villi are liable to be broken off, and ulcers may be formed. Very intractable diarrhœa results from this degeneration, owing probably to exudation of increased quantity of serous fluid through the degenerated vessels. Absorption may also be interfered with, and nutrition correspondingly impaired. If the stomach is the seat of amyloid disease, obstinate vomiting may be the result.

Amyloid Kidney.—The appearance of the kidney when subject to amyloid change varies with the extent of the lesion and with the amount of the inflammatory process which often accompanies it. In the earliest stage the kidney may look practically normal, and only reveal on treatment with iodine a few scattered brown

points or streaks showing the existence of the disease. Later on the organ becomes large, the cortex being pale and anæmic, the pyramids somewhat dark in colour. The capsule still strips easily, and the cut surface is smooth and glistening. On addition of iodine the Malpighian bodies show up as brown dots in the swollen cortex, and the arteries are mapped out as brown lines. Small yellowish streaks of fatty degeneration are usually present. Still later, in cases complicated by much nephritis, there may be shrinking of the newly-formed fibrous tissue: cysts may be found in the cortex of the organ, and the capsule may become adherent. The naked-eye appearance resembles very closely the ordinary large and small white kidneys of chronic tubal nephritis. Microscopically, it is found that the change starts in the glomeruli and spreads to the middle coat of the afferent arteries and arteriæ rectæ, finally involving also the connective tissue throughout the organ. The epithelium of the tubes does not undergo amyloid change, but is frequently found in a condition of cloudy swelling and degeneration, owing to accompanying nephritis. In chronic cases interstitial inflammation may cause increased formation of fibrous tissue, and the tubules may become blocked and give rise to small cysts. The exact relation of the nephritis to the amyloid disease is not known, but it seems probable that the existence of the degeneration and the pressure exerted by the new material diminish the resistance of the essential cells of the kidney, and predispose them to attack by irritant substances, one of which may even be the same toxin which gives rise to amyloid degeneration. It is noteworthy that when amyloid disease of the kidneys is secondary to suppuration occurring in one of these organs, both are equally affected by the degenerative process.

*Effects.*—It is impossible to separate the effects produced by amyloid disease of the liver and spleen from those of the primary cause of the degeneration. In the case of the kidney and intestine, however, definite symptoms are produced, disease of the former leading to albuminuria and even dropsy and uræmia (see *infra*), that of the latter to a severe and intractible form of diarrhœa, which invariably terminates fatally.

*Clinical Characters.*—Patients suffering from lardaceous disease due to syphilis or occurring as a "primary" condition may not, at first, present any very marked degree of wasting; but in cases due to suppuration emaciation is profound, and all the symptoms of hectic fever are usually present. There is marked anæmia in all cases. The abdomen is enlarged owing to the increase in size of the liver and spleen, and occasionally to accompanying ascites; but, apart from cases with general dropsy, coexisting cirrhosis, or perihepatitis, it is probable that this symptom only occurs when there are enlarged glands pressing on the portal vein, or in the rare instances in which the radicles of this vein are affected by the degeneration. The organs are generally painless and not tender to the touch, but in some cases both pain and tenderness may be found. The edges of both liver and spleen may be easily felt through the thin abdominal wall, smooth, firm, and regular, reaching often below the umbilicus, and sometimes separated by no very clear dividing line. The urine is increased in quantity and contains albumen in varying but always considerable amounts; and diarrhœa, due to implication of the intestine, appears towards the end. Experience of cases that have occurred in Charing Cross Hospital shows that the liver is the organ usually first recognised as affected, and that it may attain a large size before the spleen is palpable. The kidney suffers next to the spleen, and the intestine only in very advanced stages. In some cases, however, especially those due to syphilis or to primary renal tuberculosis, the kidney may be, from the outset, most markedly affected, amyloid change in the other organs being only detected after death.

The course of the disease is almost invariably progressive, owing to the difficulty of treating the primary cause. Cases, however, in which the suppuration can be stopped, may recover from the amyloid degeneration; and this has been shown to occur also in animals. Amyloid disease is a comparatively rare affection at the present day, owing to the introduction of antiseptic methods in surgery, and the consequent diminution in the number of cases of suppuration. Children suffering from hip-disease are probably its most frequent victims, but, apart from suppuration, it is a rare affection at this period of life.

The clinical symptoms of amyloid kidney must be considered in detail, since the degeneration may affect this organ primarily, and give rise to phenomena liable to be attributed to ordinary nephritis. In cases due to continued suppuration the liver and spleen generally suffer first. After a time the urine begins to increase in quantity and becomes pale in colour, the density being correspondingly diminished; later on it becomes albuminous, the percentage of albumen at first being small. As a rule the disease of the kidney is not, in such cases, of any vital importance. In syphilitic cases, on the other hand, or in such as are apparently primary, it is often only in advanced stages that the sufferers come for treatment. In addition to the pallor and malnutrition of the patient, the urine is found loaded with albumen, pale in colour, and often neutral or alkaline in reaction. The quantity, at this stage, may be normal or even diminished, but is more often increased to

seventy or eighty ounces, rising in some cases to as many as two hundred ounces per diem. Hyaline and granular casts are found, and may stain brown with iodine. Ziegler denies that they are really amyloid. The salts of the urine are diminished in amount, the percentage of urea falling along with the rest, but not, as a rule, to a dangerous extent. This feature is probably due to accompanying nephritis and not directly to the degenerative change. The amount of albumen is very variable, and the cause of its presence has been differently explained. Thus some maintain that it is proportionate to the extent of the amyloid change; others (Lecorché) affirm that its presence is a proof of nephritis. In some few cases it may be entirely absent (Litten). Paraglobulin is often present in large amount, and may exceed the quantity of serum-albumen. Intercurrent attacks of nephritis occur in which hæmaturia may be prominent, and the urine scanty, dark, and of high density; it may even be actually suppressed. Dropsy may be well marked, and uræmic convulsions or coma may supervene in rare instances. No alteration in the vascular tension usually occurs in cases of amyloid kidney, and the heart is of normal or reduced size. Failure of the heart may occur, leading to diminished urinary flow, and in advanced cases to actual death.

Diagnosis.—In patients suffering from chronic suppuration no difficulty is found in recognising the onset of amyloid disease. The gradual enlargement of liver and spleen, the increasing albuminuria and polyuria, and the final onset of uncontrollable diarrhœa, present a striking clinical picture. On the other hand, in cases where the suppuration has ceased, and in those due to syphilis or some undiscovered cause, considerable doubt may exist. If a patient presents a uniform enlargement of both liver and spleen, there may arise, in adults, suspicion of cirrhosis of the liver or of leuchæmia; in children, of congenital syphilis, or of mediastinitis with adherent pericardium and strangulation of the inferior vena cava. In alcoholic cirrhosis the history of the case may be an aid to diagnosis, and the appearance of the patient is seldom suggestive of lardaceous disease, the tendency being to venous stigmata and congestion of the face rather than pallor. Diarrhœa may occur in either condition, but is more intermittent in cirrhosis, and ascites is far more likely to occur in the latter disease. This last symptom is also prominent in adherent pericardium, in which, however, pleural thickening or effusion is frequently present, while the area of cardiac dulness is markedly increased. Leuchæmia will be excluded by an examination of the blood, though the increase of leucocytes may be temporarily absent. It must be remembered that some degree of leucocytosis may occur in suppurative cases; but here the leucocytes will be found all to belong to the multinucleated variety, no increase occurring in the lymphocytes or in the large uninucleated cells. Syphilitic enlargement of liver and spleen in children may be indistinguishable from amyloid disease, but the latter does not often occur at this age as a manifestation of syphilis. A history may be obtained of some suppurating lesion if such has existed, and will point to probable amyloid degeneration. If only the liver or the spleen is enlarged, diagnosis may be very difficult or even impossible. The history of the case will be the greatest aid, and the possibility of amyloid change must be constantly borne in mind. The existence of scars, pointing to past syphilitic lesions, or to old sinuses and disease of bone, is often of importance. Signs of congenital syphilis may be found in keratitis, in scars at the corners of the mouth, in malformation of the teeth, and in deafness due to bilateral otitis media. A case is recorded (Affleck) in which a misshapen amyloid left lobe of the liver exactly resembled a splenic tumour, and no certain diagnosis was possible during life between amyloid disease and splenic anæmia. In this case there was no history of syphilis nor of any other recognised cause of amyloid degeneration. In cases commencing in the kidney, diagnosis from ordinary tubal nephritis is often difficult—indeed, the two conditions may actually be coexistent. Signs of amyloid disease elsewhere may be found, in slight enlargement of the liver and spleen, which might escape notice if not carefully sought. Any signs of syphilis will be very suggestive of degeneration. Past residence in the Tropics is also to be looked upon with suspicion, as malaria and dysentery are possible factors in the production of amyloid. The condition of the heart and arteries is an important diagnostic feature, since in nephritis sufficiently advanced to cause the amount of albuminuria met with in amyloid disease the pulse will almost certainly be of high tension, and the left ventricle of the heart enlarged so as to cause the apex-beat to be displaced. In uncomplicated amyloid disease neither of these features is found. Heart-failure in renal disease may, however, somewhat mask the extent of the vascular change, while on the other hand it must be borne in mind that in children under six years of age the apex-beat may be normally near the nipple-line. If casts which stain brown with iodine are found, they are probably diagnostic of amyloid change, but the occurrence is too rare to afford much aid. The diarrhœa occurring in phthisical patients from amyloid disease is distinguishable from that due to ulceration of the bowel only by concurrent signs of the degeneration in other organs, and, perhaps, by its even more intractible character.

Prognosis.—This depends, in most cases, on the chances of removing the cause of the disease. If suppuration can be checked before the patient is too exhausted, there is good ground for hoping

that the amyloid disease will spontaneously disappear, the degenerative product being absorbed, and the cells of the organ resuming their activity. That such recovery may occur in animals has been proved by an experiment of Lubarsch, who produced the condition artificially, and demonstrated the presence of amyloid in an excised portion of spleen. The animal was then allowed to recover from the suppuration which had caused the condition, and when it was subsequently killed all trace of amyloid substance had disappeared. In renal cases, uncomplicated by nephritis, the outlook is best in those due to syphilis, and in cases in which there is not very much enlargement of liver and spleen. In patients with pulmonary tuberculosis, the onset of amyloid disease is always an ominous sign. If the cause cannot be removed, no definite limit of time can be fixed for the duration of life. The condition may last from one to ten years, according to circumstances. In cases of continued suppuration, it is unlikely that the patient will survive more than two years from the appearance of marked symptoms of amyloid change.

TREATMENT. — It seems probable that no therapeutic measures avail to act directly on the amyloid deposit. Efforts should therefore be directed to removing the cause of the degeneration and improving the general health of the patient. Cases due to suppuration should be submitted to rigorous surgical treatment, foci of infection being as far as possible removed by operation; sequestra should be sought and extracted, cavities scraped out, and the freest possible drainage provided. In obstinate cases of empyema, resection of portions of ribs may be necessary to allow the cavity to close permanently. By such means astonishingly good results may at times be attained. The patient should be placed in the most favourable circumstances possible; fresh air is essential, that of a bracing seaside climate being the best of all.

The digestive organs must be kept in good order, and plenty of nourishing food provided. In cases with much nephritis it may be advisable to exercise some caution with regard to the amount of meat allowed, and some care is necessary in selecting a suitable climate. Such cases derive most benefit from residence in a mild winter climate. The most liberal diet that can be digested is generally permissible. Cod-liver oil, either alone or combined with one of the preparations of malt, is of service; and tonics, such as iron and quinine, are useful adjuvants.

In syphilitic cases the general treatment may be the same, but iodide of potassium must be given in sufficient doses, rising to thirty grains three times a day for adults. The treatment must be continued at intervals for several (two to five) years. Very good results are often obtainable in these cases, even when the syphilitic infection is of old standing. Mercury is not of much service, and must be given cautiously in cases presenting signs of renal disturbance.

If the cause cannot be removed or directly treated, as is the case in phthisical patients, it remains only to relieve symptoms. Dropsy may be treated on ordinary lines by rest in bed and diuretic medicine, especially digitalis. Iron is also useful in such cases, the iodide being a useful salt to employ. Diarrhœa is little amenable to any treatment, and is a warning of approaching death. Opium or morphia may be tried, combined, if necessary, with sulphuric acid or with sulphate of copper. Astringents, such as catechu or krameria, may occasionally appear to afford temporary benefit.

**Largin.**—A compound of silver and protalbin, used, like protargol, in gonorrhœa and gastro-intestinal ulcers (dose, 5 to 8 grs.); it contains 11 per cent of silver, and has a bactericidal action. *See* CONJUNCTIVA, DISEASES OF (*Ophthalmia Neonatorum, Treatment*).

**Larva.**—An insect in the grub stage, or the immature form of an animal which reaches its mature form by a process of metamorphosis; various larvæ are the causes of myiasis. *See* MALARIA (*Mosquitoes*); MYIASIS; NOSE, FOREIGN BODIES, ETC. (*Parasites of the Nose*).

**Larval or Larvated.** — A masked, latent, or incompletely developed condition, *e.g.* a disease, such as scarlet fever or epilepsy in a masked form; also used of a skin disease which covers the face as with a mask.

**Larva Migrans.**—Dermamyiasis linearis migrans œstrosa. *See* CREEPING ERUPTION.

**Laryngeal Crises.** *See* TABES DORSALIS (*Symptomatology, Affections of Vago-Accessorius*).

**Laryngeal Injections.** *See* BRONCHI, BRONCHIECTASIS (*Treatment, Intra-laryngeal Injections*).

**Laryngeal Obstruction.** *See* DIPHTHERIA (*Diagnosis*); LARYNX, CONGENITAL LARYNGEAL STRIDOR.

**Laryngeal Paralysis.** *See* AORTA, THORACIC, ANEURYSM OF (*Symptoms, Laryngeal Paralysis*); LARYNX, NEUROSES OF (*Paralysis of the Vocal Cords*); TYPHOID FEVER (*Complications and Sequelæ, Paralysis of Vocal Cords*).

**Laryngeal Phthisis.** *See* LARYNX, CHRONIC INFECTIVE DISEASES (*Phthisis*).

**Laryngeal Spasm.** *See* ASTHMA; LARYNX, LARYNGISMUS STRIDULUS.

**Laryngectomy.**—Excision or extirpation of the larynx. *See* LARYNX, MALIGNANT DISEASE (*Treatment, Operative*).

**Laryngismus Stridulus.**—A spasmodic affection of the larynx, characterised by arrested respiration, cyanosis of the face, and followed by noisy inspiration. *See* Asphyxia; Asthma; Bronchi, Bronchial Glands (*Diagnosis*); Bronchi, Bronchitis (*Diagnosis*); Convulsions, Infantile (*Etiology, Rickets*); Larynx, Laryngismus Stridulus; Nose, Post-nasal Adenoid Growths; Rickets (*Clinical Features*); Tetany; Thymus Gland (*Status Lymphaticus*).

**Laryngitis.** *See* Larynx, Acute and Chronic Inflammations; *see also* Burns and Scalds (*Clinical Features, Laryngitis and Œdema Glottidis*); Cough (*Clinical Varieties, Diseases of Larynx*); Diphtheria (*Diagnosis*); Measles (*Complications, Laryngeal Catarrh*); Nose, Diseases of Nasal Orifices and Septum (*Benign Tumours of Nasal Fossæ*).

**Laryngo-Fissure.** — Thyrotomy or splitting of the thyroid cartilage. *See* Larynx, Affections of the Cartilages (*Stenosis, Treatment*).

**Laryngoscopy.** *See* Larynx, Examination of (*Laryngoscopy*).

**Larynx.**—This subject is treated in the following sections:—

1. **Examination of.**
2. **Acute and Chronic Inflammations. Injuries, Foreign Bodies, etc.**
3. **Chronic Infective Diseases.**
4. **Neoplasms**—(*a*) **Simple.** (*b*) **Malignant.**
5. **Neuroses. Hypertrophy of Lingual Tonsil.**
6. **Affections of the Cartilages. Stenosis of the Larynx.**
7. **Laryngeal Stridor, Congenital.**
8. **Laryngismus Stridulus.**

*See also* Aorta, Thoracic, Aneurysm of (*Symptoms, Laryngeal Paralysis*); Asphyxia; Asthma; Brain, Physiology of (*Functions of Cerebral Cortex, Motor Areas, Movements of Larynx*); Breath (*Clinical Diagnosis, Odour, Ulceration of Larynx*); Bronchi, Bronchial Glands (*Diagnosis*); Bronchi, Bronchiectasis (*Treatment, Intra-laryngeal Injections*); Bronchi, Bronchitis (*Diagnosis*); Convulsions, Infantile (*Etiology, Rickets*); Diphtheria (*Diagnosis*); Gout (*Irregular Gout, Respiratory System, Laryngeal Catarrh*); Intubation; Lung, Tuberculosis of (*Complications*); Nose, Post-nasal Adenoids; Physiology, Respiration (*Voice*); Rickets (*Clinical Features, Laryngismus Stridulus*); Syphilis (*Visceral Syphilis, Trachea and Lungs*); Tetany; Thymus Gland (*Status Lymphaticus*); Trachea, Affections of (*Treatment, Tracheotomy*); Typhoid Fever (*Complications and Sequelæ, Paralysis of Vocal Cords*).

**Examination of Larynx**


1. Laryngoscopy . . . . . 320
2. Direct Inspection of Larynx . . 322
3. Transillumination . . . . . 322
4. Skiagraphy of Larynx . . . 323
5. Palpation . . . . . 323
6. Examination of Trachea . . . 323


1. *Laryngoscopy.* — The apparatus required for examining the larynx consists of a laryngeal mirror, a forehead reflector, and a good light. As every senior student nowadays is familiar with the laryngoscopic mirror and reflector, it is no longer necessary to give a detailed description of them. But some practical directions as to how to use them, and what to observe, may be of service:—

The patient should sit erect on a stool or chair with his head slightly inclined backward. The source of light, whether oil, gas, or electric lamp, should be placed at his right or left shoulder, and on a level with his ear. The observer, seating himself in front of the patient, places the reflecting mirror over his eye nearest to the lamp, so as to screen it from the light, and looks through the hole in the reflector while keeping both eyes open. The source of light, the patient's mouth, and the eye of the observer should be on one level. It is well before proceeding further to make a careful inspection of the mouth, fauces, and pharynx by means of a tongue-depressor and the reflected light. Observe if there be any general anæmia or congestion of the parts, and note the presence of any swelling, ulceration, cicatrix, or membranous deposit, also any dryness or excess of secretion, and the condition of the tonsils and uvula. The observations made at this stage will often help us in the diagnosis of the laryngeal condition, and the practice which it gives in using the reflector so as to obtain the best illumination —a great difficulty with beginners—will prove of real service in the later stages of the examination. Having completed the examination of the pharynx, we direct the patient to open the mouth widely and push his tongue well forward. With the aid of a tongue-cloth the tip of the tongue should be grasped firmly, but gently, between the thumb and forefinger of the left hand and held out, but not pulled upon or dragged down on the lower teeth. The laryngeal mirror should then be held over the lamp till a slight film of moisture forms on its surface and passes off, and its temperature tested by applying it to the back of the hand. Next throw a disc of light on to the fauces, so that its centre corresponds with the base of the uvula; and holding the laryngeal mirror on the right hand lightly like a pen, and with its reflecting surface downwards, pass it quickly to the back of the mouth, taking care not to touch the tongue or palate in doing so. The patient, meanwhile, should breathe deeply but

quietly through the mouth, so as to raise the palate and uvula away from the tongue. The back of the mirror being placed against the uvula, the whole palate should be raised upwards and backwards by a steady pressure. If the patient's throat is at all tolerant, the lower edge of the mirror may be allowed to rest on the back wall of the pharynx; but very often this will produce retching, and prevent an examination.

When the mirror is in position it should be held steadily, while the handle is carried to the left angle of the mouth, so as to be out of the line of vision, and by very slightly raising or depressing the hand, so as to alter the inclination of the mirror, the base of the tongue, epiglottis, and more or less of the laryngeal cavity should come into view. If the epiglottis is erect, we may at once get a view of the whole larynx and trachea. As a rule, however, it will be necessary to make the patient sing "eh" or "ee" in a slight falsetto, so as to raise the epiglottis and expose the larynx.

The examination of the larynx should be conducted systematically, beginning with the vocal cords, which will at once strike the eye by their pearly white colour and their movement on phonation and inspiration. Note in addition to any change in colour, any thickening or irregularity of their edges, any breach of surface, or the presence of clumps or strands of mucus. During phonation observe if the cords come together in their whole length, with their edges tense and sharply defined. Be sure you see right to the anterior commissure where the cords meet, and follow them backwards to the vocal processes, which show as yellow spots, or are sometimes pinkish in colour in voice-users. Observe their position on quiet and deep inspiration, and see if the amount of abduction is normal and equal on the two sides. Special attention should be given to the posterior wall of the larynx, the inter-arytenoid space, which is so frequently the seat of pathological changes. It is best seen during deep inspiration. Look out for any thickening, irregularity of surface, or mucous crusts, conditions which interfere with the approximation of the arytenoids. Just above each cord notice the dark line running parallel to it, the opening to the ventricle, and immediately above that the rounded fold of mucous membrane, sharply defined below and sloping away above into the aryepiglottic folds, the ventricular band. Compare the two sides carefully and note any swelling, new growth, or ulceration.

The aryepiglottic folds should then be inspected, following them downwards and backwards from the sides of the epiglottis to the arytenoid cartilages. Notice their delicate sharp edges above, especially on phonation, and the nodular thickenings corresponding to the cartilages of Wrisberg, and the small capitula Santorini seated on the arytenoid cartilages. Observe the symmetry of the two sides, look out for any swelling or œdema, and watch the movements of the arytenoid cartilages on phonation and inspiration, carefully comparing the freedom of movement of the two sides. The epiglottis should then be examined. It will be found to vary greatly in shape and position in different individuals. In some it is broad and bent forwards towards the tongue, so as to show its posterior surface only; in others it is narrow, folded laterally, and inclined back over the larynx, so that only the anterior surface is seen in the mirror. The anterior surface is yellowish pink in colour, and has large veins coursing over it; the edge is more distinctly yellow, and the posterior surface is pinkish red, deepening in colour towards the prominence at its base, the cushion of the epiglottis. The edge of the epiglottis should be thin and sharply defined. Notice if any thickening, œdema, or loss of substance of its edge is present, or any ulceration of the posterior surface. Next examine the base of the tongue, between the circumvallate papillæ and the epiglottis, observing the amount of adenoid tissue present (lingual tonsil), and the numerous large, superficial veins, and conclude with a survey of the lower pharynx and of the pyriform sinuses which lie outside the larynx between the aryepiglottic folds and the inner surface of the thyroid cartilage.

The colour of the laryngeal mucous membrane generally is a pale pink, like that lining the cheeks, while over the cartilages it becomes slightly paler, resembling that of the hard palate. It is, however, subject to rapid variations, and may change from very pale pink to deep red in the course of a laryngoscopic examination.

To the beginner the partial inversion of the laryngeal image may cause a little confusion. The only inversion is antero-posteriorly; the epiglottis, which is seen in the upper part of the mirror, appearing farthest away, while the posterior wall, reflected in the lower part of the mirror, appears nearest the observer. Laterally there is no transposition; but as the observer sits facing the patient, what is left from the side of the patient is to the right of the observer, and *vice versâ*.

The chief difficulties in making a laryngoscopic examination arise either from nervous apprehension on the part of the patient or from hyperæsthesia of the pharynx. The former, which is more common in women, is best overcome by assuring the patient that we are only going to make an *examination* and not to *do* anything, and by introducing the mirror for a brief space and withdrawing it again, even without seeing the larynx. A little patience and manœuvring will soon attain our object, whilst any haste or impatience will only lead to failure.

Local hyperæsthesia, so common in men, can be got rid of by the application of a 10 per cent solution of cocaine, but the practised laryngoscopist will only rarely require to employ it for this purpose. By placing the mirror just in front of the uvula while the patient sings a high-pitched "ee," at other times by depressing the tongue instead of holding it out, a sufficient view will be obtained even in the most irritable. As a rule, the hyperæsthesia of the pharynx is but another name for the clumsiness of the observer.

The tongue at times causes difficulty by the frænum being so short as to prevent its being protruded, or it may be so thick and fleshy as to fill the cavity of the mouth, or more frequently by a reflex action the dorsum becomes so arched that the introduction of the mirror is impossible. All these difficulties are removed by using a tongue-depressor and then introducing the mirror in the ordinary way.

In a small number of cases the epiglottis lies so far backwards over the larynx as to prevent a view of its interior. The singing of a high-pitched "ee," or the making a few rapid and noisy inspirations, will usually suffice to raise the epiglottis; or a tongue-depressor, such as that of Mount Bleyer or Schmidt, may be employed to pull forward the root of the tongue. If these means fail we can paint the larynx with a 20 per cent solution of cocaine, and then by means of the laryngeal sound raise the epiglottis.

The examination of children, though often easily accomplished, at other times presents great difficulties. If the child is old enough to understand, we should try to gain its confidence, and proceed exactly as in adults. Very young or unruly children should be placed on a nurse's knee, with the legs fixed between hers, and held erect while the head is steadied by an assistant. By using a tongue-depressor and quickly introducing the laryngeal mirror, a view sufficient for the purposes of diagnosis may sometimes be obtained. Too often, however, the excess of frothy mucus in the lower pharynx and the rolling together of the epiglottis will defeat our purpose. If it is imperative to make an examination, we can of course give a general anæsthetic and employ a gag and tongue-depressor, or we may have recourse to the direct method of Kirstein. Lack has suggested a method which is specially valuable in very young children. The tip of the left forefinger is passed into the right pyriform sinus, and the terminal phalanx hooked round the hyoid bone, which is pulled forward. A small laryngeal mirror is then introduced. In children with teeth he uses a curved tongue-depressor instead of the finger. The younger the child the easier the examination, and no anæsthetic is required.

There are a number of modifications of the usual method of laryngoscopy, which enable us to obtain better views of certain parts of the larynx. Thus, by throwing the head well backwards, and holding the mirror more vertically, we can get a better view of the anterior commissure; while to see the posterior wall we adopt Killian's position, in which the head is bent forward Avellis has described two methods which will be found of value in getting a view of one side of the larynx, so as to see the under surface of the ventricular bands and into the ventricles. These consist in either bending or rotating the head towards the side to be examined, while the mirror is placed at the opposite side of the uvula and held at an appropriate angle. Thus to examine the right side of the larynx bend the head towards the right shoulder, or rotate it in the same direction, while placing the mirror to the left side of the uvula.

2. *Direct Inspection.*—Under the name autoscopy a method of examining the larynx and trachea by direct inspection was introduced a few years ago by Kirstein of Berlin. By means of a specially constructed tongue-depressor the base of the tongue and epiglottis are pulled forwards and downwards, whilst light is thrown directly into the larynx from an electric lamp either attached to the handle of the tongue-depressor or worn on the forehead. The patient should be seated on a chair with his neck freed of all clothing, and should bend the upper part of his body forward, while his head is slightly inclined backwards. The observer standing in front introduces the spatula, so that its tip catches in the groove between the tongue and epiglottis, and draws the base of the tongue evenly and steadily downwards and forwards. Kirstein claims that he can see the whole larynx and trachea, except the anterior commissure, in about a fourth of all adults; and that about one-half of all people can be fairly well examined, so that the posterior region of the larynx is exposed to view. It is evidently, therefore, not a method to replace laryngoscopy, but where practicable it is of great value in enabling us to obtain a better view of the posterior wall of the larynx and trachea than the laryngoscope gives. It is a method, however, which requires a very great deal of practice to acquire, and which calls for considerable endurance on the part of the patient. One undoubted advantage it has is the ease with which children can be examined in this way when under chloroform. The head of the child should be drawn over the edge of the table and held by an assistant. The spatula is then introduced and the tongue pressed forward in the usual manner; the head of the child is then raised or lowered, till the correct position is obtained which exposes the larynx to view.

3. *Transillumination.*—If a bright light be concentrated on the side of the neck, and the laryngeal mirror be introduced in the usual

way, a sufficient view of the larynx can be obtained to make out the different parts more or less distinctly. This fact was observed by Czermak, but not considered of any diagnostic importance. Voltolini afterwards took up this method and pursued it with great diligence, employing a small electric lamp with a water lens as the source of illumination. It can be most conveniently carried out by employing the ordinary frontal sinus-lamp, which is best placed above or below the thyroid cartilage, and either at the side or in front of the neck. Voltolini expected great things from this method in the direction of distinguishing simple from infiltrating growths, and in determining the thickness of laryngeal webs. As a matter of fact it has little or no practical value, and the experience of Gottstein, that "in no case does it tell us more than the laryngoscope, but always less," coincides with that of the great majority of observers.

4. *Skiagraphy of the Larynx.*—The actual value of the X-ray method in examining the larynx is still comparatively small. That it enables us at times to locate more exactly the situation and lie of a foreign body in the larynx or trachea, is beyond doubt. But the hopes which have been expressed that the infiltration of malignant growths, or ankylosis of the crico-arytenoid articulations, might be detected by the use of the rays, have so far remained unfulfilled.

By means of the photographic plate a more or less distinct picture of the hyoid bone and the laryngeal cartilages can be obtained, but the outline of the cartilages is so poorly defined that the diagnostic value of this method must be very little if any. Ossification of cartilage and fracture of the hyoid bone are said to have been detected by the use of the X-rays.

5. *Palpation.*—Through the introduction of laryngoscopy the diagnosis of laryngeal diseases by the educated finger has become a lost art. A sentence from Gairdner's *Clinical Medicine*, published in 1862, is of interest in this connection. "I am still of opinion," he writes, "that any one who has accustomed himself to the careful and scientific use of the finger in the diagnosis of laryngeal diseases will but rarely find his knowledge increased by the comparatively troublesome and difficult method of laryngoscopy."

*Internal palpation* is still of value in searching for foreign bodies, and in determining the size and consistence of growths of the epiglottis, aryepiglottic folds, and entrance to the larynx. In young children, too, it may at times help us to a diagnosis, as in multiple papillomata, which may be felt when situated above the glottis. The laryngeal *sound*, introduced under the guidance of the mirror, enables us to palpate those parts which are beyond the reach of the finger. It is the first instrument with the use of which the beginner should become familiar, as it will educate his eye and hand for the carrying out of all other intra-laryngeal manipulations. It will be safest for him to practise with Schroetter's sound, which is simply an English bougie stiffened by having a stout wire run through its centre, before taking to the finer instruments made of silver, copper, or aluminium.

The sound is employed to determine the mobility and consistence of tumours or swellings, to detect fluctuation, to gauge the depth of an ulcer and find the condition of the underlying cartilage, to hold aside growths or swellings at the entrance to the larynx, to raise the epiglottis when pendent, and to test the sensibility of the laryngeal mucous membrane.

*External palpation* will often be employed in examining the larynx and trachea. It enables us to determine the amount of lateral displacement of these organs from the pressure of growths in the neck, and at times to discover the cause of a stenosis not explained by the laryngoscope. We may detect crepitation in fracture or necrosis of the cartilages, and tenderness and swelling in external perichondritis. In many cases of tracheal obstruction we can *feel* the stridor, and locate it better by the fingers than by the stethoscope. The value of palpation in detecting enlarged glands in syphilis and malignant disease hardly requires mentioning.

Occasionally it will be found that pain in swallowing, for which no cause is discovered in the pharynx, or larynx, is due to a rheumatic affection of one of the external muscles, which will be tender on pressure.

6. *Examination of the Trachea.*—For this purpose a more intense light is required than for ordinary laryngoscopy. Where the trachea is straight and the epiglottis erect, we may often get a good view of the anterior wall and down to the bifurcation, by simply altering the angle at which the mirror is held. Slight external pressure will often assist in straightening the trachea, or the same result may be obtained by placing the patient sideways in a chair, and then rotating his head so as to face the observer.

The most successful method, however, and the only one which gives us a view of the posterior wall, is that suggested by Killian. The patient, having loosened all clothing about his neck, should stand with his head bent forward till the chin touches the sternum. The observer, either sitting or kneeling before him, reflects the light from below into the mouth. The laryngeal mirror, of as large a size as possible, should be placed rather farther forward, and held more horizontally than in ordinary laryngoscopy, while the soft palate is pushed strongly upwards.

Where tracheotomy has been performed, a view of the under surface of the cords and of

the whole trachea may be got by introducing a small steel mirror through the tracheotomy wound. By this method growths and cicatricial webs of the larynx have been discovered, as well as foreign bodies in the trachea.

## Larynx, Acute and Chronic Inflammations

LARYNGITIS—
ACUTE . . . . . 324
CHRONIC . . . . . 327
HYPERTROPHIC . . . . . 331
MEMBRANOUS . . . . . 331
ŒDEMATOUS . . . . . 332
CHRONIC SUBGLOTTIC . . . 333
NODULAR . . . . . 334
SICCA . . . . . 335
ACUTE, IN CHILDREN . . . 336
LARYNX—
INJURIES TO . . . . . 336
DISLOCATIONS OF . . . . . 337
FOREIGN BODIES IN . . . . 337
LARYNGEAL HÆMORRHAGE . . . 339
GLOTTIS—
CONGENITAL GLOTTIC STENOSIS . . 339
PACHYDERMIA LARYNGIS . . . . 340
BLENNORRHŒA . . . . . 341

### Acute Laryngitis

SYNONYMS: *Acute Inflammation of the Larynx, Cynanche Laryngea, Angina Laryngea, Acute Catarrh of the Larynx.*

DEFINITION. — An acute catarrhal inflammation of the mucous membrane of the larynx, characterised by hoarseness or aphonia, pain, and cough. It is, when uncomplicated, without danger to life, and subsides spontaneously in three to ten days.

ETIOLOGY. — *Predisposing Causes.* — Acute laryngitis is more apt to occur in the subjects of chronic affections, viz., those with defective digestive, vascular, renal, or respiratory systems. Over-indulgence in eating and drinking, and what is termed a "loaded" condition of the stomach and liver, are amongst the most potent predisposing factors. Defects of circulation naturally make the system less resisting, and, like affections of the kidney, increase the proneness to local œdema and catarrh. Amongst the predisposing causes may be numbered gout and rheumatism.

Acute inflammation frequently attacks the larynx primarily, and then extends down the trachea. More rarely it may first develop in the bronchi and then spread upwards. It is so uncommon for acute inflammation of the lungs or pleuræ to be found with a similar condition of the larynx, that the association can only be looked upon as accidental; on the other hand, it is very usual for catarrhal inflammation to make its appearance in the nose and pharynx, and then spread downwards to the larynx.

Not only does acute inflammatory catarrh frequently start in the nose and by contiguity spread directly downwards to the larynx, but chronic affections of the nose and pharynx are amongst the most frequent predisposing causes of acute laryngitis. For further consideration of the etiological influence of nasal affections see section on Chronic Laryngitis, p. 327.

Those who lead an indoor or sedentary life, especially in ill-ventilated and dusty rooms, are much more prone to attacks of laryngitis than those who are occupied with an outdoor or hardier existence. This tendency is greatly contributed to by the habit of loading the body with unnecessary clothes, wrapping up the neck, and fearfully avoiding every current of fresh air from the supposed dread effects of a "draught."

Both extreme youth and extreme age predispose to laryngitis, and in the young the condition is so important and presents so many special characters that it will be considered separately (*vide* Acute Laryngitis in Children, p. 336). In the elderly the condition is apt to occur from their diminished powers of resistance. Men suffer from acute catarrhal affections of the larynx more frequently than women, and this has generally been attributed to their greater exposure to vicissitudes of weather. But, as has just been pointed out, an open-air life in itself is rather a preventive of laryngitis, and it is much more probable that the affection occurs more frequently in members of the male sex from their greater self-indulgence. A very common event is for a patient to pass some hours, stimulated in many cases with alcohol, in the vitiated atmosphere of a crowded and smoke-laden room and then go out directly into a cold, and possibly damp air. The cold air is, of course, blamed for an attack of laryngitis in which it only played a subsidiary part.

The disease is more frequently met with in the months of winter and spring. It is more apt to occur in the subjects of chronic laryngitis.

*Exciting Causes.*—An attack of acute laryngitis is generally directly attributed to exposure to cold and wet. This is more operative when there has been a sudden fall in the temperature associated with increase in the moisture in the air—such as occurs in this climate with east winds. Apart from this it may be directly excited by the inhalation of the irritant fumes of chlorine, bromine, iodine, ammonia, or of sulphuric, nitric, or other fuming acids. The dust of chromic acid, brick dust, and similar powders in factories and workshops will give rise to it. Improper use of the voice —as in the yelling and shouting of street demonstrations—will often induce acute laryngitis, especially if the enthusiasm has been stimulated by free indulgence in alcohol. Even without other exciting agents it will sometimes ensue on the vomiting and retching following an alcoholic debauch, and I have known it to be induced by sea-sickness. The passage of foreign bodies into the larynx can give rise to

acute inflammation, and amongst other traumatic causes may be mentioned the clumsy introductions of instruments, or the accidental irritation produced when powders or paints intended only for the pharynx happen to fall into the glottis. It has been caused by bungling attempts to introduce the stomach-pump.

It may occur in acute infectious fevers, such as influenza, measles, whooping-cough, small-pox, typhoid and scarlet fever. Apart from these specific affections, acute laryngitis is doubtless frequently of septic origin, and is occasionally infectious.

*Pathology.*—The pathology of acute laryngitis does not differ from that of inflammation of other respiratory mucous surfaces. In the first stage there is hyperæmia, with dryness from arrest of the mucous secretion. As this first stage abates there is increased flow of mucus, mixed with the cast-off leucocytes. There has been a good deal of discussion as to whether actual ulceration is ever found as a result of a simple catarrhal process. The defects which are sometimes seen on the vocal cords are probably more apparent than real, and at the most are only abrasions of the epithelial surface. The opportunities of post-mortem verification are too infrequent to settle the point.

The affection may limit itself more particularly to one part of the larynx, receiving accordingly the name of epiglottiditis, arytenoiditis, chorditis, etc.

Symptoms.—If the laryngitis is due to the spread of inflammation from the nose or pharynx, the symptoms will have been ushered in with those of the primary affection.

The onset may be preceded by a feeling of chill or even a slight rigor, but as a rule the constitutional symptoms are slight. Generally speaking, the first symptom is discomfort in the throat, and a feeling of fulness followed by dysphonia or hoarseness. The voice may sound shriller or slightly falsetto; but it is much more usual for it to sink to a bass, while it loses its tone. Complete aphonia may occur; and the voice is generally worse in the morning. Cough is not at all a usual symptom, and if it occurs in this stage it is short, harsh, and ineffective. There is no expectoration. Talking becomes excessively painful, and often excites the action of swallowing, which adds to the patient's distress. This dysphagia is more marked on swallowing merely saliva than on the ingestion of food, and it is more apt to occur when the inflammation particularly attacks the arytenoid region.

Indeed, there is often the sensation as of a foreign body in the larynx, producing a constant desire to swallow. This only aggravates the discomfort, and spreads the sore feeling upwards towards the ears. There is seldom any external pain or tenderness, and, indeed, the firm grasping of the larynx frequently gives a feeling of support and comfort.

With these local symptoms there may be very little general disturbance; in some cases there may be slight feverishness, while in others there is considerable malaise; but there is never high fever, the appetite is never completely lost, and the night's rest is rarely destroyed.

At the end of twenty-four to forty-eight hours relief is generally ushered in by a freer secretion of mucus from the lining membrane, not only of the larynx, but also of the trachea, which is very commonly affected at the same time. The voice at once becomes less toneless and loses its hoarse and harsh character, the dysphagia disappears, and cough, if previously present, is no longer painful. If not present before, it is now started by the necessity of expelling the freely secreted mucus. As this is expectorated a sensation of rawness is generally referred to the front of the trachea.

With the restoration of voice all feelings of malaise commence to disappear; and if the restored function of vocalisation is not abused, the *restitutio ad integrum* is complete in a few days.

When the larynx is examined with the laryngoscope in the early stage of this disease, the visible changes may appear slight and insignificant in proportion to the disturbance of voice and the general discomfort. The vocal cords themselves may show no signs of inflammation, or nothing beyond a slightly catarrhal surface. The rest of the laryngeal mucous membrane—that covering the arytenoid cartilages, the aryepiglottic folds, the ventricular bands, and the inter-arytenoid space—is at first duskily injected. This becomes brighter and more marked as the hoarseness increases, when the vocal cords will be seen to be injected, while their flat, ribbon-like surface becomes dull and rounded from the infiltration of the mucous membrane. Owing to the absence of submucous tissue and the consequent close adhesion of the epithelium to the subjacent tissue, the swelling and œdema, which may occur in other parts of the larynx, is very rare here. Although there may be considerable hoarseness without marked inflammation of the vocal cords, still in acute cases they may entirely lose their white appearance and become not only pink but so acutely injected as to assume exactly the same colour as the ventricular bands. In severe cases the ventricular bands and aryepiglottic folds are so congested and swollen as to more or less completely conceal the vocal cords, even on phonation. Part of the aphonia is no doubt due to the inflammation affecting the subjacent internal crico-arytenoid muscles, and, on phonation, the cords are seen not to approximate owing to deficiency

of the internal tensors. In other cases this approximation of the cords is due to the swelling of the lax mucous membrane lying over the inter-arytenoid region. Hæmorrhage occasionally takes place into the submucous tissue, and blood may even escape—generally in small streaks only—from the surface. This latter variety has been termed *hæmorrhagic laryngitis*. In the first stage the absence of mucus in the larynx is noticeable. As the inflammation abates the parts are seen to be bathed in secretion, generally of a muco-purulent character, which is observed coming up from the trachea and welling over the inter-arytenoid region to pass into the œsophagus.

The epiglottis is not usually involved in ordinary cases of laryngitis, although the lower part of the laryngeal surface (the cushion) may present an injected and velvety appearance.

The appearances of acute inflammation will generally be found at the same time in the nose and pharynx; and the mucous membrane of the trachea frequently shares in the inflammation.

As recovery takes place the cords are restored first to a dirty grey colour, and then to their normal tint, while they lose their rounded upper surface and assume their flat appearance. Some paresis of the internal tensors may be evident for some time, particularly in cases where the voice was not rested during the illness, or used too soon during convalescence.

Diagnosis.—The diagnosis of complete laryngitis presents very little difficulty. An examination of the nose and pharynx will exclude other possible affections; and the use of the laryngoscope will reveal the exact condition of affairs. It will be seen that the hoarseness is not due to any growth or paralytic affection, while the bilateral character of the affection and its uniform distribution will point to its catarrhal character. The comparatively sudden onset of the affection is also a characteristic.

The possibility of a foreign body having entered the larynx should never be overlooked.

Prognosis.—When a primary affection, acute laryngitis is free from danger. Recovery generally takes place within three to eight days, or else the condition passes into a chronic affection. It is of graver importance when it occurs in the aged, the broken down, or as a complication of infectious fevers or systemic conditions.

Treatment.—The treatment may be considered as local, general, and preventive. The first and most important point in treatment, and one too often neglected, is the insistence on complete rest for the voice. This should be as near absolute silence as possible, and even whispering should be avoided. The custom of attempting to treat a patient's larynx, when affected with acute inflammation, so as to enable him to sing or speak at some public function, should be severely discouraged; it is as unphysiological as to allow a patient to walk about with acute synovitis in the knee-joint. Next to rest of the voice there is little doubt that, whenever possible, general rest should be enjoined, and the patient is much more likely to make a rapid recovery if he is put to bed, and treated with a dose of calomel in the evening followed by a morning saline cathartic. The diet should be light, but need not be restricted; alcoholic stimulants and smoking being, of course, strictly forbidden. The room should be kept warm but freely ventilated. The custom of overheating the room, excluding every breath of fresh air, and filling the atmosphere with the steam from a bronchitis kettle, is only mentioned to be discouraged; the vitiated air and unnecessary heat can only depress the recuperative power of the patient, while the clouds of steam soon condense in chilling damps on his body and bedclothes. Internally, quinine is frequently prescribed; but I have never seen any benefit accruing from it, while it often adds to the discomfort of the patient. A few doses of salicine, say 10 grains every three hours, are much oftener attended with relief. Tincture of aconite, in drop doses every quarter of an hour until perspiration is induced, is said to mitigate the severity of an attack.

The action of the skin may be encouraged by warm drinks, diaphoretics, or the administration of pilocarpine gr. $\frac{1}{20}$ every three or four hours.

In the early stage it is wiser to refrain entirely from direct medication of the larynx, and all astringents, as well as the use of gargles and the insufflation of powders, should be avoided. Counter-irritation over the neck and chest by blisters, etc., have generally been discarded, but the gentle warmth of turpentine liniment over the front of the neck is sometimes comforting. Cold compresses, frequently renewed, will give relief, or cold may be applied by means of Leiter's coils. Sucking small pieces of ice will sometimes relieve the soreness, especially if dysphagia is present. A lozenge containing codeia gr. $\frac{1}{8}$, heroin gr. $\frac{1}{12}$, morphia sulp. $\frac{1}{4}$, or other sedative will generally ease the pain and check the useless cough.

The natural history of acute laryngitis shows that discomfort is overcome as soon as free secretion of mucus takes place, and there can be little doubt that there is no more soothing application to an inflamed laryngeal mucous membrane than its own mucus. With this object in view we should order inhalations of steam, from a jug or specially constructed inhaler containing half a pint of water at a temperature of 120° F. (65° C.), to which has been added a teaspoonful of compound tincture of benzoin, hemlock, or hops, or some stimulant oils, such as camphor, oil of turpentine, oil of peppermint, oil of tar, creasote, and others.

The steam should be inhaled deeply through the nose and mouth for five minutes every

two or three hours. The steam may also be obtained from a Siegle's spray, the water being medicated with the addition of 2 per cent of benzoate of soda, or other mucus solvent.

The onset of secretion may be encouraged by sipping hot milk mixed with Vichy, Vals, or Ems water, or by the administration of small doses of iodide of potassium.

As the second stage of laryngitis develops the secretion of mucus may be further encouraged by sucking the trochisci morphiæ c. ipecacuanha of the British Pharmacopœia, or by the administration of the usual expectorants. I have found that apomorphine in doses of gr. $\frac{1}{50}$ will prove satisfactory, or the following prescription:—℞ Ammon. chloridi grs. iv., Spirit. ether. nitrosi ℥ xv., Syrup. scillæ ℥ xx., Syrup. tolut. ad ʒj. Ft. Dosis. Sig.: A teaspoonful in water every three or four hours. Small repeated doses of iodide of potassium undoubtedly promote secretion; and vinum antimonialis is a useful drug either in combination with the iodide or given separately.

As the acute stage passes off these "vapores" may be superseded by the sprays of liquid vaseline—known under various names as paroleine, alboleine, cimoline, benzoinol, etc.—either plain or with the addition of menthol, camphor, eucalyptus, oleum gaultherium, oil of peppermint, tar, creasote, etc.

As soon as the acute symptoms are past, the sooner the patient gets out of doors again the better. The use of the voice should be resumed with care, as otherwise a condition of chronic laryngitis may be set up. If there is left a want of tension in the cords this can be improved by the administration of strychnine or nux vomica, or by the use of electricity and massage.

As in a large majority of cases there is considerable catarrh of the nose and pharynx, great relief can be obtained by cleansing the nasal fossæ and post-nasal space with a warm alkaline solution. Some of the various modifications of Dobell's solution will be found suitable for this; or simply 5 grains to the ounce of either borax, bicarbonate of soda, or table salt will be found sufficient. The addition of a small quantity of cocaine hydrochlorate, about gr. $\frac{1}{20}$ to the ounce, is very comforting, and in such a small proportion is without risk, except in children, in whom it should be entirely avoided. This cleansing is best effected with the post-nasal syringe, but it can be done with an ordinary anterior nasal syringe or coarse spray.

The methods of prevention have been indicated in what has been already said. Moderation in the use of alcohol and tobacco, the avoidance of dusty, crowded, and overheated rooms, misuse of the voice, and observation of the ordinary rules of hygiene, should be enjoined. Locally, any chronic affections of the air-passages should receive attention

## Chronic Laryngitis

SYNONYMS: *Chronic Catarrh of the Larynx, Chronic Inflammation of the Larynx, Chronic Laryngeal Catarrh.*

DEFINITION.—A chronic catarrhal inflammation of the mucous membrane of the larynx, the chief symptom being alteration and impairment of the voice.

ETIOLOGY.—The mucous membrane of the larynx is the part of the respiratory passages which is least seldom the primary or sole seat of chronic inflammation. Except, perhaps, in the case of professional voice-users, it is quite exceptional to find idiopathic chronic laryngitis. There are probably two causes which explain this observation. One is that the arrangements in the nose and naso-pharynx are so perfect for protecting the organism from deleterious conditions of the atmosphere, that the inspired air is in the most suitable conditions as regards warmth, moisture, and filtration before it reaches the vocal cords. The second reason is possibly that the non-vascularity of the vocal cords themselves, and the scarcity of glands in the larynx, are both conditions which would lend little foothold to chronic catarrhal processes in the absence of other causes contiguous or constitutional.

Disorders of the nasal and post-nasal cavities, and to a less extent of the pharynx and mouth, are the most potent factors in the origination of chronic laryngitis. The importance of nasal respiration is now so generally recognised that it is sufficient to call attention to the deterioration which must occur in the pharynx and larynx from chronic mouth-breathing. By its passage through the nasal fossæ the current of air is charged with moisture, raised to the temperature of the body, and filtered from dust and other gross impurities.[1] In ordinary conditions it is also deprived of the micro-organisms which float in it, the greater number being arrested at the very entrance of the nostrils, while those which penetrate farther are enclosed in the nasal mucus, which is inimical both to their development and to their farther penetration. The leucocytes also help in resisting bacterial invasion, while the ciliated epithelium rapidly removes the arrested organisms.[2]

Now chronic catarrhal affections by narrowing the calibre of the nasal chambers, diminishing or altering the secretion of their mucous surfaces, and destroying the properties of the ciliated epithelium, interfere with these physiological

[1] Aschenbrandt: *Die Bedeutung der Nase für die Atmung*, Würzburg, 1886; Kayser, "Die Bedeutung der Nase für die Respiration," Pfluger's *Archiv*, Bd. xli., 1887; Bloch, "Zur Physiologie der Nasenatmung," *Zeitschrift f. Ohrenheilk.*, Bd. xviii., 1888; MacDonald, *Respiratory Functions of the Nose*, London, 1889; Schutter, *Annales des mal. de l'oreille*, April 1893.

[2] St. Clair Thomson and Hewlett, *Lancet*, January 1896.

safeguards. The inspired air then impinges directly on the pharynx and larynx, and being cold, dry, and unfiltered, it deposits its impurities on these surfaces, which it robs of warmth and moisture. There are not in the pharynx any arrangements similar to those in the turbinal bodies of the nose for the protection of the organism, and consequently its mucous membrane becomes dry, congested, and chronically inflamed.

Besides thus influencing the properties of inspired air, nasal and pharyngeal affections also predispose to chronic laryngitis by the possible spread of catarrh by direct continuity of tissue, and by the septic and irritant matter which may find its way directly into the larynx. Chronic nasal troubles also predispose to laryngitis by the "hemming" and hawking which they sometimes excite, and also by interfering with one of the chief resonating cavities of the voice. In this way increased strain is thrown on the laryngeal muscles, and catarrh and paresis are more easily induced.

Chronic laryngitis may be the consequence of chronic catarrh of the trachea and bronchi. Whether this is the result of direct propagation, or is due to the coughing produced and the straining efforts necessitated to expel the mucus, it is certain that it is a much more common cause than in the acute affection. Many cases of laryngeal catarrh are overlooked from a neglect to examine the chest for emphysema and chronic bronchitis. An inveterate form of laryngitis sometimes precedes the development of any physical sign of laryngeal or pulmonary tuberculosis.

Any inflammatory or ulcerative processes in the mouth, uncleaned or carious teeth, and pyorrhœa alveolaris, are also conditions which may be etiological factors in the disease.

Diathetic or visceral disorders are frequent causes of catarrh of the upper air-passages. Thus gastro-intestinal, hepatic, cardiac, and even renal defects will either directly cause catarrh, or will produce a reflex cough, which in its turn and through its persistency sets up chronic inflammation. The same may be said for rheumatism and gout, the former producing a painful and the latter a very persistent form of laryngitis. All the direct or reflex causes of cough may be claimed as productive of laryngitis, in that the cough will itself induce a laryngeal catarrh. It may form part of an attack of asthma or hay-fever; and has been traced to frequent fits of weeping.

Excessive, and more particularly faulty, use of the voice is a potent factor in production of chronic laryngitis, particularly in those who are predisposed by any of the conditions already mentioned.

The chronic affection very frequently dates from an attack of acute laryngitis, especially if the patient has not rested the voice carefully during the illness, or has returned too soon to his use of it, or to unsanitary surroundings.

All ages are subject to this affection, although it is more commonly met with in adult life. Both sexes may be affected, although men, from exposure to the causes already enumerated, are more prone to it than women. Women are said to be more subject to it if exposed to any of the exciting causes during the period of menstruation. John Mackenzie has drawn attention in an interesting study to the relationship between disorders of the sexual system and affections of the larynx.[1]

Some drugs, and particularly iodide of potassium, will produce a laryngeal catarrh which might be mistaken for chronic laryngitis. With sensitive subjects the inhalation of certain odours are even sufficient to induce an attack.[2]

As a secondary phenomenon chronic laryngitis is nearly always present in long-continued diseases of the larynx, such as tuberculosis, lupus, syphilis, leprosy, paralysis, and in the formation of new growths, simple and malignant.

Amongst the general external conditions which give rise to chronic laryngitis the reader is referred to the article on the acute form. It is here sufficient to recall that the chief enemies of the larynx are dust, alcohol, and tobacco.

Pathology.—In this affection there is permanent hyperæmia of the blood-vessels from long-standing irritation. There is small-celled infiltration of the submucous tissues. The epithelium may be abraded in parts. In many cases there is a certain amount of myositis, owing to the proximity of the intrinsic muscles to the mucous surfaces. The mucous glands are stimulated into increased flow of a thick, tenacious secretion; but it is hardly likely that the racemose glands should be so exclusively affected as to justify the description of a separate variety under the title of glandular laryngitis, as has been done by the older authors.

Symptoms.—The constant, and sometimes the only, symptom complained of is the alteration of the voice. This is husky at first, with intervals when it may somewhat suddenly and unexpectedly resume its natural clearness; but as the affection becomes established the hoarseness is more persistent. The tone of the voice is always lowered, and the vocalisation becomes harsh. Aphonia is seldom complete, except after prolonged or extreme forcing of the damaged organ. The hoarseness is more marked after a rest, or on rising in the morning, and tends to disappear after a little use; but if this restoration of voice is at all freely made use of, the hoarseness tends to become worse than before, and in any case it is apt to recur later in the day.

The patient is conscious of the increased

[1] John Mackenzie, *Journ. of Laryng.*, March 1898.
[2] Joal, *Revue de laryngol.*, 1894.

effort which is made in vocalisation, and this produces a sense of fatigue and soreness in the throat. There is not necessarily any cough, but constant "hemming" and hawking in the efforts to clear the larynx of the sticky mucus which hangs about the affected parts. Abundant expectoration generally indicates that the trachea and bronchi are affected with the same catarrhal process. Cases have occurred of profuse catarrh from the larynx, to which the term laryngorrhœa has been applied.

The alterations in voice are more noticeable in women and in tenor voices, than with baritones or basses. That is to say, a condition of laryngeal catarrh which would cause a decided change in the speaking voice of women and tenors might hardly be noticeable in a bass, and would probably not prevent him from singing with his usual success, at least for a while.

In a considerable number of cases the patient will also present the symptoms of concomitant nasal or pharyngeal catarrh.

EXAMINATION.—The laryngoscope will reveal some variety in the condition according to the duration and severity of the case and the parts principally affected. In all cases there will be certain constant conditions observable. For instance, there will be an absence of acute inflammation, and the changes will generally be bilateral and more or less symmetrical.

The mucous membrane has a congested appearance, the colour varying according to the subject — being frequently darker and more purplish in basses and in the more chronic cases, while it is apt to be redder in female voices and in tenors. Pellets of mucus are frequently seen, generally hanging about the inter-arytenoid space, the vocal processes, or the ventricular bands, and more rarely on the anterior two-thirds of the vocal cords. The cords themselves in many cases are only slightly affected, appearing only dull or a dirty grey in colour. At other times arborescent vessels are seen ramifying on them. (In the normal condition no blood-vessels are to be seen on the vocal cords in the same way, for instance, that they are met with on the epiglottis.) In the worst form the cords may assume a dull deep-red colour. Their surface is generally more rounded, and on phonation it is seen that their approximation is frequently incomplete, either from paresis of the internal tensors of the cords or from the mechanical obstruction presented by thickening in the inter-arytenoid space.

Shallow abrasions of the epithelial surface are sometimes met with, especially towards the inter-arytenoid region. The ventricular bands share in the general congestion, and it is frequently seen on phonation that they are considerably thickened. This may be due to small-celled infiltration, or to muscular hypertrophy from vicarious action of the false vocal cords—the pain or inefficacy of the true vocal cords being supplemented by forcible adduction of the ventricular bands. The aryepiglottic folds share in the process, and the epiglottis shows increased vascularity and sometimes thickening of the petiolus.

Störck has described a fissure as particularly apt to occur amidst the folds of mucous membrane in the inter-arytenoid space when they are pressed together in phonation.

DIAGNOSIS. — The chronic nature of the affection, the absence of constitutional symptoms, and the bilateral and generally symmetrical nature of the affection are usually sufficient to remove any difficulty in the way of diagnosis. Particular care should be taken in excluding the possibility of early tubercle, as this disease is often preceded by a laryngitis of a very inveterate character. The presence of marked anæmia of the air-passages, or any marked constitutional changes, should lead to a careful examination of the temperature, chest, sputum, etc.

PROGNOSIS.—Once established, this affection shows little tendency to spontaneous resolution. In many instances the exigencies of their profession prevent patients from giving the necessary rest to their voice, while in others the drawbacks of their surroundings render a complete cure impossible. In some patients the defects in their upper air-passages may have been overlooked in youth, and so have left behind conditions which are irremediable. In others, again, faulty methods of singing or voice production have become too ingrained to be eradicated.

There is no danger to life in the affection, but the promise of a spontaneous cure in a well-established case is too remote to be taken into consideration. On the other hand, appropriate treatment will lead to recovery in a large number of cases, especially if the patient is willing and able to carry out advice.

TREATMENT.—Success in the treatment of chronic laryngitis will principally depend on the successful detection of the chief etiological factors, and it is surprising how often these will be found outside the larynx itself. The removal of the primary causes is the important point; topical applications, although helpful, fill a secondary rôle. Thus attention to the digestive functions or the action of the kidneys, the regulation of uterine disturbances, the detection of gout and rheumatism, may in some cases be the chief indications for treatment. The customs of the patient as regards food, drink, clothing, sleep, exercise, tobacco, fresh air and ventilation, may require attention. In a large number of cases — the majority, according to Bosworth[1] —treatment will have to be directed to the nose or naso-pharynx. Any morbid process, or marked structural

[1] *Diseases of the Nose and Throat*, 3rd ed., 1897, p. 625.

variation from the normal, should be attended to (see "Nose"). When the principal cause is found—possibly by a process of elimination—to be in the larynx itself, it will generally be discovered that the laryngitis is attributable to faulty use of the voice. It is more misuse than over-use which is responsible for chronic laryngitis, and in many cases it will be found necessary to see that the patient acquires a proper method of voice-production and singing. In most cases, however, treatment should generally begin by rest to the affected parts, and this is only secured by strict silence.

For those to whom this is an impossibility all shouting, public speaking in the open air, lecturing in close, crowded, dusty, or stuffy rooms should certainly be avoided, and the use of the voice limited to the bare necessities of the patient's surroundings.

It is so seldom that the necessities for talking imposed by the ordinary duties of life allow of strict silence, that, for those who can afford it, it is doubtless wiser to go away for a change. Besides, there can be no more natural healer for a chronically inflamed larynx than pure air. Any country air is doubtless better than the air of cities; but general experience has shown that the high, dry air of mountains is apt to be too irritating, and that for an inflamed larynx it is better to choose milder and softer climates, such as those of Madeira, Palermo, Pisa, or the south-westerly coasts of our own shores. There is some difference of opinion, and also of idiosyncrasy, with regard to sea air; but it is fairly certain that strong winds are prejudicial, and that the shelter from them afforded by woods—especially pine woods—is a distinct desirability.

When there is much mucus about the larynx any local treatment should be preceded by a cleansing alkaline spray, such as bicarbonate of soda, borax, salt, either alone or in combination with one another, or with chlorate of potash, salicylate of soda, sugar, etc.

By some it is recommended that these sprays should be used warm; but Moritz Schmidt points out that warm sprays to the nose and throat for chronic conditions only lead to further passive congestion and foster catarrh, whereas the cold spray is not only harmless but much more bracing and stimulating. These sprays may be rendered more soothing where there is cough or discomfort, by the addition of a small quantity of cocaine. In the strength of one grain to the ounce there is no risk in placing it in the hands of a patient, or fear of its starting the cocaine habit. Antipyrine (grs. v. to the ounce) or carbolic acid (grs. ii. to the ounce) also have a sedative action, and if the sprays are made up with fresh peppermint water they are rendered both more pleasant, more soothing, and more antiseptic. A stimulant effect can be produced by the addition of menthol (gr. i.), eucalyptus, oleum gaultherium, oil of cassia, or pine oil.

Or any of these oils can be sprayed into the larynx when made up with a basis of liquid vaseline (paroleine, alboleine, cimoline, etc.). These oily sprays have to a large extent superseded the steam inhalations which were formerly employed for carrying the essential oils into the air-passages.

It may be found necessary to make use of astringents in inveterate cases of chronic laryngitis. This is best done in the form of laryngeal sprays, to which are added one or other of the following:—Nitrate of silver (grs. ii. to v.), sulphate of zinc (grs. v. to x.), chloride of zinc (grs. ii. to vi.), perchloride of iron (grs. iii.), sulphate of copper (grs. iii. to x.) to the ounce. Massei recommends a 2 per cent spray of lactic acid.

If the secretion is thick and tenacious, it may be loosened by pastilles of chloride of ammonia and benzoic acid.

Astringents may be employed in the form of powders insufflated into the larynx, but as they are quickly expelled it is doubtful if their action is ever other than that of a stimulant. Gargles need only be mentioned to be condemned as useless.

The laryngeal brush is seldom nowadays resorted to. In a large number of cases, even when applied with the greatest skill, it produces such an amount of spasm and local reaction, and runs such a risk—from movement on the part of the patient—of local traumatism, that the drawbacks attendant on its use far outweigh the benefits to be derived from it. If cocaine is required before each application the disadvantages of the cocaine may counterbalance the medicinal advantages of the pigment. It may be required in some inveterate cases, and then we generally make use of nitrate of silver, beginning with a solution of the strength of 10 grains to the ounce and increasing it gradually, according to the local reaction produced, upwards, till a strength of 100 grains to the ounce is reached. It has been recommended that the application should be made daily, but there are few cases in which an application once a week is not sufficient. The frequency must be proportionate to the local condition and to the reaction produced. In milder cases, chloride of zinc may be used in solutions of the strength of 20 or 30 grains to the ounce. It has been advised to treat any varicosity in the vocal cords by applications of fused chromic acid, and Krause recommends in chronic cases minute longitudinal incisions into the cords with a suitable laryngeal lancet. Such dangerous proceedings are uncalled for, and it is seldom that milder measures will not secure better results.

Finally, many cases of chronic laryngitis can be greatly relieved, in persons who can afford it, by a suitable change of climate, and particularly

by a visit to such spas as Ems, Mont Dore, Eaux Bonnes, Marlioz, Challes, Cauterets, etc.

Any remaining paresis of the muscles may be met with doses of strychnine or the use of electricity.

Elderly patients who are subject to winter attacks of chronic laryngitis should be recommended a change to a warmer climate.

## Hypertrophic Laryngitis

### General Hypertrophic Laryngitis

Definition.—A form of chronic laryngitis in which the lining membrane of the larynx is more or less uniformly thickened.

Etiology.—A reference to the article on Chronic Laryngitis will show that when long continued there is a tendency to overgrowth of the mucous membrane, more marked in certain regions than in others. All the causes which have been detailed as productive of chronic catarrh of the larynx, are also operative in producing chronic general hypertrophic laryngitis. The latter form is, however, more apt to be met with when the chronic condition has been neglected for some time, and the use of the voice has been insisted on, or the external causes have not been removed. Hence the condition is very frequent in such occupations as that of a street hawker, or those who are exposed to the irritation of dusty occupations.

It is particularly apt to occur in patients who, in addition to the usual causes, indulge freely in alcohol. It is frequently met with in syphilitic subjects, in whom the process does not show any distinct specific character, and may, in fact, manifest great resistance to the influence of syphilitic treatment. This has been termed para-syphilitic [1] laryngitis.

Symptoms.—For a description of the symptoms the reader is referred to the section on Chronic Laryngitis (p. 327). In the chronic hypertrophic variety the change of voice is more marked; there is less tendency to cough; and, although subject to acute or subacute exacerbations, the patient suffers less in using his chronically husky and toneless voice.

Examination.—In addition to the general condition described under the heading of Chronic Laryngitis (*q.v.*), the laryngoscope reveals the thickening which has occurred in the mucous lining of the larynx. This is found most commonly in the inter-arytenoid space, where the hypertrophied mucous membrane may be heaped up into one central mass, though through the frequent approximation of the vocal processes in phonation it has more commonly been folded and so divided into three or more heaps. These are symmetrical, neither inflamed nor ulcerated, and generally of the same colour as that of the mucous membrane usually found in the inter-arytenoid space. The surface may be rough, but is uniform, and in many cases is coated with sticky mucus. On phonation this overgrowth is seen to be compressed between the posterior ends of the vocal cords, and by interfering with their approximation causes huskiness.

In other instances, or in addition to the above, the hypertrophy is found on the ventricular bands, which may be so much thickened as to more or less completely conceal the true cords. This is particularly apparent on phonation, and, indeed, much of this hypertrophy is possibly muscular and due to the ventricular bands having been called into action to support or replace the inflamed or fatigued true cords.

The arytenoid region and the aryepiglottic folds may also be chronically thickened. Hypertrophy of normal tissue is rarely found on the epiglottis.

Prognosis.—Very slight improvement is to be expected without treatment. Strict rest of the voice is seldom secured, and only too often a return to voice use, or exposure to the primary irritating causes, will induce a recrudescence of the affection. That form which has been referred to as para-syphilitic laryngitis gives rise to one of the most inveterate forms of hoarseness (*vide* Syphilitic Laryngitis). Considerable improvement can be secured when due to other causes, provided the patient will carry out the somewhat tedious treatment—a treatment which may also entail considerable expense from the enforced rest to the voice.

Treatment.—Reference should be made to the section on the treatment of Chronic Laryngitis (p. 329). In the hypertrophic form the treatment has to be more persevering and more thorough; it is in this variety that application of caustics on the laryngeal brush find their chief indication. In a few cases it may even be necessary to remove portions of the hypertrophy when it is situated in the inter-arytenoid space. In other regions of the larynx surgical interference is seldom called for.

For patients who can afford it, the method of treatment carried out at Ems, Mont Dore, Marlioz, and similar health resorts is particularly useful in this form of laryngitis.

## Membranous Laryngitis, Non-diphtheritic

Synonym: *Fibrinous Laryngitis.*

Definition.—An inflammation of the mucous membrane of the larynx, accompanied by the formation of a membrane, and not caused by the Klebs-Loeffler bacillus. It is associated with the presence of various other micro-organisms. It may be acute or subacute.

Etiology.—The occurrence of a false membrane in the larynx is not frequently observed; and when the cases in which it is of diphtheritic origin are excluded, it may be said to be a very rare affection.

It may be due to the application of strong

[1] Massei, *Annales des mal. de l'oreille*, vi., No. 2, 1899.

caustics to the larynx, and has been caused by traumatism or the inhalation of boiling steam or irritating vapours.

In some instances it is of undoubtedly septic origin, the micro-organisms which are apparently causative being various staphylo- and streptococci. The membrane presents the same naked-eye and microscopic appearances as in diphtheria, but it is not—as frequently in that affection—found in the pharynx or nose.

SYMPTOMS.—Hoarseness, a croupy cough, and other laryngeal symptoms indicate the region attacked. Dyspnœa may appear early in acute cases, but be little marked in those in which the membrane forms slowly. Constitutional symptoms will depend entirely on the causative factor; but, as a rule, there is not the early and grave depression which is generally characteristic of diphtheria.

Examination will show the presence of a greyish-white or dirty grey membrane on the vestibulum laryngis, or even on the true vocal cords. If due to the bacillus pyocyaneus, it may be of a blue colour. It will be found to be closely adherent to the mucous surface.

DIAGNOSIS. — The principal affection from which this form of membranous laryngitis must be diagnosed is diphtheritic laryngitis. The latter, in the majority of cases, is associated with the presence of membrane in the pharynx as well as the larynx; the Klebs-Loeffler bacillus can be cultivated from a swab taken from the membrane; and the constitutional symptoms are more marked. Membranous laryngitis, in fact, is a local affection causing some general disturbance; but diphtheritic laryngitis is a general systemic infection from laryngeal inoculation. When there is any doubt as to the diagnosis, it is safer to treat the case as if it were one of true laryngeal diphtheria.

PROGNOSIS.—This will depend upon the cause of the affection, on the amount of constitutional reaction, and on the degree of interference with respiration. The prognosis becomes grave when imperfect aeration of the blood is observed, or when cyanosis develops.

TREATMENT.—The treatment should be symptomatic, and will be conducted on the lines indicated in the section on Acute Laryngitis. When the respiration is interfered with, either intubation or tracheotomy may be required.

## Œdematous Laryngitis

SYNONYMS: *Laryngitis, Phlegmonosa, Erysipelas of the Larynx, Œdema of the Glottis, Œdema Glottidis, Œdema of the Larynx.*

DEFINITION.—A certain amount of subacute or passive œdema is apt to occur in many of the ulcerative processes in the laryngeal mucous membrane — syphilitic, tuberculous, malignant —as well as in connection with other affections. But, as generally understood by many, the term is reserved for an acute œdematous infiltration of the tissues bounding the upper larynx, more particularly the aryepiglottic folds and the inter-arytenoid region, and dependent on the following causes:—

ETIOLOGY.—Acute œdema may be induced by the following causes:—The impaction of foreign bodies in the larynx, the inhalation of boiling steam or liquids (as when children drink from the spout of a kettle or teapot), the drinking of scalding or corrosive fluids, the inhalation of very irritating smoke or chemical vapours, or the injudicious or accidental application of caustics to the larynx. Many of the causes which produce acute laryngitis may also excite œdema, but it is rare to find it dependent only on excessive voice use. It may accompany the acute laryngitis of the infectious fevers — measles, scarlatina, diphtheria, enteric, erysipelas, whooping-cough.

Inflammation in the neighbourhood of the larynx, as in malignant disease of the œsophagus, peritonsillar abscess, inflammation at the base of the tongue, etc., may lead to œdematous infiltration of the laryngeal mucous membrane. It may accompany Bright's disease, diabetes, cardiac anasarca, Quincke's œdema (angioneurotic œdema), and myxœdema. It is sometimes produced by iodide of potassium, and even by small doses in susceptible subjects.

One of the most dangerous forms is that dependent on septic infection and often met with in the course of Ludwig's angina, phlegmonous sore throat, erysipelas of the pharynx or larynx, and similar septic infections.

Œdema of the larynx has been met with in hydrophobia, and as an early complication of typhoid fever.

Any growth comprising the tributaries of the superior vena cava, such as goitres, bronchial glands, and mediastinal growths, may lead to passive congestion of the larynx.

PATHOLOGY. — The loose attachment of the mucous membrane to the underlying tissues in the neighbourhood of the aryepiglottic folds, the inter-arytenoid region, and the ventricular bands, readily allows these parts to be infiltrated with serous effusion. Owing to the close attachment of the mucous membrane over the vocal cords and epiglottis, these regions are more rarely affected. The researches of Hajek have demonstrated anatomically how it is that œdema of the larynx does not readily spread across the middle line from one side to the other of the larynx, or from the front to the back of the epiglottis.[1] The œdema may affect chiefly or entirely the subglottic region. The exudation varies according to the cause and severity of the affection. In the passive form it is entirely serous, but in the septic and inflammatory form it is sero-purulent or purulent.

[1] Langenbeck's *Archiv für klinische Chirurgie*, Bd. xlii. Heft 1.

SYMPTOMS.—In the chronic forms—those due to passive œdema—the symptoms develop gradually. The patient has a feeling of fulness and a sensation as of a foreign body in the throat; there is some dysphagia, and from the accumulated mucus and froth about the sinus pyriformis and base of the tongue, the voice becomes thick and hoarse. In the acute septic form the suddenness and severity of the symptoms are very characteristic of œdematous laryngitis. They are often ushered in by a rigor. Dyspnœa is generally an early symptom and may become acute within a few hours. The voice becomes aphonic, and there is great pain and little result in the attempts to clear the larynx of mucus, while great distress is occasioned by any efforts at swallowing it. The pulse is small and quick; there is frequently very great anxiety; and the face, which is bathed in clammy sweat, becomes congested or palely cyanotic.

On examination with the laryngoscope, if a view of the larynx is obtainable, the most striking feature is the prominence of the large œdematous swelling of the aryepiglottic fold on each side. These may be of a dull purple colour, but more frequently they are pale and passively congested. They are either so large or so coated with mucus that an inspection of the interior of the larynx is only occasionally possible, but when this is obtainable the ventricular bands are found to share in the process. If the epiglottis is attacked it will be prominent, inflamed, swollen, and somewhat globular or turban-shaped. If the subglottic region is involved, a uniform red swelling will be seen below each vocal cord. In some cases there will be the symptoms of the causative conditions.

DIAGNOSIS.—When there is a history of a distinct cause, and the onset of the symptoms is sudden and acute, the large, pale, translucent swellings are typical of œdematous laryngitis. There is more difficulty when the history is obscure and when the condition is grafted on some chronic condition, such as tuberculosis.

PROGNOSIS.—This will depend on the cause. Speaking generally, œdema is always a serious condition, except in the instances where it is caused by iodide of potassium, angioneurotic œdema, or other causes in which it is seldom severe. Occurring in the latter stages of tuberculosis and malignant disease, it is of serious augury. It is one of the most fatal incidents in septic infection of the pharynx and neighbouring tissues. The possibility of sudden spasm must not be forgotten.

TREATMENT.—The treatment will be to some extent guided by the discovery of the cause; for instance, the presence of an impacted foreign body might at once determine a tracheotomy. In any case, the œdema must be relieved. If moderate in amount and not sufficient to cause marked laryngeal stenosis, this may be done by sucking ice and by the application of ice-bags or cold-water coils to the neck. Hypodermic injections of pilocarpine (gr. $\frac{1}{8}$) have given excellent results. Spasm may be mitigated by bromide of potassium and chloral. The œdema produced by iodide of potassium will disappear more quickly if bicarbonate of soda is freely administered. When the œdema is more threatening it should be reduced by freely scarifying the infiltrated tissues, previously cocainised, under the guidance of the laryngeal mirror. When the stenosis is very acute, the symptoms threatening, or the cyanosis increasing, tracheotomy should be performed; indeed, in all decided cases the necessity of tracheotomy, which may suddenly declare itself, should always be borne in mind. Quinine, and the tincture of the perchloride of iron in large doses, have been recommended in the septic form, and injections of antistreptococcic serum might be tried.

### Chronic Subglottic Laryngitis

SYNONYM: *Chorditis vocalis inferior hypertrophica.*

DEFINITION.—A variety of chronic hypertrophic laryngitis characterised by overgrowth or infiltration of the region immediately below the vocal cords.

ETIOLOGY.—This form of laryngitis is traceable to the same causes which have been given in describing the diffuse form. Possibly the over-use or misuse of the voice is not such an evident factor as in the other forms of laryngitis. It has been recorded as a sequela of enteric fever.[1]

SYMPTOMS. — This form of laryngitis will manifest itself by the train of symptoms which have already been described under Chronic Laryngitis (p. 328) and Chronic Hypertrophic Laryngitis (p. 331). The symptoms which particularly characterise it are the presence of marked dyspnœa, a metallic ring to the voice, and a short, sharp cough similar to that heard in obstruction of the trachea. There may be greater hoarseness and impairment of the voice. The dyspnœa will vary according to local conditions, and also from time to time; but it is frequently sufficient to cause an alarming sense of suffocation, and not infrequently necessitates active relief.

PATHOLOGY. — Some recent microscopical observations (by Sokolowski and Kuttner) describe the disease as consisting of chronic cell proliferation, both in the mucous membrane and in the submucous and muscular tissues. It may spread as far as the margins of the vocal cords, and gradually develops into a hard indurated mass.

EXAMINATION.—The laryngoscope shows that the hypertrophy is chiefly, if not entirely, limited to the subglottic region, where two uniform, rounded, symmetrical swellings present

[1] Sokolowski, *Archiv für Laryngol.* Bd. ii. Heft 1, 1894.

themselves, more or less closing up the glottic space below the level of the vocal cords. Each swelling presents a margin parallel to the vocal cord above it, and at first sight gives the suggestion of a second and inferior vocal cord. Not only do these two swellings encroach on the glottic chink and so produce dyspnœa, but on phonation it is seen that they considerably impair the complete approximation of the cords and so interfere with the voice.

The colour may be of the translucent, greyish-white character met with in nasal polypi, and in these cases, if examined with a laryngeal probe, the thickenings will sometimes be found to be œdematous. In other cases they are solid, and the colour varies from dull catarrhal pink to vivid congested red.

Diagnosis. — With increased precision in diagnosis this affection, as a primary disease, has become rarer. There was always considerable doubt as to its exact pathological nature, and in many cases it is doubtless due to tuberculous or syphilitic lesions. It has to be differentiated from rhinoscleroma. The latter is, however, a rare affection in this country, and as a rule is accompanied or even preceded by characteristic changes in the nose and pharynx. If examined with a probe the latter growth is found to be hard and cartilaginous; and if a portion of it is removed and sections properly stained, they will show the bacilli of rhinoscleroma.

Prognosis.—In common with other affections of the subglottic region, this form of chronic laryngitis renders the prognosis much graver than in the ordinary forms. This is due to two factors: one is that, being enclosed in the inextensible shield of the thyroid plate, any increase in size must press entirely towards the lumen of the air-tube, which is thus apt to become dangerously narrowed; the second factor is the difficulty of directly treating diseases situated below the vocal cords.

Treatment.—The plan of treatment recommended in chronic laryngitis (p. 329) should be followed with perseverance. The thickening and consequent narrowing may be met by the passage of dilators, such as Schroetter's tubes, or by intubation. When the swellings are œdematous they should be scarified; and if the dyspnœa becomes dangerous, tracheotomy should be performed. The tracheotomy may also give an opportunity for more complete treatment of the infraglottic stenosis. With the recent improvements in the performance of thyrotomy, and the good results obtained, this condition appears to offer a favourable field for laryngo-fissure.

### Nodular Laryngitis

Synonyms: *Singers' Nodules, Teachers' Nodules, Chorditis tuberosa, Chorditis nodosa, Trachoma of the Vocal Cords.*

Definition.—A form of chronic laryngitis, produced chiefly by faulty use of the voice, and characterised by thickening of the vocal cords.

Symptoms.—While presenting many of the symptoms of chronic laryngitis, the most characteristic ones of this variety are hoarseness and voice fatigue. A few days' rest will frequently sufficiently restore the voice for work; but it soon gets husky towards the end of the day's work, and by the end of the week the patient is frequently quite hoarse. The rest on Sunday is sometimes sufficient to enable the patient, if a teacher, to resume work in the following week, but increasing effort is required in talking, and if care is not taken distinct "nodules" are formed. In singers this result is brought about by a faulty method of voice production, particularly by attempts to sing in a register beyond the patient's powers, and by "squeezing" the voice; hence they are most commonly met with in tenor and soprano voices, and are rarely, if ever, encountered in basses and contraltos. The method called the *coup de glotte* has been particularly blamed as a cause of singers' nodules. Although these small hypertrophies have generally been called "singers' nodules," they are met with very frequently in those who only misuse their voices in talking or lecturing. Moure states that the condition is frequently met with in children who join in part singing, and are forced to take a register beyond their compass.[1]

Etiology.—This affection of the vocal cords is induced by the same causes which are responsible for chronic laryngitis and chronic hypertrophic laryngitis (*q.v.*). It differs from them in etiology in that the use, or rather the misuse, of the voice is one constant factor. In many cases it appears to be the preponderating if not the unique cause; but from the greater frequency with which it is met in young female teachers, it is clear that there are other causes at work—anæmia, irregularities of digestion and menstruation, etc.

Pathology.—The hypertrophy may be more marked on one side than on the other, but it is generally bilateral. It may affect the upper or inner surface of the cord. It is found to consist of increase of normal stratified epithelium in the simpler cases; but in others there is a small-celled infiltration of the submucous layer—very scanty in this region—while many distinct "nodules" are found under the microscope to present the characters of œdematous fibromata. In certain cases, as pointed out by Kanthack,[2] the inflammatory process is not limited to the mucous surface, but entails a certain amount of myositis.

Examination.—Inspection of the larynx always shows—together with more or less general laryngeal catarrh—hypertrophy of the vocal cords, rarely one-sided. In some cases this

[1] *Revue de laryngol.*, Feb. 8, 1896.
[2] Kanthack, *Trans. Laryngol. Soc. London*, 1897.

takes the form of a rounded eminence in the centre of the upper surface of the cord, as if half a small hemp seed had been inserted below the epithelial surface; but in the majority of cases the site of the nodule is at the junction of the upper and inner borders, and at one characteristic point, viz., at the junction of the anterior and middle thirds. Sometimes before any nodule is distinctly present, and when the patient is complaining simply of voice fatigue and occasional hoarseness, the only change apparent on inspection is a slight churning up of mucus into a little froth at this situation when the cords are approximated. Later on the cords lose their normally white surface and become dull, slightly translucent, and injected at this point. Finally a nodule appears, generally on both sides, although it is frequently more prominent on one side than on the other. This nodule may vary from the size of a turnip seed up to that of a small pea; its surface is smooth and it is generally semi-translucent, although a few vessels may be seen along its broad attached border. On attempted phonation the nodules, of course, prevent complete coaptation of the cords, and as a space is left in the glottis there is phonative waste. This gives the toneless and hollow harsh sound to the voice. Although these nodules are generally sessile and attached by a broad base, in certain instances they are more mobile, and by an increased effort of phonation the patient will be able to produce a clearer note. With the laryngoscope it will be seen that this is effected by the nodules being forced up on to the upper surface of the cords, so that the inner margins are able to approximate.

These nodules, being dependent on the conditions already mentioned, are of course always accompanied by a certain degree of general laryngeal catarrh. Interfering as they do with the free movement of the cords, the tensors of the latter necessarily become impaired. This is not only from want of use, but, as Kanthack pointed out,[1] because the condition is an inflammatory one, and affects the muscles as well as the mucous membrane.

Increasing hoarseness and sense of fatigue compel the patient to rest the voice, and this always secures a certain amount of relief; but the symptoms and the local conditions generally quickly recur as soon as the patient returns to his injurious surroundings and the over-use or misuse of the voice.

Treatment.—The early stages of this affection should be treated on the lines laid down in the sections on chronic and hypertrophic laryngitis. Before resuming professional use of the voice, it is desirable that faulty methods of using it should be corrected.

It is seldom that caustics should be used for this condition, and I cannot agree with Botey that it is ever desirable to introduce the point of a galvano-cautery into the larynx, considering how dangerous such a proceeding may be even in the hands of the most skilful, owing to sudden movement on the part of the patient and the amount of reaction always set up by a cautery. If the circumstances of the patient make prolonged treatment impossible, or the nodules are well marked, they can be removed with intra-laryngeal forceps. In the majority of cases, and in patients who can afford the time, the treatment recommended for chronic laryngitis will generally be successful, especially when combined with strict rest of the voice. In some cases the silence should be absolute, although improvement is sometimes quicker if the "humming" exercises recommended by Holbrook Curtis[1] are carried out.

[1] *Loc. cit.*

### Laryngitis Sicca

Synonyms: *Chronic Atrophic Laryngitis, Ozænatous Laryngitis, Ozæna of the Larynx.*

Definition.—A chronic inflammation of the mucous membrane of the larynx, resulting in atrophy, and generally associated with the formation of crusts.

Etiology.—It is questionable if this ever originates primarily in the larynx. When symptoms simulating it are found limited to the larynx they are generally the consequence of mouth-breathing or of syphilis.

The disease is nearly always the result of purulent processes in the nose—suppuration in the accessory sinuses, ozæna, syphilis, neglected adenoids, etc. It is originated in the larnyx either by the pus trickling into the larynx, or by the inhalation of the pyogenic organisms from the nose, or as a result of the mouth-breathing induced. It is more common in females.

Symptoms.—Interference with the voice is the leading symptom in this disease. It is worse in the morning, or after working in a dusty atmosphere. When the patient has succeeded in expelling some of the crusts adhering to the mucous surface the voice is quickly restored, although still hoarse. This expulsion of the dried secretion entails a great deal of painful coughing and hawking, and is sometimes accompanied or followed by a little hæmoptysis, due to the abrasion consequent on the separation of the sticky crusts. The expectorated crusts have sometimes a very foul ozænatous odour. The mucus begins to dry again at once, and as the crusts form the patient becomes gradually more or less aphonic and experiences considerable pain in speaking.

Examination shows the presence of chronic laryngitis, and, in addition, the atrophy and crusts which are characteristic of the disease. The latter may be found almost anywhere,

[1] *Voice-Building and Tone-Placing*, New York, 1898.

but are perhaps most common in the inter-arytenoid region, the posterior ends of the cords, and the ventricular bands; they can frequently be seen in the trachea. The mucous membrane is pale and wasted, and when the crusts are removed the surface underneath them is seen to be abraded. The tension of the cords has generally been considerably damaged.

In the majority of cases purulent processes can be traced up to the post-nasal space and the nose, and the etiological conditions mentioned will be found causing other symptoms.

PATHOLOGY. — The process begins from the mucous surface, which is first infected, and then abraded. Many of the mucous glands are destroyed. The underlying tissue is replaced by connective tissue. There is anæmia from the constant presence of septic material, and atrophy from want of use of the muscles.

PROGNOSIS.— Chronic atrophic laryngitis is a chronic disease, and seldom shows any spontaneous tendency to cure. In those cases where it is found to be dependent on a focus of suppuration in the nasal cavities which can be removed, there is good hope of effecting a practical cure, although some amount of chronic laryngitis might still be left as a legacy of the long-standing process.

TREATMENT.—From what has already been said, it will readily be gathered that in the treatment search must first be made for an etiological condition in the pharynx and nose, and treatment directed accordingly. The possibility of a syphilitic diathesis, acquired or congenital, should not be lost sight of; and even when there is nothing to point to specific disease as the primary cause, relief is frequently obtained from the stimulation of the atrophied laryngeal glands by the administration of small doses of iodide of potassium.

A healthier condition of mucous membrane may be promoted by painting with some form of Mandl's solution.[1] The use of the trochisci acidi carbolici of the Throat Hospital Pharmacopœia is cleansing and comforting.

The larynx should be sprayed or syringed out frequently with an alkaline solution, and when freed of crusts it should be lubricated with a spray of paroleine containing menthol or other antiseptics. If the mucous surface is abraded, it should be treated with nitrate of silver or similar caustics as directed in the sections on the other forms of chronic laryngitis. Dust, alcohol, and tobacco should particularly be avoided. A visit to the alkaline or sulphur spas of Ems, Mont Dore, Challas, Marlioz, Aix, or Harrogate will generally be found beneficial.

### Acute Laryngitis in Children

There are certain anatomical peculiarities connected with the larynx in children which demand some special consideration of laryngitis in young subjects.

In childhood the larynx is not only absolutely smaller than in the adult, but it is relatively small in proportion to the development in other regions. The cartilages which compose its framework are much softer than in the adult, and therefore yield more readily to either direct or negative pressure. The mucous membrane is less closely adherent to the subjacent tissues, particularly in the aryepiglottic folds and subglottic region, and as a consequence effusion, and consequent stenosis, takes place more readily.

The lymphatic supply of the mucous membrane is richer in children than in adults, and hence acute laryngitis is more apt to be attended with submucous infiltration.

In consequence of these anatomical peculiarities inflammation of the laryngeal mucous membrane produces acute symptoms much more quickly than in the adult, and the symptoms of dyspnœa and cyanosis are apt to appear early. Besides, not only is the nervous system of the child generally more unstable, but it appears to be particularly sensitive when the larynx is attacked.

In forming a prognosis it should be borne in mind that there is a possibility of risk from spasm of the glottis. Acute laryngitis is always a serious affection in childhood, and the younger the patient the greater the danger.

The symptoms sometimes give rise to what has been called *false croup*. The child may appear quite well through the day or be affected only with a slight cough. During the night dyspnœa may develop rapidly and alarming symptoms of spasm may set in; in most cases these are connected with naso-pharyngeal catarrh, and the symptoms are partly those of laryngismus stridulus.

The treatment suitable will be found under the headings of Acute Laryngitis (p. 326) and Laryngismus Stridulus (p. 374). In children it has been found that emetics are more useful than in adults, and the administration of a tea-spoonful of vinum ipecacuanhæ will often remove a quantity of obstructing secretion. Hot applications over the larynx are particularly useful in children. Finally, it must be remembered that with them life is more readily threatened by acute laryngitis, so that the practitioner should be prepared for intubation or tracheotomy.

### Injuries to the Larynx

ETIOLOGY.—Fractures of the laryngeal cartilages are nearly always the result of direct violence. They are not of common occurrence, and this is probably due to the elasticity of the cartilages and the mobility of the larynx as a whole. Concussion alone does not appear to be sufficient to fracture the thyroid or cricoid car-

[1] ℞ Iodi Pur. gr. v., Pot. Iod. gr. xv., Ol. Menth. Pip. ♏ v., Paroleine ʒj.

tilages, unless the violence is directly anterior and the vertebral column is immovable. Hence these accidents are nearly always the result of direct violence, and most commonly occur when the patient is lying on his back. They are therefore apt to be met with when people fall in the streets and carriage-wheels pass over the front of the neck. A case has been recorded (Mackenzie)[1] in an acrobat who was in the habit of lying flat on his back, while another gymnast jumped on his neck. In garrotting the larynx is often fractured, not by pressure against the vertebral column, but by lateral compression of the wings of the thyroid cartilage. It occurs in the same manner in hanging. In almost all cases these accidents have originated from some form of direct violence. The only exceptions are three or four cases of fractured hyoid bone in which the injury has been due to muscular action.

Ossification of the cartilages will render the cartilage more brittle and liable to break under the influence of violence.

Symptoms.—The symptoms produced by these injuries are local pain and tenderness, swelling of the surrounding parts, and more or less interference with respiration, articulation, mastication, and deglutition. On manipulation there will be found displacement, mobility of the fragments, and crepitus. It is important, in connection with this, to bear in mind that even when the normal larynx is moved from side to side over the cervical spine a sort of crepitus is often felt. Overriding of the fractured edges will give rise to a perceptible deformity; but, needless to say, in many cases the recognition of these signs will be impossible.

Laryngoscopic examination may reveal swelling, congestion, or hæmorrhage into the larynx, and would not only prove useful in diagnosis, but might also be of assistance in giving warning as to the amount of interference with respiration, and so indicate the necessity for an early tracheotomy.

Emphysema of the neck is likely to supervene, and the air will not only distend the cellular tissue of the neck, but may extend to the thorax, back, arms, and abdomen.

Prognosis.—Fractures of the laryngeal cartilages are attended with fatal consequences in a large proportion of recorded cases. The recorded cases of fracture of the larynx show a mortality of from 76 to 80 per cent. Fracture of the thyroid cartilage is a more serious accident than fracture of the hyoid bone, while fracture of the cricoid cartilage appears to have been fatal in every recorded case. Statistics also show that the prognosis is very much more grave when two of these cartilages are injured at the same time; and the same fact has been observed when there has been accompanying fracture of the lower jaw.

[1] *The Throat and Nose*, vol. i., 1880, p. 402.

Still, it is somewhat doubtful if these injuries are always of such a serious nature as statistics would tend to show. In many cases the fracture may pass unrecognised during the lifetime of the patient. Arbuthnot Lane,[1] in 1885, reported that he had found evidence of old fractures of the hyoid bone or laryngeal cartilages in 9 out of 100 bodies which he had examined in the dissecting room; and, indeed, in one instance, there was even a healed fracture of the cricoid cartilage.

Besides, among the cases included in the tables of various writers, there are many in which death has been caused by suicide or by homicide, and thus the mortality rate is considerably increased.

Treatment.—The chief danger lies in the interference with respiration. If this be met by an early performance of tracheotomy, there is no reason why a much larger proportion of patients should not recover than has hitherto been the case.

Some writers recommend that a tracheotomy should be done in all cases, and that even where the diagnosis is not quite certain the operation should nevertheless be carried out. It is certainly well not to allow the onset of suffocation to be the indication for performing tracheotomy. A fatal attack of dyspnœa may occur suddenly in any case, so that no patient should be left beyond the reach of an immediate tracheotomy.

It has been suggested that O'Dwyer's method of intubation (*vide* vol. iv. p. 554) might find a suitable field of usefulness in these cases; but when the cartilages are much crushed it would probably be safer to lay open the whole larynx, after a preliminary tracheotomy, and endeavour to replace the fragments in their proper position before inserting an intubation tube to act as an internal splint.

### Dislocations of the Larynx

Intralaryngeal dislocations are very rare. One or both of the arytenoid cartilages are sometimes dislocated downwards and forwards, or one may be displaced inwards.

The symptoms are seldom prominent, and the condition is frequently only encountered accidentally when making a laryngoscopic examination.

### Foreign Bodies in the Larynx

Various foreign bodies not infrequently obtain entry to the larynx, and their presence there is always fraught with great danger and sometimes with alarming symptoms. In a few cases, curiously enough, they may, for a time at least, give rise to very little distress.

It is difficult to give a complete study of the question of foreign bodies with reference to the larynx only, as they not infrequently pass from the larynx to the trachea, or lower down; and,

[1] *Path. Soc. Trans.*, vol. xxvi., 1885, pp. 82-85.

on the other hand, foreign bodies which at first are lodged in the lower air-passages may become impacted afterwards in the larynx. The occurrence of foreign bodies in the upper food-passages has also to be frequently considered at the same time. References to these regions will therefore complete any omissions in the present section.

Etiology.—Large foreign bodies generally consist of imperfectly masticated boluses of food which become fixed in the laryngo-pharynx. Sometimes they consist of some substance—food or other material—swallowed for a wager. Smaller bodies may be particles of food slipping unexpectedly out of the mouth, swallowed hastily, or unexpectedly met with, as when portions of bone are drunk with soup. Vomited matter sometimes finds its way into the larynx, and this is most likely to occur during or after general anæsthesia. Food is also apt to "go the wrong way" when the sensation of the pharynx and larynx is blunted, as in alcoholic intoxication, in the insane, and in certain neuroses of these regions. In diphtheritic paralysis such articles of diet as tea, milk, and bread and butter have been inspired into the larynx.[1] The same accident is apt to occur with epileptics, and from accidents, as when a man is thrown from horseback when smoking a pipe.

The blood effused in hæmoptysis may act as a foreign body, especially if the patient faints and is placed on his back instead of on his side.[2]

Foreign bodies are sometimes pushed through the nose and drop into the larynx. The accident may occur in surgical procedures, as when adenoid growths are removed in such a manner as to allow of their dropping into it. The use of throat brushes and instruments, with easily detachable extremities, is also fraught with this danger.

One of the most usual methods by which a foreign body enters the larynx is the following:—Some substance is introduced temporarily into the mouth, and, owing to the patient's attention being attracted elsewhere, is partly forgotten. Some unexpected cause initiates the deep inspiration which precedes a start, cry, laugh, or sneeze, and the foreign substance is drawn directly into the larynx.

The list of substances, in addition to articles of food, which may be met with, is too varied to attempt to make it complete; but the following have been found in the larynx—coins, pins, needles, buttons, various seeds (beans, peas, corn), toys, pieces of wood, portions of pipe-stalks and cigar-holders, and leeches. Bronchial glands have ulcerated through into the trachea, and been coughed up into the larynx.

When the substance is large it generally obstructs the *aditus ad laryngem*. Smaller bodies may rest on the ventricular bands, or get wedged between the vocal cords, sometimes with one edge in a ventricle of Morgagni. On deep inspiration, or injudicious attempts at removal, they may pass downwards into the trachea.

Symptoms.—When a large bolus of food or other foreign body completely blocks the laryngo-pharynx, death by asphyxia rapidly occurs unless relief is obtained. A barman was in the habit of showing how he could place a billiard ball in his mouth and close his lips over it. While performing the trick he was, of course, quite aphonic. On one occasion the billiard ball slipped into the lower pharynx, and his frantic signs for relief were regarded by the amused onlookers as part of his jest. He died, and the ball was found entirely blocking the upper larynx.

When the substance is small enough to enter the larynx, it produces dyspnœa and inspiratory stridor in proportion to its size and its situation over the glottis. If it gets between the cords it is apt to become grasped spasmodically, producing great anxiety. The trauma produced may lead to acute œdematous laryngitis (*q.v.*).

The cough induced is frequently extremely insistent, and will sometimes continue for some time after the foreign body is expelled.

Examination.—In all cases when the symptoms are not very urgent, the throat should be carefully examined before making any attempt at removal. A laryngeal inspection can generally be obtained by the use of cocaine and the infusion of a little confidence into the patient. The size, nature, and position of the body can thus be exactly observed. Sometimes, owing either to the thick strings of mucus which may extend from one side of the throat to the other, or to the translucent character of the foreign body (as fish-bones or glass beads), it remains invisible. In such cases the careful use of a probe will often assist in the examination, and it is only when these efforts at locating it have proved fruitless that it is justifiable to attempt to use the forefinger to detect the body. This latter proceeding, however, is frequently required in young children, in whom a laryngoscopic examination is not always possible.

If the presence of the foreign body in the upper air or food passages still remains doubtful, the power of swallowing should be tested and the chest carefully examined.

In many cases, particularly where metallic substances have been inspired into the larynx, the use of the Röntgen rays has proved of great service both in settling the presence of a foreign body and in determining its exact position.

When, in spite of careful examination, the situation of the foreign body cannot be diagnosed, but the suspicious symptoms still suggest its presence, the patient should be kept under observation, when, either by the

[1] Hale White, *Trans. Clin. Soc.*, Feb. 23, 1894.
[2] Bowles, *Brit. Med. Jour.*, July 23, 1898.

development of fresh symptoms or by the shifting of the substance, the diagnosis cannot be completed.

In many cases, probably the majority of those which present themselves, no trace of the offending substance can be found. The patient can then be reassured and the irritation remaining can be treated. In certain cases, in spite of the fact that the discomfort is referred by the sufferer to the larynx, the irritant will be found outside the larynx—in the sinus pyriformis, the base of the tongue, the tonsils, or elsewhere.

TREATMENT.—Once the presence of a foreign body in the larynx has been definitely detected, it ought never to be left there, even if it is causing no serious symptoms. When the foreign body is even suspected, no attempt at treatment should be made until the implements for a speedy tracheotomy are all at hand. Under the effect of cocaine, and with good illumination, intralaryngeal removal should then be attempted by one of the various forms of intralaryngeal forceps. This is much more scientific than the frequently recommended plans of inversion with slapping the back. The latter may be had recourse to when attempts at intralaryngeal extraction have failed, always provided that a tracheotomy can be performed on the spot if the foreign body should happen to shift from a harmless to a dangerous position, or suddenly produce threatening spasm.

When dyspnœa is marked, or other methods have failed, or the patient has to be left out of call of prompt relief if required, then the trachea should be opened. When in doubt, it is much safer to do the operation than to leave the patient with a foreign body in the larynx. The substance can be sought for from the tracheotomy wound, and may either be extracted through it or pushed up into the mouth and so removed. If impacted in the larynx, it can be removed by a subsequent thyrotomy.

### Laryngeal Hæmorrhage

This includes two separate pathological conditions.

(*a*) *Submucous hæmorrhage of the vocal cords* is a rare affection, and occurs chiefly in singers. It takes place suddenly, and the patient complains of hoarseness. Probably it is predisposed to by slight local catarrh. The coincidence of menstruation appears to be a predisposing factor. It has also followed sneezing, coughing, and attempts at topical applications.

Examination shows an effusion of blood below the mucous membrane, generally limited to one cord. Sometimes it appears as a small, round, dark-red cystic tumour. (For treatment, *vide* Chronic Laryngitis.)

(*b*) *Superficial Laryngeal Hæmorrhage.*—This is alarming, and often difficult to diagnose. It may be due to (*a*) acute inflammation and various forms of ulceration, (*b*) changes in the blood and blood-vessels, and (*c*) trauma following strangulation or slighter causes.

In the second group are angioma of the larynx, local varicose veins, and small varicose aneurysms, also cirrhosis of the liver, heart disease, albuminuria, diabetes, phthisis, malignant fevers, hæmophilia, purpura, leukæmia, anæmia, etc.

*Diagnosis* is made by the laryngoscope. Special attention must be paid to the nose, the region at the base of the tongue, and the trachea. In all cases where the bleeding point is not discoverable, examine for early indications of pulmonary tuberculosis (*q.v.*).

*Treatment.*—Local and general rest, and that generally indicated in hæmorrhage. Abstention from use of the voice, sucking of ice, and the administration of morphia will generally be sufficient. The application of astringents is of doubtful value, and apt to irritate.

### Congenital Glottic Stenosis

SYNONYMS: *Webs of the Larynx, Pseudo-membranous Stenosis, Diaphragms of the Larynx, Congenital Laryngeal Stenosis.*

The occurrence of congenital diaphragm in the larnyx is a rare condition. Semon has recently published a case which, from his researches, appears to be only the sixteenth placed on record. In 1893 Paul Bruns published an essay enumerating twelve cases of this class which he had found in literature, and adding one of his own. Single observations have been recorded by Chiari and Lacoarret.

ETIOLOGY.—It has sometimes been doubted if these webs are ever really congenital, and it is, of course, rather difficult to establish the point, as it is only when a child begins to acquire the faculty of speech that any defects of phonation become strikingly obvious. This matter, however, appears to have been settled by Seifert, who found that in a family of six persons no less than four showed more or less marked evidence of larnygeal webs. From a careful consideration of the history of these cases it was fairly clear that the affection is not only apt to be hereditary, but is also undoubtedly congenital.

SYMPTOMS.—If the laryngeal diaphragm is well marked the first symptom which it may cause will be stridor, chiefly inspiratory, and this will be noticeable at or soon after birth, and will be associated with other symptoms similar to those described under Congenital Laryngeal Stridor (p. 372). But the cry will be more or less hoarse, and when speech develops the voice will be harsh and weak. Dyspnœa on exertion, and inspiratory stridor, as well as the interfer-

ence with voice, will all be proportionate to the size and position of the web.

EXAMINATION.—The laryngoscope reveals the cause of the above symptoms. The diaphragm is nearly always limited to the anterior part of the glottic space. The rest of the larynx is usually normal. The web is seen to stretch across the anterior commissure from one vocal cord to the other. It is symmetrical, somewhat translucent and membranous-looking, and in some instances slightly pink; it is triangular in shape, with the apex at the junction of the cords. The base or free border is not quite rectilinear, but is generally curved, and the restricted glottic space lies between this border and the inter-arytenoid space. This edge, as a rule, is white, and appears to be thicker than the main part of the membrane. The membrane increases in thickness again as it approaches the anterior commissure. On phonation it is seen that the cords move freely, but their complete approximation anteriorly is prevented by the membrane, which becomes folded between them.

Slighter indications of such a membrane are frequently met with. In many cases it is foreshadowed only by a rounding of the ordinary acute angle at the anterior junction of the vocal cords; in other cases a small fold of membrane is seen in the subglottic region below the anterior commissure entirely unconnected with the cords, and apparently causing no symptoms. The thickness of the web increases from behind forwards.

In only one case—that of Chiari's—did the web occupy the posterior region of the larynx.

PATHOLOGY.—As to the pathological causation of these webs there is nothing in their appearance to suggest an inflammatory or pathological origin, and the source of the malformation has not yet been explained. There is nothing in the development of the larynx to throw light on the subject; but Roth has shown that the upper part of the air-tube in its first development is glued together, the epithelial gluing matter being formed in part from elements of the outer terminal layer (epiblast), and in part from the elements of the intestinal gland layer (hypoblast). Bruns finds in these observations a clue to the explanation of the occurrence of these laryngeal webs. The cause of their formation would seem to be traceable to the agglutination of the original formation being only incompletely loosened and persisting in part.

TREATMENT.—The treatment will depend entirely on the amount of interference with respiration. In the slighter forms, and particularly when there is no dyspnœa or stridor, and the voice is not greatly interfered with, the wisest plan is to leave the web strictly alone, warning the patient of his condition and of the greater precautions he should take in the event of laryngitis or other inflammation of the respiratory tract. When there is more or less complete aphonia, when there is stridor or dyspnœa, relief must be obtained by surgical measures. The most complete and radical method of removing the web would, at first sight, appear to be by splitting the thyroid cartilage (laryngo-fissure or thyrotomy), but it has been found that in these cases there is marked tendency for adhesion to take place after this operation between the raw anterior extremities of the cords, so that intralaryngeal treatment is required for some time afterwards to prevent an even worse form of stenosis. Before inaugurating treatment for the removal of the obstruction by intralaryngeal methods, it is well to bear in mind the fact that in several instances these webs have been found to be extremely tough; so much so that in Seifert's case[1] the intralaryngeal knife actually broke in the tough tissue, and Semon found it quite impossible to make an incision into the web. He was successful by first using the galvano-cautery to divide the web up into portions, which were later on removed by intralaryngeal forceps. Other cases have required careful dilatation by Schroetter's or other laryngeal dilators, and such cases appear to offer a useful field for O'Dwyer's method of intubation. Some cases have required tracheotomy, and then the stenosed portion has been attacked from below. (For further methods of treatment, *vide* p. 372.)

### Pachydermia Laryngis

DEFINITION.—This term has been applied to a form of hypertrophic laryngitis which has received considerable attention from the fact that its pathology has been fully investigated by Virchow. Possibly this has secured for the affection more attention than it warrants, for, as will be seen later on, it is but one clinical form of the hypertrophic variety of chronic laryngitis. In Virchow's original paper[2] he described two forms of pachydermia. One, in which the hypertrophy was limited to the anterior part of the vocal cords, he called pachydermia verrucosa or the warty form. To the second form he applied the term diffuse pachydermia. The former need not be considered; it hardly ever established itself as a pathological entity, and with the progress of laryngology it has disappeared. With regard to Virchow's second group, although its claim to a separate class may be doubted, it is still convenient to retain it as a form of hypertrophic laryngitis. It is a chronic affection characterised by more or less symmetrical thickenings over the posterior ends of the vocal cords and the neighbouring parts of the inter-arytenoid space.

SYMPTOMS.—As the disease is more particularly limited to the posterior part of the larynx and so does not interfere with the approximation of the greater portion of the cords, it causes a

[1] *Berlin. klin. Woch.*, 1886, No. 10.
[2] *Ibid.*, No. 32, 1887.

less degree of hoarseness in the earlier stages than a smaller growth would produce if situated more anteriorly; otherwise the symptoms are much the same as those of chronic laryngitis. In marked cases there is dyspnœa on exertion.

ETIOLOGY.—The disease is more common in men than in women, and usually occurs in middle life, from thirty to sixty years of age. It is frequently attributable to the same causes as catarrhal chronic laryngitis, and more particularly to excess in alcohol and in smoking. Still, cases of it do occur in which it is difficult to ascribe it to any of the ordinary causes of laryngitis, and it does not appear to be particularly attributable to over-use of the voice.

EXAMINATION.—The laryngoscope reveals an affection of the posterior ends of both vocal cords. Situated over the vocal process on one side is an even, elongated, pink or grey thickening, with a slight central depression facing towards the opposite side. On the opposite vocal process is another hypertrophy, generally somewhat smaller, and either with a blunt summit or with a smaller central depression, which, on phonation, is seen to fit into the cup-like depression on the opposite side. It was formerly thought that the depression on one side was caused by the pressure of the hypertrophy of the opposite vocal process. It is more likely that a close examination would reveal a depression on both sides, although more marked on the side with the larger hypertrophy, and that in both cases the dimpling in the centre is due to the closer attachment of the mucous membrane at that point to the subjacent cartilage. The hypertrophies are free from inflammation or ulceration; they are generally bathed in sticky mucus, which may stretch across in threads from one side to the other after the thickenings are pressed together on phonation, and then gape apart in respiration.

Occasionally the hypertrophy is limited to one side, in which case the opposite processus vocalis may be indented from pressure.

In some cases the rest of the larynx is normal, while in others there are the usual symptoms of chronic laryngitis. The movements of the cords are frequently somewhat impaired.

PATHOLOGY.—The hypertrophies are found to be formed of a white or grey-white thickening, which can be stripped off in layers and is found to consist of epithelium thickened and undergoing epidermoidal change. The subepithelial connective tissue is also thickened and sends upwards papilliform processes into the epithelial layer. All degrees may be met with, from a slight elevation due to some heaped-up epithelial cells, to an outgrowth of some size. Inflammatory changes may be observed in the thickened subepithelial connective tissue, but there is always a distinct line of demarcation between epithelium and connective tissue.

DIAGNOSIS.—In some cases the movement of one of the cords may be affected and the suspicion of malignant disease may be aroused. From epithelioma, and from other affections, pachydermia can be distinguished by the facts that it occurs in middle-aged males; by the history; by the slow growth; by the discovery of a similar condition—even if not so marked—on the opposite side; and by the crateriform depression. In doubtful cases the removal of a portion of the growth for microscopic examination may be advisable, although only positive evidence of cancer would be of any value (see also p. 359).

The diagnosis has sometimes to be made between simple pachydermia and that due to syphilis and tubercle.

PROGNOSIS.—The prognosis is favourable as regards life, and continued—if impaired—use of the voice can generally be promised.

The disease is a very chronic one, and not very amenable to treatment. There is no clinical evidence that it is apt to assume a malignant character.

TREATMENT.—The reader is referred to the sections on the treatment of chronic laryngitis—catarrhal and hypertrophic. The internal administration of iodide of potassium is generally recommended. Sprays or laryngeal washes of salt water are frequently of use. Painting with nitrate of silver in solutions of increasing strengths here finds its most suitable field of application. Iodine has failed in the hands of Stoerk and Gottstein. Sulphur spas may be tried, and painting with lactic acid or salicylic acid in alcohol may be tried. Electrolysis is recommended by Chiari.[1] Attempts to extirpate the growth are liable to set up perichondritis.

### Blennorrhœa

Under this title a particular condition has been described by Stoerk.[2] It is an affection which is extremely rare, if not unknown, in this country, but appears to be not infrequently met with in Poland, Wallachia, and neighbouring parts of Central Europe.

It is said to assume a form of chronic laryngitis, chiefly subglottic, with free secretion, and is sometimes followed by stenosis or adhesion between the anterior parts of the vocal cords. It may be accompanied by an analogous condition in the nose.

It is possible that the condition—references to which are seldom met with in literature—has been confused with chronic subglottic laryngitis and with rhinoscleroma (see also p. 346).

## Larynx, Chronic Infective Diseases

LARYNGEAL PHTHISIS . . . . . 342
*Manner of Infection in* . . . 342
*Symptoms* . . . . . 342

[1] *Archiv für Laryngol.*, Bd. ii. 1, 1894.
[2] *Klinik der Krankheiten des Kehlkopfes*, Halfte 1, Stuttgart, 1876.

LARYNGEAL PHTHISIS—*continued*
*Diagnosis* . . . . . 343
*Prognosis* . . . . . 343
*Treatment* . . . . . 343
LUPUS OF THE LARYNX . . . . 345
SYPHILIS OF THE LARYNX—
*Clinical Features* . . . 345
*Treatment* . . . . . 346
SCLEROMA . . . . . 346
LEPROSY . . . . . 347

### LARYNGEAL PHTHISIS

Laryngeal phthisis is commonly secondary to a similar condition of the lungs. It is still an open question how the infection of the windpipe occurs. According to some authorities it results from bacilli either entering the glands by way of the ducts or making their way through injured epithelium, while others maintain that these organisms may pass through healthy cells without leaving any trace of their passage. A third group of observers affirm that infection of the larynx usually results from the circulation, either vascular or lymphatic.

While in most cases laryngeal phthisis is associated with and dependent upon pulmonary disease, various authors (Demme, E. Fränkel, and others) have published undoubtedly authentic instances of primary tuberculosis of the larynx. The first morbid appearances result from tubercular infiltration, which involves both the mucous and the submucous layers, while the glands are usually also affected. Very rarely, as described by Schech and Heinze, tubercular deposits may occur in the laryngeal muscles.

In most if not all cases infiltration is followed sooner or later by ulceration. The resulting ulcers may be superficial or deep, and in the former case are often multiple. In certain instances laryngeal tuberculosis shows itself by the presence of tumours. Sometimes a circumscribed sessile neoplasm occurs in the inter-arytenoid space. Such growths are composed, according to Gouguenheim and Glover, of epithelium and connective tissue, with perhaps a layer of tubercular granulations in process of evolution; but these authors failed to detect bacilli. Again, more definitely tubercular growths may be present. These may be single or multiple, are usually pale in colour, and vary in size from a pea to a hazel-nut. Finally, Avellis has shown that a proportion of those growths which have been hitherto regarded as papillomata and fibromata are in reality tuberculous.

Like all laryngeal affections, phthisis is more common in males than in females, while the period of life which statistics have shown to be most liable to attack is between the ages of twenty and forty.

So far I have referred merely to the lesions which occur after infection has taken place, but there can be no doubt that there are certain changes in the larynx which, when they exist, ought to warn the practitioner to make a careful examination of the lungs. It is no uncommon thing to find at an extremely early stage of pulmonary phthisis various signs and symptoms which lead the patient to consult a throat specialist rather than a physician. Thus the larynx may be the seat of abnormal sensations, and examination will then often reveal very marked anæmia of the mucosa, which may perhaps flush during examination. Again, there may be paresis of adduction, often also associated with pallor. These conditions cannot as yet be said to have any recognised pathology. On the borderland between these on the one hand, and conditions obviously due to infection on the other, we must consider the very obstinate laryngeal catarrh which is sometimes a precursor of tubercular infection of the part—if, indeed, it be not in certain cases an early manifestation of such infection.

*Symptoms.*—In considering the semeiology of laryngeal phthisis it has seemed to me better to give the symptoms of each clinical form which the practitioner is likely to meet with, as they vary according to the seat of the infiltration or ulceration.

Perhaps, on the whole, interference with the voice is the commonest symptom. This varies in degree from slight hoarseness to complete aphonia. In certain cases, too, vocal effort is accompanied by pain. Various localisations of laryngeal phthisis may produce the above symptoms.

As before said, there is a form of laryngeal catarrh which occurs in phthisical persons, and which may eventually be followed by infiltration and breaking down of tissue. In many instances there are no objective points by which it can be distinguished from the non-tubercular form. In other cases suspicion may be aroused by a tendency to a localisation; thus the occurrence of redness confined to one vocal cord is suspicious. Again, ordinary catarrh is not usually accompanied by any marked thickening, which if present in a phthisical subject will usually be found to be tubercular. At a later stage of the disease one or both cords may become distinctly infiltrated. The infiltration is usually of a red colour, and may result in uniform enlargement; on the other hand, it may be more or less circumscribed. Occasionally infiltration may occur at the anterior commissure or just below it, but this is a somewhat rare appearance. Much more common is a distinct flat tumour occupying the inter-arytenoid region. This is sessile, usually of a red colour, and may have a relatively well-defined regular surface, or may be uneven and papillary. These inter-arytenoid tumours may precede all other manifestations of phthisis, and much importance was attached to them by Stoerk,

who considered them infallible indications of a tubercular taint.

After a time infiltrations such as have been described tend to break down, and ulceration results. In the vocal cords various appearances may be produced. Sometimes small losses of substance occur at the edges, and these are separated from each other by red infiltrated tissue, which may appear like granulations. Occasionally the free edge becomes distinctly serrated, while not uncommonly a relatively deep excavation occurs in the neighbourhood of the vocal process. A peculiar appearance is sometimes produced by an ulcer extending along the cord; in such cases this part looks as if doubled or cleft longitudinally.

When the ulceration occurs on the posterior laryngeal wall the surface of the ulcer is usually not well seen; but the raised upper margin, often with a ragged outline, can be distinguished. Ulcers in this situation may interfere little with the voice, but sometimes give rise to most distressing cough.

Infiltration of the false cords may occur without any prominent symptoms, unless the parts be sufficiently enlarged to interfere with the movements of the vocal cords. When breaking down occurs, numerous small and often superficial ulcers may result, or several softened areas may coalesce, and lead to a deeper loss of substance.

Pain, either spontaneous or associated with phonation and deglutition, with or without huskiness, may be present when the epiglottis and aryepiglottic folds become involved.

Infiltration of the epiglottis usually shows itself by very marked and more or less uniform thickening of the part, which may be red in colour, but which is frequently of a bluish-grey tint. Associated with the enlargement there is usually interference with mobility. Ulcers, when they occur in this situation, are usually multiple and superficial, although at times deeper destruction of tissue may occur.

A very common lesion in phthisis is infiltration of the aryepiglottic folds; these appear much enlarged, and in the region of the arytenoid cartilages present pale pyriform œdematous-looking tumours. After a time small scattered ulcers may appear, but it is not common to see deep losses of substance in this situation.

It will be obvious from what has just been said, that hoarseness and pain are the two prominent symptoms produced by laryngeal phthisis. The former is liable to occur when the vocal cords are attacked, and may also be sometimes present when the ventricular bands are much enlarged.

When the epiglottis and aryepiglottic folds are the chief seats of disease, pain becomes a frequent symptom. In slight cases this is only noticed on speaking or swallowing, but in aggravated instances deglutition may become well-nigh impossible. Not infrequently it is complained of as shooting up to the ear. When more or less fixation of the arytenoid cartilages has resulted, marked huskiness and even aphonia may be present.

As the disease advances, the whole larynx is liable to become attacked, and at this stage the secretion from the various ulcers may be so great as to cover the parts and prevent any detailed diagnosis as to their condition. In the later stages, too, perichondritis and diffuse general swelling may lead to interference with respiration. In a previous paragraph tubercular tumours have been referred to; their presence can be diagnosed by means of the laryngoscope, but it is questionable whether we possess any data by which we can distinguish their nature, short of removal followed by microscopic examination. Of the symptoms liable to be caused, huskiness is the most prominent. These neoplasms are rarely large enough to interfere with respiration, and do not seem ever to give rise to pain.

*The diagnosis* of laryngeal phthisis is, as a rule, not very difficult. Perhaps the most common appearance met with is the pyriform swelling of the aryepiglottic folds. The pale colour and characteristic shape make this form readily recognisable. A localised sessile tumour in the inter-arytenoid region is suggestive of tuberculosis; but syphilitic ulcers occasionally occur in this situation, and the upper margin may become the seat of granulations which conceal the ulcer from view. When the epiglottis is infiltrated the pale colour and turban shape are very characteristic, and unlikely to be mistaken. Again, the eaten-out edges of the vocal cords and the longitudinal ulceration, giving rise to an appearance of cleavage, are strongly suggestive of phthisis. Finally, the presence of pulmonary disease and the existence of tubercle bacilli in the sputum will in most cases give corroborative evidence.

In the early stages the presence of localised redness and swelling may afford grounds for suspicion, but not for a definite diagnosis. In cases of laryngeal phthisis the pharynx is often markedly anæmic, and sometimes the unusually pale mucosa is relieved by dilated vessels coursing over it—an appearance very suggestive of threatened or actual tuberculosis. I have not referred to injections of tuberculin as of value in diagnosis, since they can hardly be considered justifiable after past experience.

*The prognosis* of laryngeal phthisis is extremely grave, but it must not be forgotten that many cases of cure have been recorded. The prospect for the larynx is better according as the lung lesion is slight or improving, and *vice versâ*. Moreover, much can be done by treatment to retard even if it does not cure.

*Treatment.*—In considering the therapeutics of laryngeal phthisis I shall refer merely to

those points which relate to the special condition, leaving it to be understood that suitable general treatment must be carried out.

One of the first questions that usually confronts us in connection with phthisis is whether the patient should be sent to winter abroad. In selecting a climate for a case of laryngeal phthisis, as a rule, three points should be considered, viz.: (1) warmth and sunshine; (2) the air should have a degree of humidity; (3) there should be little dust. As examples of places fulfilling these indications may be mentioned Madeira, Pisa, Capri, and the Canary Islands. It was formerly supposed that high altitudes were always contraindicated where the larynx had become involved, but, as pointed out by Clinton Wagner, this is not always the case. The effect, however, should be carefully watched if the experiment be tried. Under certain circumstances it may be desirable to place the patient in one of the institutions which are now springing up in this country, and which have long existed in Germany, *e.g.* Falkenstein, Reiboldsgrün, Görbersdorf, where the open-air treatment is carried out on scientific lines, and where the necessary local remedies can also be employed.

As to general hygiene and regimen — the voice should be saved as much as possible, and a nourishing, wholesome diet given, while smoking should be altogether forbidden indoors. At the same time, a cigar or pipe smoked in the open air may usually be permitted. When dysphagia is a marked symptom the administration of all nourishment often becomes difficult. In such cases it will be found that soups (thickened with arrowroot or an equivalent), raw eggs and milk, calf-foot jelly, and sometimes ices, will be acceptable. A little culinary ingenuity will suggest food of a suitable consistence, *i.e.* semisolid, and of sufficient variety, while where cold is well borne nutritious materials may be incorporated with ices. Wolfenden found that patients in whom dysphagia is severe may be enabled to swallow in comparative comfort by lying on the face with the head over a bed or couch, and sucking up food from a basin held below the mouth and connected with it by a tube.

It is sometimes, however, necessary to resort to local anæsthetics, such as cocaine, 10 to 20 per cent applied with a spray or brush, and eucaine. A solution of from 20 to 30 per cent of menthol in paroleine may also be employed for this purpose, by syringing it into the larynx. When painful deglutition is due to ulceration, orthoform blown on to the affected part is often very successful, the anæsthesia sometimes lasting for many hours.

Local treatment applied to the larynx with the object of arresting or curing the disease should be adapted to the condition of the patient. Thus, if the pulmonary disease be advanced and the patient weak and emaciated, it is well to avoid all active treatment of a painful kind. Volatile substances, such as balsam of Peru, may then be employed, added to hot water and the steam inhaled, or a spray of from 5 to 20 per cent of menthol in paroleine may be used. As pointed out by Rosenberg, however, this drug is best used by means of a syringe with which a drachm or more of a 20 per cent solution in oil may be injected into the larynx. The insufflation of antiseptic powders which may be mixed with orthoform or morphia, if these be indicated by the presence of pain, may be employed in persons who are no longer sufficiently robust to tolerate more active treatment. Equal parts of boracic and iodoform have been recommended, and Lublinski has had satisfactory results from iodol. There can, however, be no doubt that lactic acid, as first suggested by Krause, is the best local remedy we possess.

Its great efficacy in ulceration is admitted by all, but there can be no doubt that it is also beneficial in infiltrations. Lactic acid should be first used in 20 per cent solution, but gradually this should be strengthened, until, if well tolerated, it is employed in a strength of 80 per cent. It is best applied by means of a cotton-wool holder, and should be rubbed well into the parts. As a general rule, when the stronger solutions are used several days should elapse between the applications.

Various other active remedies have been suggested, and no doubt in some cases they may act well—*e.g.* sulforicinic solution of carbolic, 30 per cent (Ruault); oleaginous solution of creasote and menthol (Chappell); para-ortho and mono-chlorophenol (Simanowski, Spengler, and Hedderick), from 5 to 20 per cent dissolved in glycerine; and concentrated solution of iodoform in equal parts of alcohol and ether, recommended by Newman as a spray.

Of late years surgical treatment of laryngeal phthisis has come much into vogue and has given excellent results, especially in the hands of Continental authorities. It goes without saying that operative measures are only justifiable in patients whose strength has been well maintained and in whom the pulmonary disease is not actively advancing. The objects aimed at are (1) the removal of infiltrations; (2) curetting ulcerated surfaces. Heryng may justly claim to have been a pioneer in this matter, and his instruments, together with the double curette of Krause, are generally employed. Some laryngologists, however, use the electric cautery, and even electrolysis. Schmidt formerly recommended multiple incisions followed by the application of lactic acid, while he was also an advocate of tracheotomy in certain cases. Submucous injections of lactic acid, creasote diluted with oil, etc., have also been advocated. In suitable cases, how-

ever, curetting soft tissue and ulcerated surfaces, together with the removal by means of the double curette of infiltrations, seem to have given the best results—more particularly when these operations were followed by applications of lactic acid.

In the case of tubercular tumours without infiltration the removal of the neoplasm is indicated, and must be carried out by one of the methods discussed in another portion of this work.

### Lupus of the Larynx

As lupus is in a sense a form of tuberculosis, the reader may pertinently ask why it is discussed under a separate heading. The reply lies in the clinical differences which exist between what we may term true tuberculosis and the form known as lupus.

It was formerly held that lupus of the larynx is usually secondary to lupus of the skin of the face. My own experience has shown me that it is by no means uncommon to find the affection confined to the mucous membrane of the nose and throat, while sometimes it develops only in the larynx.

The disease certainly attacks by preference young females, but it may occur both in boys and in men of early middle age.

Lupus of the larynx produces extremely slight symptoms, and, indeed, may cause none unless the infiltration be so situated as to interfere with phonation, or much more rarely, respiration.

The part most frequently affected is the epiglottis, but the characteristic infiltration may appear on other parts as well, *e.g.* the ary-epiglottic folds, posterior wall, ventricular bands, and true cords. On examination the parts affected are seen to be thickened and nodular. Individual nodules vary in size from a pin-head to several times as large; they are situated close together, so that the whole affected area is studded with them. It is stated by most authors that after a time slow ulceration sets in followed by cicatrisation, and that sometimes fresh nodules appear on the surface of such cicatrices. My own impression is that it is extremely doubtful whether there is in lupus of mucous membranes any tendency towards breaking down by breach of surface.

If the part be so situated as to be exposed to injury, then of course infection and surface ulceration may result.

*The diagnosis* is not as a rule difficult. The nodular character of the infiltration (which is usually of a bright red colour), the absence of any pronounced tendency to ulceration, and the painless, slow course are more or less pathognomonic.

*The prognosis* of this affection is somewhat uncertain. While some cases seem to be readily checked for a time at least, or even cured, by general and local treatment, others are most obstinate. The course of the malady is always a slow one, but gradually it may lead to loss of voice and even to dyspnœa.

The chief indication for *treatment* is to remove the diseased tissue or destroy it with the electric cautery. For the former purpose the instruments already referred to in discussing laryngeal phthisis may be employed. The cautery, however, sometimes acts beneficially, not only upon the part burned, but upon surrounding nodules. Lactic acid, too, I have found very serviceable. Together with local remedies general treatment must be prescribed. Fresh air, milk, arsenic, and cod-liver oil are specially indicated.

I am not sure that in obstinate cases of laryngeal lupus it might not be justifiable to resort to Koch's original tuberculin. In one of my cases treated by this method rapid cure resulted, although a considerable degree of laryngeal stenosis resulted, and this had to be treated by dilatation.

### Syphilis of the Larynx

The larynx is more commonly affected by acquired than by inherited syphilis, and it has been shown by statistics that syphilitic males are more prone to be attacked than females.

*Clinical Features.*—As in other parts, so in the larynx, the disease may appear in many forms.

Syphilitic catarrh, while commonly an early symptom, may recur at later periods throughout the disease. The appearances are rarely distinctive, although sometimes there is a patchy redness which is highly suggestive.

Much more characteristic is the presence of mucous patches, which appear as whitish areas on various parts of the larynx, and may result in superficial ulceration. Very rarely true condylomata may be found in the larynx, and Gerhardt quotes a case observed by Heymann in which they were so numerous as to cause marked dyspnœa. As a rule, these earlier manifestations give rise only to huskiness and some local discomfort.

Gummata frequently occur in the larynx, but as a general rule they are not observed until ulceration has set in. Three forms are usually described, viz.: (1) relatively large rounded growths; (2) groups of small nodules; (3) diffuse infiltration. They cause symptoms in proportion to their size and situation.

Commonly they tend to break down rapidly and give rise to deep ulceration. The margins of the ulcer are generally raised, while the floor is of a whitish colour owing to its being covered with detritus. The mucosa immediately surrounding it is red and swollen. These tertiary ulcers have a great tendency to spread both in width and in depth. The epiglottis is often attacked, and frequently destruction of the whole, or at least of a large portion, of

this organ results. Again, the true and false cords may be the seat of disease, and one or both sides of the larynx may be extensively destroyed.

Tertiary ulcers of the larynx invariably leave more or less marked changes after they heal. Thus in extreme cases great narrowing of the lumen of the glottis may occur, while the various parts of the larynx are so altered as to be hardly distinguishable. Again, not uncommonly a cicatricial web occludes more or less of the space between the cords, which may themselves be so changed as to be recognised with difficulty. Sometimes, as a result of ulcers near the arytenoid cartilages, these become fixed, and an appearance is produced which by the casual observer might be taken for recurrent paralysis.

When, instead of healing, ulceration tends to extend, perichondritis may result, and occasionally œdema supervenes and renders a rapid resort to tracheotomy necessary. The symptoms of laryngeal syphilis are, as a rule, not very marked. In the catarrhal stage, huskiness and mere discomfort only are experienced. Very rarely, as we have seen, condylomata may occur and lead to dyspnœa during the second period. Gummata may, according to their situation, lead to hoarseness or dyspnœa. When ulceration has become established more or less pain may be complained of, the breath becomes fœtid, and hoarseness is often pronounced. If the epiglottis only be involved, difficulty in swallowing may be a marked symptom; but even the total disappearance of this part does not, *per se*, cause any permanent interference with deglutition.

*The diagnosis* of laryngeal syphilis is usually easy if other evidences of the disease be present, *e.g.* cutaneous, lymphatic, buccal, or pharyngeal. As we have seen, specific catarrh may not have any distinctive characters. Mucous patches may be simulated by herpes, but more particularly by pemphigus after the blebs have burst. It is, therefore, not a simple matter to diagnose even the earlier forms unless we have a definite history or other manifestations. The same difficulty confronts us more frequently with regard to tertiary conditions. As a general rule gummatous infiltrations are of a red colour, while frequently tubercular deposits are pale. When the stage of ulceration has been reached, therefore, the syphilitic ulcer is surrounded by a raised angry red zone. Moreover, it is often single, its floor is covered with whitish detritus, and it spreads with great rapidity if no constitutional treatment be adopted. Occasionally both in tubercle and syphilis an ulcer is found in the inter-arytenoid fold. So far as I know, the appearances are identical, and diagnosis must then depend upon the condition of the lungs, the presence or absence of tubercle bacilli in the sputum, and of other evidences of syphilis. On the other hand, extensive deep ulceration of the epiglottis spreading from its lingual surface is usually specific. Infiltrations which have begun to break down may also be mistaken for malignant disease, and in some cases only the effect of antisyphilitic remedies can determine the true nature of the affection.

Primary lupus of the larynx differs from syphilis in that the infiltration is uniformly nodular and does not tend to break down—or, at least, if ulceration occurs, its progress is excessively slow. Moreover, the patients are commonly young persons, although not always. Leprosy only occurs in the larynx as part of the general disease, while in scleroma there is no tendency to ulceration.

*The treatment* of laryngeal syphilis must, of course, be constitutional. Mercury by the mouth or by inunction should be employed in the secondary stages, while in the tertiary period our main reliance must be placed upon iodide of potassium. It is, however, well to remember that sometimes even in late manifestations, a course of mercurial inunction, either at home, or preferably at Aix-la-Chapelle if means permit, may expedite a cure.

If syphilitic catarrh be obstinate, it may be treated by the local application of solutions of chloride of zinc or nitrate of silver (gr. 20 ad ℥j). When ulceration has occurred a spray of corrosive (1 in 2000), of boracic (gr. 10 ad ℥j), or of menthol in paroleine (5 per cent) may be ordered. If ulcers refuse to heal, they may be touched with nitrate of silver or chronic acid.

It is of great consequence, when extensive destruction of tissue has occurred, to prevent as far as may be subsequent stenosis. It may therefore be necessary during this period to dilate the larynx with Schroetter's bougies, or possibly the introduction of an O'Dwyer's tube may be indicated. If the case be only seen after stenosis has occurred, it must be treated according to the rules laid down in another part of this work.

### Scleroma of the Larynx

This disease, probably due to the bacillus discovered by Frisch, rarely if ever occurs in the British Isles; indeed, it seems to be confined to certain well-defined areas. It is common in the south-west of Russia and adjacent provinces over the borders; it has also been met with in Central America and the Antilles.

In the larynx the subglottic space is most frequently attacked. As a rule, two symmetrical hard swellings appear below the cords; in colour they are grey or pink, while when touched with a probe they are felt to be of very firm consistence. Infiltration may, however, involve the aryepiglottic folds and

ventricular bands, and occasionally other parts also.

Ulceration does not seem to occur, but gradual cicatrisation may take place.

*The symptoms* vary according to the parts affected. Thus huskiness may occur first in one case and dyspnœa in another. The last named will, however, sooner or later set in, as even if the laryngeal stricture be dilated, the disease tends to spread to the trachea and finally to the bronchi.

*The only treatment* which seems to avail is surgical interference. Thus persistent dilatation may prevent the occurrence of dangerous stenosis for a time at least, while at a later stage tracheotomy may be required. Pawlowsky has advocated the employment of a liquid prepared from the bacillus, which he terms rhinosclerin.

### Leprosy of the Larynx

This disease only attacks the larynx after it has already become manifest in other parts.

Infiltration may occur in any position, although the epiglottis is stated to suffer most frequently. The aryepiglottic folds are also often involved, and by their traction produce further changes in the shape and position of the epiglottis. According to Bergengrün the part is drawn backwards, its edges are turned in, and the contour resembles the Greek omega (Ω). At a later period more circumscribed granulomata occur in various parts. These vary much in shape, size, and consistence. Thus they may be smooth or granular, sometimes even resembling papillomata; they may be only as large as a pin-head, but have been met with up to the size of a pigeon's egg. According to Bergengrun they are always anæsthetic. These leprous nodules seem to have usually a more or less pale colour, while although firm at first they become softer as time goes on. As a rule, the vocal cords seem to escape for a time; but if the patient survives, they become first infiltrated and they later develop nodules.

As the disease advances ulceration sets in; the ulcers may be deep and spread rapidly, or superficial, while sometimes clefts and furrows occur in the infiltrated parts. Finally, cicatrisation may occur at parts although the disease progresses elsewhere.

The more important symptoms are, as would be expected, huskiness in the early stages and dyspnœa later.

As the general disease is always present this facilitates diagnosis. The chief distinctive points in the local lesions are—(1) the very slow progress; (2) anæsthesia; while in doubtful cases the bacillus lepræ may be discovered in a removed fragment.

The treatment must be purely palliative, and tracheotomy may become necessary.

## Benign Growths of Larynx


Introductory . . . . . 347
Varieties of . . . . . 347
Etiology . . . . . 349
Clinical Features . . . . 349
Diagnosis . . . . . 349
Course and Prognosis . . . 350
Treatment . . . . . 350


The opinion has been often expressed that benign growths of the larynx are met with less frequently now than in the early days of laryngology, and it has been suggested that this is due to those throat ailments which favour the formation of neoplasms, now receiving earlier and more efficient treatment. Leaving aside the fact that we do not know what "throat ailments favour the formation of neoplasms," we are not aware of any reliable evidence showing that the frequency of simple growths of the larynx has diminished in this country. When we remember that Mackenzie, when he was almost the only worker in the field, took ten years to collect his first hundred cases, that these cases are now divided among many workers throughout the country, and, further, that the majority are never published, the alleged diminution will at least appear doubtful. Still, there is no doubt that simple laryngeal growths, if we except the so-called "singers' nodule," are comparatively rare. Newman gives the percentage as from 2 to 2½ of all chronic laryngeal diseases, and Lennox Browne puts it at 2·5 per cent of all diseases of the larynx.

Of the many varieties of new growths which have been met with in the larnyx only three are of frequent occurrence—papilloma, fibroma, and cystoma. All the others, such as lypoma, angioma, chondroma, adenoma, myxoma, lymphoma, and thyroid gland-tissue tumours, are very rare. It is usual to describe the so-called "singers' nodule" as a distinct clinical variety of new growth, since its histological structure varies in different specimens.

Papilloma.—From Semon's collective investigation statistics this has been proved to be the commonest form of laryngeal growth, though many observers have found fibromata to form a much larger proportion of their cases. It may be single or multiple, and may grow from any part of the larynx, though most frequently from the vocal cords, rarely from the epiglottis, and hardly ever, even in the multiple papillomata of children, from the inter-arytenoid region. The growths may be broad-based, flat, and firm, but more frequently are more or less pedunculated, cauliflower-like masses, of softer consistence. They vary in size from a millet seed to a walnut, and may be white, pink, or red in colour. They are, as a rule, easily recognised by their distinctly irregular warty surface.

Fibroma.—This is almost invariably a single growth, with smooth surface, rounded or semi-

globular in shape, occasionally lobulated, often distinctly pedunculated, but more frequently sessile, and may be greyish-white, pink, or dark-red in colour. The consistence of fibromata varies from a hard nodule to a soft, cystic-looking growth. Histologically they consist of connective tissue and elastic fibres, with a covering of epithelium, and in the softer varieties are found cavernous blood spaces, serous infiltrations, and hæmorrhages. In the vast majority of cases the growth arises from the edge of one of the cords; sometimes from the upper surface or anterior commissure; rarely from the ventricular bands, aryepiglottic folds, or epiglottis. In two cases only have they been seen to originate from the inter-arytenoid region. At times the pedicle is long and thin, so that the growth drops beneath the cords on inspiration, and is thrown on to their upper surface in forced expiration. They may be of minute size (singers' nodule), or large enough to fill the cavity of the larynx.

This form of growth can usually be easily recognised by its smooth surface, and its origin from a vocal cord. At times, however, it is difficult to distinguish a small soft fibroma from a cyst.

Cystoma. — This form of growth, though much rarer than the two former, is by no means uncommon. Its most frequent situation is the anterior surface of the epiglottis, where it is often overlooked, as it may not give rise to any symptoms. Cysts may also originate, though rarely, from the edges of the cords. It is probable that most cases recorded in this situation were really fibromata which had undergone cystic degeneration. They have also been seen to grow from the ventricles, the aryepiglottic folds, and the posterior wall of the larynx. They may be broad based or pedunculated, and are smooth, globular, semi-transparent growths, of greyish-pink, yellowish, or red colour. They are of the nature of retention cysts, and arise from obstruction of the ducts of the mucous glands. Jurasz has suggested that, in those at the base of the tongue, the obstruction is probably caused by particles of food, as in this situation there is rarely any evidence of inflammation.

A cyst can generally be recognised by its globular shape and translucent appearance, with the distended vessels coursing over its surface. But often its true nature is only discovered on attempted removal. Small cysts on the vocal cords can only be distinguished from degenerated fibromata by microscopical examination.

Singers' Nodule. — This term has been applied to minute growths which often form on the edge, or upper surface, of one or both cords in singers, especially tenors and sopranos, and in female teachers. The name, however, is an unfortunate one, as they are also to be seen at times in children, and in persons who do not use their voices professionally. They are distinguished clinically by their minute size, and their situation at the junction of the anterior and middle thirds of the vocal cords. Histologically these growths may be minute fibromata, or cysts, or simply epithelial thickenings. Occasionally there is only one nodule, but more frequently there are two seated symmetrically at the point mentioned, or there may be three or four along the edge of one cord. They are seldom larger than a pin-head, and are greyish-white or pink in colour.

In a very small proportion of cases these nodules have been found to be cystic, but the great majority are simply inflammatory thickenings, and should rather be considered as a variety of pachyderma laryngis than as true new growths.

Lipoma.—Of this rare form of growth only ten cases have been recorded. It is usually a large solitary growth, filling the entrance to the larynx, and more or less obstructing both breathing and swallowing. The tumour may be smooth, lobulated, or branched, of pale pink or yellowish colour, elastic consistence, broad based or pedunculated, and freely movable. They have been observed to grow from the epiglottis, aryepiglottic folds, and posterior wall of the larynx.

Seifert is of opinion that if we find a large, pale pink, slow-growing tumour at the entrance to the larynx, we may conclude it is a lipoma.

Angioma. — Of this form of growth under twenty cases are on record. It is usually a single, sessile growth, very rarely pedunculated, varying in size from a lentil to a cherry, and of a bright or dark red colour. Its commonest situation is on the vocal cords, but it has also been seen on the ventricular bands, in the ventricles, and on the aryepiglottic folds.

The appearance of the growth is unmistakable, and Browne has noted as characteristic that the colour of the same growth varies at different times from white or pale pink to florid red.

Myxoma.—Considerable doubt exists as to whether a true myxoma ever occurs in the larynx. It is probable that the growths described as myxomata were really degenerated fibromata, as held by Eppinger.

In the cases recorded the growth originated almost invariably from the cords, was of a grey or pink colour, pedunculated or sessile, of a jelly-like transparency, and varying in size from a pea to a cherry. In some the surface was mammillated, and the growth looked like a papilloma.

Chondroma. — Cartilaginous tumours are rarely met with in the larynx, as only about fifteen cases have been recorded. They may grow from any of the laryngeal cartilages, but most commonly from the cricoid. They are hard sessile growths, flat or irregular in outline, and covered by normal mucous membrane.

They are usually solitary, rarely multiple, and tend to grow into the cavity of the larynx. They may be distinguished by their intense hardness, slow growth, and the absence of inflammatory symptoms.

Among the exceedingly rare growths which have been met with in the larynx are *lymphoma*, *adenoma*, and *thyroid gland tumours*, and in one instance a growth removed by Schroetter was found composed of *muscle tissue*.

Etiology.—We are still as far as ever from settling the question of the *cause* of laryngeal growths. That papillomata, cysts, and epithelial thickenings are occasionally congenital, is beyond doubt. Inheritance and constitutional predisposition have been suggested, in explanation of cases where several members of the same family have suffered, and it has been held (Oertel) that, in the case of papillomata, scrofula and hereditary syphilis are important factors. The vast majority of observers are agreed that chronic laryngeal congestion is the most frequent cause of benign neoplasms, and consequently chronic catarrh, excessive or wrong use of the vocal organs, the inhaling of dust-ladened air, smoking, the abuse of alcohol, and the acute infectious diseases, have all been held responsible for their occurrence. Schech, Jurasz, and others have actually seen new growths to arise during a chronic laryngitis. On the other hand, Schroetter and Semon have not found this view supported by their own cases, and regard the congestion as rather the result of the presence of a growth than the cause. It is rather curious that syphilis and tubercle, two of the commonest causes of laryngeal congestion, should be so generally held to have no bearing on the occurrence of true neoplasms. I have notes of two cases, one of multiple papillomata, and another of fibroma, in which the growths appeared while the patients were suffering from chronic laryngitis of syphilitic origin. That nasal obstruction may favour the occurrence of growths is quite probable; but that the removal of tonsils and adenoids, as has been suggested, will cause the growths to disappear, I do not believe, after seeing cases in which these measures have been adopted.

Beyond all question *age* and *sex* are the two most important etiological factors. At all ages males are more subject than females in the proportion of three to one. As to age, if we omit the first years of life, there is a steady increase in frequency up to the age of 40, followed by a decline as age advances. By far the largest number of cases occur between 30 and 40 years of age, while the period 20 to 50 may be said to be that within which there is a liability to benign growths of the larynx.

*The symptoms* produced by a laryngeal growth will depend on its size, its situation, and the nature of its attachment. In 95 per cent of cases it is hoarseness, or aphonia, which causes the patient to seek advice. This arises from the fact that the vast majority of growths originate from the vocal cords. A very small growth on the edge of a cord will cause hoarseness, and one at the anterior commissure may produce complete aphonia, while a growth with a broad attachment will almost certainly disturb the voice more than one with a narrow pedicle. Diphthonia, a rare form of vocal disturbance, was first described by Turck. It occurs where a growth on the edge of one cord divides the glottis into two unequal portions, and consequently two notes of different pitch are heard together.

*Dyspnœa* is present in about one-third of all cases, and is most common in multiple papillomata and in large growths such as lipomata.

*Dysphagia* is very rare, and only occurs in large growths at the entrance to the larynx.

A feeling of something obstructing the larynx, and giving rise to a frequent desire to clear the throat, is not unusual, but cough is a rare symptom. At times, however, it is severe and paroxysmal, when the growth has a long pedicle which allows of its free movement.

In a unique case, reported by Sommerbrodt, severe epileptic seizures, which had resisted all treatment, were cured by the removal of a large fibroma.

There is never any pain complained of in simple growths, and the general health is unaffected, except in cases of severe cough or dyspnœa.

Diagnosis. — Though we may suspect the presence of a laryngeal growth from the symptoms, the only certain method of diagnosis is by a laryngoscopic examination. As a rule this will not only reveal the presence of a growth, but also enable us at once to determine its character.

Only the very inexperienced will mistake the excrescences around a tubercular or syphilitic ulcer for a new growth. The warty growth in the inter-arytenoid space, so common in laryngeal phthisis, can hardly be mistaken for papilloma, as in this situation papillomata are practically never found. At the anterior commissure, however, I have known a tubercular tumour to be indistinguishable from a simple growth, till the microscope revealed its true nature. Such cases, however, are rare; but a difficulty will more frequently arise in distinguishing a simple from a malignant papilloma at an early stage. The importance of this subject demands a fuller statement, and attention to the following points will assist us in making a diagnosis:—

1. Age of patient.—Simple growths rarely originate after fifty years of age, therefore the presumption is strongly in favour of malignancy, if the growth is recent in a patient over that age. Under forty, malignant disease of the larynx is exceedingly rare.

2. Situation of the growth.—Simple growths, except multiple papillomata, are confined to the anterior two-thirds of the vocal cords in the vast majority of cases, and practically never occur in the inter-arytenoid region. A single growth on the aryepiglottic folds, epiglottis, or neighbourhood of the vocal processes, especially in patients over forty-five, is strongly suspicious of malignant disease.

3. Simple growths appear to *grow out of*, malignant growths to *invade*, the parts in which they are situated.—This is a sign of great value to the practised eye.

4. A malignant growth has generally an inflamed base, or the whole cord on which it is situated may be hyperæmic.

5. Any interference with movement of the cord on which the growth is situated, not due to purely mechanical causes, will be strongly presumptive of malignancy.

6. The tendency to ulceration of malignant growths, even at an early stage, will often settle the diagnosis.

Course and Prognosis.—The natural history of a simple laryngeal growth will depend principally on its character. Fibromata, after attaining a certain size, generally remain stationary for years, though very rarely they continue to grow till they come to obstruct the breathing. Papillomata may be divided into two kinds: in the one there does not appear to be any tendency to rapid growth, even when multiple; in the other they appear to have almost a malignant character, recur rapidly when removed, and spread down the trachea, to the edges of the tracheotomy wound, and along the cicatrix left after a thyrotomy. The latter form is mostly seen in young children, the former in adults.

From the small number of cases of papilloma seen between the ages of ten and twenty, I am of opinion that in the larynx as elsewhere, these growths tend to disappear at puberty, and this view has been confirmed by my own observations as well as by others. That they may also remain from childhood throughout adult life is shown by a case which I saw some years ago. A gentleman, aged sixty-three, had lost his voice at the age of ten after an attack of measles. He had gone through life practically voiceless, and without ever having his larynx examined. I removed a large number of papillomata from the edges of the cords and anterior commissure, which have shown no tendency to recur.

Papillomata have been noticed in a few instances to disappear after attacks of acute infectious disease, and many times after tracheotomy. Laryngeal growths have also been coughed up, or have sloughed off through the pedicle becoming twisted. On one occasion a lady, from whom I removed a large fibroma growing from the ventricle, brought me a similar growth which she had coughed up four years previously, and had preserved in spirit.

The only danger to life arises from the sudden onset of asphyxia, and although this can generally be prevented by a timely tracheotomy, I know of several cases among the children of the poor where death has resulted from suffocation.

As to restoration of voice, prognosis is very good on the whole; but in multiple papilloma, and in growths with broad attachments, the voice is not likely to regain its full range and purity. In the case of singers and other professional voice-users, the prognosis should be very guarded, if the growth springs from the cords, or if there is much catarrhal thickening.

As regards recurrence, it is only to be feared in papilloma. A fibroma, once thoroughly removed, does not recur, and the same is true of other simple growths. Papilloma, however, may even recur after the larynx has been free for years. The question of the malignant degeneration of benign growths has been finally settled by the collective investigation instituted by Semon. That such a transformation may occur is possible, but it is an event of the greatest rarity, and one which is in no way influenced by intralaryngeal treatment.

Treatment.—It may be regarded as certain that no internal treatment has any influence on the progress of laryngeal neoplasms, though arsenic has been said to have a specific influence on papillomata. Cases have been recorded of growths disappearing under the use of astringent or alkaline sprays. These were no doubt purely inflammatory products, and vocal rest may have had as much to do with the result as the local application. An exceptional case is that reported by Delavan, in which a large papilloma disappeared completely under the prolonged use of a spray of absolute alcohol.

Except in the case of "singers' nodule," which is often cured by prolonged rest of the voice, we may say that practically all laryngeal growths require operative treatment for their removal. In a small number of cases, however, the growth may be left alone, either because it produces no symptoms, or because the symptoms are so trifling that the patient is unwilling to submit to operation. Examples of this sort which have come under my notice have been epiglottic cysts, and small fibromata of the vocal cords.

The introduction of cocaine has done much to simplify all intralaryngeal operations, and to shorten, or do away with the need for, the preliminary training of the patient. It has, however, by no means removed all difficulties, or made it safe for any but those who have undergone prolonged training of eye and hand, to undertake these operations.

A great variety of instruments—forceps, knives, curettes, guillotines, and snares—have been employed in the removal of laryngeal

growths; and while the choice of instrument will partly depend on the situation, size, and nature of the growth to be removed, the individual preference of the operator is probably the most important factor. It is unnecessary to describe in detail all these instruments; but we may state that two, a cutting forceps and a snare, will be found sufficient for all purposes. In fact, we might almost say that the former alone is sufficient, as the chief merit of the snare is that with it one can scarcely do any harm. It is occasionally of use, however, in the difficult growths at the anterior commissure. I have never seen the advantage of a large and powerful forceps, such as that of Mackenzie, though it has always been the favourite instrument in this country; and the right-angled curve, which he adopted in order to avoid touching the epiglottis, is no longer required with cocaine anæsthesia.

The delicate, catheter-curved instruments, such as the forceps of Schroetter or Jurasz, or the double curette of Krause, permit of all manipulations being completely controlled by the eye, and can be used to raise the epiglottis so as to get at growths in the anterior commissure. Another advantage of Schroetter's forceps is that, being made of soft metal, the curve can be altered so as to make the instrument suitable for growths in any part of the larynx. Whatever forceps be used, the blades should be well sharpened, so that the growth is cut off and not torn away. The so-called "safety forceps" of Dundas Grant has been highly spoken of for growths on the edges of the cords, and I know of no objection to this instrument beyond its name. "Safety" must lie in the trained hand and eye of the operator, and not in the instrument he employs. Though caustics are no longer employed for the destruction of growths, the galvano-cautery has still its advocates. In the case of angiomata, or other highly vascular tumours where bleeding is feared, it may be a serviceable instrument, but there is always a risk of doing permanent injury to the voice when it is used on a vocal cord.

The multiple papillomata of young children present special difficulties in the way of treatment. That thyrotomy would prove the best method of dealing with many of them there is little doubt, if we knew how to prevent recurrence. Till we can do this I think that, on the whole, Semon's advice is the best: to perform a tracheotomy as soon as it becomes necessary, and then to wait till the child is old enough to permit of intralaryngeal treatment. In a very small proportion of cases the growths may disappear spontaneously after the larynx has been put at rest by the tracheotomy. This plan of waiting, however, is not always so successful as one could wish, and more than once I have advised thyrotomy, as the health of the child has suffered while wearing a cannula, or frequent attacks of bronchitis or broncho-pneumonia have become a source of danger. Two methods of operating which have been introduced in recent years promise to be of value in these difficult cases. One is the method of Scanes Spicer for operating under chloroform anæsthesia, combined with the local application of cocaine to arrest the secretions of the larynx and pharynx; the other is the direct method of Kirstein. Both have been successfully adopted in a few cases, and will no doubt at times enable us to avoid a tracheotomy.

Apart from cases of multiple papillomata in children, external operation will scarcely ever be necessary in the treatment of benign growths. The rule is that "an external operation in a case of a benign growth of the larynx is only indicated when an experienced laryngologist has failed to remove the neoplasm *per vias naturales*." The number of cases coming under this category will be exceedingly small, and is practically confined to sessile subglottic growths of great rarity.

Sub-hyoid laryngotomy has been employed in a few cases for the removal of large growths situated at the entrance to the larynx. In the case of simple growths this operation is never called for, as removal through the mouth is always possible, either with or without a preliminary tracheotomy.

To prevent the recurrence of papillomata many applications have been tried. Nitrate of silver, chromic acid, the electric and thermo-cautery, have all proved unavailing. Pure lactic acid, as recommended by Schmidt, has been of most value in my experience.

## Malignant Disease of Larynx


| | |
|---|---|
| Definition and Introductory Remarks | 351 |
| Etiology | 352 |
| Pathology | 353 |
| Symptoms | 353 |
| Diagnosis | 355 |
| *From Benign Tumours* | 355 |
| *Chronic Laryngitis* | 357 |
| *Syphilis* | 357 |
| *Tuberculosis* | 358 |
| *Lupus* | 358 |
| *Pachydermia Laryngis* | 359 |
| *Laryngeal Paralysis* | 359 |
| Prognosis | 359 |
| Treatment | 360 |


Definition and Introductory Remarks.—The term malignant disease of the larynx comprises the two affections known otherwise as carcinoma and sarcoma of the larynx. Both are rare, sarcoma even much more so than carcinoma. According to Gurlt's large statistics, laryngeal cancer amounts to 0·5 per cent of cancer in general, and the proportion of sarcoma to carcinoma is as 1 to 11-12. In spite of the

rarity of the disease, however, it is of the greatest importance that the general practitioner, to whom this class of patients almost always applies at first, should be well acquainted with the early symptoms. It will be shown that, if recognised in the initial stages, a much larger proportion of cases of malignant disease of the larynx can be radically and lastingly cured than is at present considered possible, whilst if its recognition should be much delayed, owing to the erroneous notions which, unfortunately, still very generally prevail with regard to the early symptoms of the disease, the patient's chances are becoming much worse, or are even entirely lost.

The description of both carcinoma and sarcoma of the larynx may well be combined, inasmuch as, with regard to the symptomatology, diagnosis, prognosis, and treatment, the two forms of malignant growths run so very similar a course that to describe them separately would entail useless repetitions.

From the practical point of view, however, it is desirable to adopt the late Professor Krishaber's terminology, and to distinguish between "intrinsic and extrinsic carcinoma."

The former variety comprises cancers arising from the interior proper of the larynx, *i.e.* from the vocal cords, ventricular bands, ventricles of Morgagni, and the subglottic cavity. Extrinsic carcinomas grow from the epiglottis, the aryteno-epiglottic folds, the inter-arytenoid fold, and the posterior surface of the cricoid cartilage. Sarcoma of the larynx much more frequently belongs to the intrinsic than to the extrinsic variety.

Etiology.—The origin of malignant disease of the larynx is as little known as that of malignant disease in general. It is still quite uncertain whether the affection be due to a microbic invasion or to a developmental error. A few facts, however, of great practical importance are known with regard to the natural history of the disease.

First of all, the affection is almost always primary, *i.e.* it either arises in the larynx itself or reaches that organ by direct contiguity; metatastic or secondary cancer and sarcoma of the larynx, if occurring at all, are extremely rare.

On the other hand, primary cancer of the larynx has little or no tendency to secondarily affect the internal organs or distant parts of the body. But there is that great and, from a practical point of view, most important difference between intrinsic and extrinsic cancer of the larynx that, in the former, the lymphatic glands of the neck are only affected very late in the progress of the disease, and sometimes not at all, whilst in the extrinsic form these glands become implicated to a large extent and, as a rule, at a very early period. Sarcoma of the larynx, as a rule, shows equally little tendency to affect the lymphatic glands of the neck and the internal organs of the body.

Secondly, the male sex is undoubtedly much more prone to cancer of the larynx than the female. In my own experience the proportion is about three to one, and this, I believe, agrees with the general experience. At the same time it is extremely curious that whilst among my male patients one-fourth only of the total number suffered from purely extrinsic malignant disease, considerably the greater half of my female patients were affected with this much more intractable form of the fell disease, the new growth usually starting from the posterior surface of the cricoid cartilage. The causes of these differences are quite obscure; the facts, however, can be vouched for.

Thirdly, the overwhelming proportion of all cases of malignant disease coming under observation is formed by the thirty years of life between 40 and 70; and of these thirty years, again, the decade between 50 and 60 takes up by far the largest individual proportion. Neither younger nor greater age, however, is spared by the scourge; I have myself seen and described three cases in which the age of the patient was 80 or more, and several in which the age was between 20 and 40. My youngest patient was 27 years old, and recently, by a curious coincidence, I have within three weeks seen three patients afflicted with laryngeal cancer, whose ages were between 30 and 35. Even younger patients suffering from laryngeal cancer have been seen by other observers, and, in a very few cases, even children have been described as suffering from this terrible disease.

Fourthly, occupation, heredity, and habits, according to my experience, have no influence whatever upon the production of the disease. It is met with in the upper classes just as frequently, if not more so, than in the lower; the strong are, if anything, more frequently attacked than the weak; smokers and people addicted to alcohol are not more liable to the affection than total abstainers; people leading an active life are just as prone as those whose occupations are sedentary; and professional voice-users run no greater danger than silent people.

Fifthly, the assertion that there was a special liability of benign laryngeal growths to undergo malignant degeneration, particularly after intralaryngeal operation, has been shown by me on the basis of collective investigation, made amongst the leading laryngologists of the world, to have been totally unfounded. In 8216 cases of intralaryngeal operation, five cases only were reported in which such a transformation could be admitted as certain, *i.e.* 1 in 1645. In seven further cases the transformation, though not certain, was probable, and in another ten doubtful; so that even if the probable and doubtful cases were admitted, in addition to the certain

ones, into the category of malignant degenerations of previously benign laryngeal growths, the proportion would be as 1 in 373, whilst if the "certain" and "probable" cases only were admitted, the proportion would be 1 in 685.

Under any circumstances, there is not the least corroboration by actual facts of the assertion that there existed a special liability of benign growths to undergo malignant degeneration after intralaryngeal operation, the less so as the collective investigation referred to has also shown that actually a larger number of spontaneous degenerations occurred in non-operated cases than post-operative degenerations, in cases which had been submitted to intralaryngeal operation.

PATHOLOGY.—By far the greatest number of cases of laryngeal carcinoma belong to the squamous-celled variety (epithelioma); scirrhus and medullary cancer are infinitely rarer. In one isolated case I have observed columnar-celled carcinoma, and in one other case villous cancer. Spheroidal-celled or glandular-celled carcinoma (adeno-carcinoma) I have never seen, but the latter variety has been described by several observers. The enormous preponderance of epithelioma observed in my own cases is fully in accordance with general experience.

Sarcoma occurs in both the round and spindle-celled varieties, and additionally in combination with other forms of growths, as fibro-sarcoma, myxo-sarcoma, and very rarely lympho-sarcoma. The histological characteristics of malignant growths in the larynx in no way differ from those of analogous tumours in other parts of the body.

SYMPTOMS. — The symptoms of malignant disease of the larynx, including both carcinoma and sarcoma, in their early stages vary very greatly according to the primary localisation of the growth. The still almost universal notion, viz., that constitutional and grave local symptoms necessarily accompany cancer or sarcoma of the larynx from their very onset, is absolutely wrong, so far as the more frequent variety, the intrinsic, is concerned, and the sooner this fact becomes generally admitted the better for the sufferers, and their chances of being saved.

In the intrinsic variety the initial symptoms are very trivial. If the growth starts from one of the vocal cords, or their anterior commissure, the first, and, *for a long time*, the *only* symptom is hoarseness. I have known a good many cases in which simple huskiness or hoarseness of the voice, unattended by pain or any other local or constitutional symptom, remained for a year, or even more, the *only* symptom of the grave affection. It should, therefore, be an invariable rule for every general practitioner to carefully examine, or have examined by an expert, the larynx of any middle-aged patient who, for any length of time, has been suffering from obstinate hoarseness, even if there be no other symptoms of any kind. The hoarseness in such cases develops gradually, in proportion to the increase of the growth, and finally ends in complete or almost complete aphonia. Should, meanwhile, the glottic space have been considerably encroached upon by the new growth, difficulty of breathing, usually steadily increasing, but, in a few rare cases, occasionally intensified by violent spasmodic attacks, makes its appearance, and if the disease be allowed to progress without hindrance, usually becomes so severe as to necessitate the performance of tracheotomy.

Sometimes, however, even when the stage of complete aphonia and considerable dyspnœa has been reached, a temporary fallacious improvement takes place, owing to peripheral breakdown of the neoplasm. In such cases temporarily free respiration and almost normal voice may be for a short time restored, and the patient and his friends may hope that an erroneous diagnosis had been arrived at. Several such cases are within my own cognisance. Soon, however, the growth manifests fresh activity, and the old symptoms return with increased severity.

When once the stage of ulceration has been reached, and not rarely even long before that time, there is much increased production of phlegm, usually frothy in character. Later on the expectoration is sometimes blood-stained, and occasionally little hæmorrhages occur. At this period the breath also often becomes fœtid, but even at that time no deterioration of the general health need have occurred, and there may be no pain, no difficulty in swallowing, and no enlargement of the cervical glands. It is perhaps here the place for the observation that the significance of pain in malignant disease has, according to my own experience—which in this class of cases is exceptionally large—been greatly overstated. In a number of close upon two hundred cases of this kind which I have seen, I hardly remember a single one in which pain played the predominant rôle; often enough it was either quite insignificant, or even completely absent till the patient's death, although it must not be denied that in a few cases it was described as an early symptom.

Should the new growth be allowed to extend and to transgress the confines of the larynx proper, the cervical lymphatic glands, as a rule, become enlarged and form smaller or larger clusters of hard masses, which vary in their mobility, and not rarely ultimately become fixed to the neighbouring parts. Should the œsophagus be affected, dysphagia becomes a prominent feature. Should the disease extend to the deeper structures and involve the cartilaginous framework, perceptible broadening of the larynx will be perceived, and later on symptoms of perichondritis may occur, which in some cases so entirely overshadow the original disease that the latter can only be

diagnosed with the greatest difficulty or even not at all.

Finally, when the new growth has found its way, either through the thyro-hyoid membrane or through destruction of the laryngeal cartilages themselves, into the soft parts surrounding the larynx, smart hæmorrhages may be caused by invasion of the blood-vessels; violent neuralgia or motor paralysis may be due to invasion of the nerves of the neck; and finally, the external integument may be broken through, and the new growth appear externally as a fungating irregular tumour, which alternates between breaking down and luxuriantly sprouting, and is often covered with ichorous pus. In more than one case in which tracheotomy had been performed, I have seen that the tracheal wound, having been invaded by the new growth, was gradually enlarged by ever-recurring breakdown of cancerous vegetations, occupying its borders, until finally the tracheal cannula was lying in a huge cavity formed by the remnants of the larynx and the upper part of the trachea, which had been almost entirely destroyed by the progress of the disease. In such cases, occasionally, almost the whole or, at any rate, the greater part of the cartilaginous framework is expectorated during life in smaller or larger necrosed fragments.

The final stages both of the intrinsic and the extrinsic variety, if the disease has been allowed to take its natural course, are usually those of general cachexia. In some cases the hæmorrhages towards the end get more frequent and abundant, and the patient sinks from sheer exhaustion; in other cases in which the œsophagus has become involved, increasing dysphagia hastens the fatal end. Not rarely fistulous communications are being established between the air- and food-passages, and the termination is often brought about by septic pneumonia, which is set up by the entry of particles of food into the air-passages, or by the secretion from the ulcerated surfaces.

The duration of the disease enormously varies in different cases. The longest case I have seen extended, between the appearance of a small nodule on the anterior part of the right vocal cord and the end of life, to just four and a half years. It is, however, very rare that patients survive more than three years after the commencement of the disease, and often the total duration, if the disease be allowed to proceed without let or hindrance, is no more than from one to two years.

The initial stages of intrinsic malignant disease of the larynx are, of course, somewhat different, if not the vocal cords themselves, but some other part of the interior of the larynx, such as the ventricular bands or the subglottic cavity, should be the original seat of the mischief. In such cases for some considerable time no subjective symptoms may be produced at all; the onset of such would arise when either the space for respiration is encroached upon or the action of the vocal cords interfered with. In such cases the growth may have attained considerable dimensions previous to causing any symptoms. The further development of the subjective symptoms will in such cases, of course, be similar to the events sketched as characterising the later stages of malignant disease of the vocal cords.

The extrinsic variety, as a rule, draws the attention of the patient and of his medical adviser at a much earlier time to the existence of grave mischief than the intrinsic. When the new growth is situated on the posterior surface of the cricoid plate, difficulty, and sometimes pain in swallowing, together with secretion of at first purely frothy, later on sometimes slightly blood-stained phlegm, are the first signs of the disease. Soon in most cases enlargement of the cervical lymphatic glands, at first under the angle of the jaw, later on along the whole root of the neck, becomes noticeable. This enlargement, according to the situation of the new growth, is developed sometimes on one, sometimes on both sides. Occasionally it attains such considerable dimensions already in early stages, when the internal disease does not yet cause any troublesome symptoms, that the original focus may be quite overlooked and the glandular disease supposed to be primary. Several such instances have come under my own notice. As the growth in the variety now under consideration increases in size and covers almost the whole plate of the cricoid cartilage, not only does dysphagia increase, but also, owing to the mechanical destruction of the muscular substance of the abductors of the vocal cords (the posterior crico-arytenoid muscles), myopathic paralysis of these muscles and gradually increasing narrowing of the glottis supervene, which often enough require early tracheotomy. This class of cases is, owing to these circumstances, perhaps the most cruel variety of malignant disease of the larynx, the poor patient hovering between starvation and suffocation. In still later stages symptoms of perichondritis develop, and the final course is similar to that of the intrinsic variety.

In cases in which the epiglottis is the primary seat of the mischief, at first often simply some difficulty and pain in swallowing and change in the timbre of the voice are noticed, the latter assuming a curiously "throaty" thick character as the epiglottis is changed into a large tumour. The further progress depends upon the direction in which the new growth progresses. Usually it affects the root of the tongue and the lateral walls of the pharynx and the œsophagus, when dysphagia will, of course, materially increase. Sometimes it descends into the larynx and causes respiratory difficulties in addition to hoarseness and loss of voice. In a third variety

it extends in both directions, when, of course, all the symptoms named will make their appearance in combination. In this variety, too, the implication of the cervical lymphatic glands may occur at so early a period that no suspicion is entertained of the existence of the internal disease, and the glandular tumour is considered to be primary.

In very rare cases a malignant tumour, particularly when starting from the arytenoepiglottidean fold, may be at first pedunculated, when the symptoms, of course, would vary according to whether it falls into the larynx or rests in the pyriform sinus, causing vocal disturbances and dyspnœa at one, and inconvenience in deglutition at another time.

Finally, that extremely rare variety must be mentioned in which, from the very first, the symptoms are so much those of perichondritis (pain, difficulty in swallowing, vocal changes, febrility, etc.) that the existence of malignant disease is hardly taken into consideration at all. I have quite recently seen a case of that kind, in which the diagnosis of tuberculous perichondritis had been made, and in which my diagnosis of malignant disease was received rather incredulously. The further progress of the case, however, fully established its correctness.

Diagnosis. — The diagnosis of malignant disease of the larynx in its earliest stages often is very difficult, inasmuch as its appearances at its very onset are so protean that it may easily be mistaken for various other affections, the differential diagnosis from which will be treated later on.

The very earliest sign, as a rule, is simple congestion of the parts, which afterwards become tumefied. This, of course, will be most manifest if the disease starts from one of the vocal cords, and the unilateral character of the congestion will at once draw the attention of the experienced observer to the probability of impending graver mischief.

In other cases the disease, from the first, begins in the form of a diffuse tumefaction. This tumefaction may occupy any part of the larynx, but its seats of predilection are distinctly the vocal cords, and after them the ventricular bands. In its further progress it may take either the form of a general infiltration of the affected parts, in which all the preformed parts attacked completely perish, or it may form a more definite tumour, appearing as a rule as a somewhat globular, irregular, nodulated, sessile mass, the colour of which may be either that of the surrounding mucous membrane, or somewhat more pale or more dusky-looking than the latter. Neither of the two categories just described, as a rule, offers much difficulty in diagnosis when the new growth has attained a certain size. Occasionally, however, particularly in cases of general tumefaction, matters are not so easy, and mistakes may be committed even by the most experienced observer. (See further on the differential diagnosis between malignant disease, syphilis, and tuberculosis.)

The really difficult cases, however, are those in which cancer or sarcoma of the larynx make their appearance in the form of an apparently innocent new growth. Malignant disease often enough shows itself first either in the form of a somewhat nondescript tumour, or even completely simulates the appearance of a papilloma or a fibroma. The similarity becomes even more striking if, as in rare instances, the malignant new growth is pedunculated. Should, as in a unique case observed by me, the papillomatous appearance of a small epithelioma be additionally concealed by a large blood-clot, which had formed round the papillomatous excrescences, the new growth may be taken—as, indeed, it was in this case by several competent observers —for an angioma.

Whilst the difficulties in this class of cases are sometimes undoubtedly very great, yet there are certain points which will help us in establishing the differential diagnosis between benign and malignant growths of the larynx.

First of all the *age* of the patient comes into question. Although benign growths of the larynx may arise at any time of life—in fact, from intra-uterine existence up to the age of eighty or more—yet the earlier half of life up to the age of forty certainly is much more prone to such growths than the later. On the other hand, as already stated, malignant growths, though in rare cases they may arise at an early period of life, are infinitely more frequent from the age of forty upwards. Thus a growth, otherwise innocent-looking enough, the history of which shows that it had arisen after the fortieth year, is *a priori* suspicious.

Secondly, a malignant new growth, even in its early stages, is often, though not always, surrounded by a zone of circumscribed dusky hyperæmia, which, particularly when the growth, occupies the middle part of the vocal cord, is in striking contrast to the brilliant white colour of the anterior and posterior ends of the cord. It must, however, be emphasised that this hyperæmia is not always present, and that its absence does not exclude malignancy.

Thirdly, with regard to the differential diagnosis of malignant neoplasms from individual forms of new growths, this is to be said: a laryngeal cancer may at first look entirely like a papilloma, but a benign papilloma shows a decided tendency to localise itself on the *anterior* parts of the vocal cords, and it is therefore *a priori* suspicious if a papillomatous growth, particularly in a person advanced in years, should be met with on the *posterior* parts of the vocal cords, or, worse still, in the interarytenoid fold, where benign growths are hardly ever found.

The same applies to apparently innocent papillomata situated on the epiglottis, or on the aryteno-epiglottidean folds. Again, the experienced observer will at once think of the possibility of malignancy if he finds that the individual projections of an otherwise apparently simple papilloma are *pointed* instead of *rounded*, as those of an ordinary papilloma are; and this suspicion will be increased if the colour of the new growth is *snowy white* instead of pinkish, as usual with laryngeal papillomata. In some such cases the growth looks like a miniature snow-covered meadow. Needless to say, the suspicion will be increased if several of the suspect features so far mentioned are met with simultaneously, *i.e.* if a snowy meadow such as just described is seen to occupy a position unusual for ordinary papillomata—such as, for instance, the aryteno-epiglottidean fold—in the larynx of an elderly person. Further, one's attention ought to be roused if one finds a sort of papillomatous fringe occupying almost the entire length of one vocal cord, whilst the other one is perfectly free.

A sign of grave diagnostic importance is impairment of the mobility of the vocal cord from which the new growth springs. The value of this sign, to which I was the first to draw attention, has been repeatedly decried, and my utterances on the subject have been curiously misunderstood by some Continental authors. I wish, therefore, to declare as plainly as possible that I neither believe such impairment of mobility to be present in *every* case of early malignant disease of the vocal cords, nor that its *absence* in any way militates *against* the disease being malignant. All I contend is, that if in the case of a doubtful growth springing from a vocal cord—and not only when the growth is situated near the crico-arytenoid articulation, but even in the anterior part of the vocal cord—an impairment of mobility, *i.e.* some sluggishness of the movements of the affected cord, is observed, this is a most valuable sign, pointing to the malignant nature of the affection. This impairment of mobility is, of course, due to the *infiltrating* character of the new growth, as against the mere surface-excrescence formed by a benign neoplasm. It need, of course, not be present if the malignant new growth should itself be more a superficial one—as, for instance, seen in rodent ulcer—or if it should not yet have deeply penetrated into the tissues; but *if* it be present, I have so often found it a sign of great value for the early diagnosis of laryngeal malignant disease that no amount of contradiction will shake my conviction.

It goes, however, without saying that this applies only to cases in which malignant disease appears at first in the form of a distinct *tumour*; should it take from its onset the form of a *general infiltration* and tumefaction, the question of mobility of the vocal cord cannot be utilised for a differential diagnosis, inasmuch as a syphilitic or tuberculous infiltration or a perichondritic process or similar causes may also lead to impairment of the mobility of the vocal cords, indistinguishable from that produced by malignant infiltrating disease.

Only in very rare instances, and only in the early stages, will there be any danger of mistaking a malignant new growth for a fibroma. Some doubt may occur when a red semiglobular sessile tumour makes its appearance on the vocal cord of a middle-aged person. But whilst in fibroma the semiglobular form is throughout maintained, and no impairment of mobility of the cord nor any ulceration of the tumour itself occurs, even when the growth has attained a very considerable size, in the further progress of malignant disease the tumour becomes mammillated, loses its semiglobular form, becomes ulcerated, and interferes with the free mobility of the cord itself. In one of my cases, even after microscopic examination of the removed tumour the diagnosis remained doubtful between fibro-sarcoma and soft continuous fibroma.

I am not aware that there is much danger of mistaking malignant disease of the larynx for any other form of benign laryngeal neoplasms, my own case, in which a suspected angioma turned out to be an epithelioma, having so far remained unique.

It need hardly be said that in all cases in which the clinical examination alone does not suffice to establish the diagnosis, the aid of the microscope, if possible, should be invoked. That is to say, a fragment of the growth should be intralaryngeally removed, and subjected to *searching* microscopical examination. No conclusion ought to be drawn from a single slide, unless the appearances are absolutely characteristic of squamous-celled carcinoma. The fragment removed should be examined throughout, and some of the cuts should, if possible, be carried rectangularly to one another, so as to diminish the possibility of mistakes. With all that, it ought to be remembered that growths are not necessarily homogeneous in their structure, that the peripheral parts may contain no characteristic elements, and that the more or less fortuitous character of intralaryngeal removal gives no guarantee that really characteristic portions have been reached by the laryngeal forceps with which the removal had been carried out.

Matters therefore stand thus: should the microscopic examination definitely establish the histological characteristics of a malignant new growth, well and good; the diagnosis is settled. Should, however, the microscopical evidence be simply negative, the inverse conclusion, viz., that the growth was not malignant, is absolutely unpermissible, and the clinical observer must continue to watch the progress

of the suspected growth as anxiously as he had done before the microscopical examination was made. He has no right to throw the responsibility for an erroneous diagnosis upon the microscopist, and must, if needs be, have the courage of his own opinions, and proceed to radical operative interference even in the face of negative microscopic evidence. Needless to say, the aid of the microscope is only available if there be a projection sufficiently large to be intralaryngeally removed. Often enough, in cases of general infiltration, this is not possible.

Having attained a certain size, which sometimes may be so considerable that they practically fill the entire larynx, malignant new growths, whether originally appearing in the form of a definite neoplasm or of a more general infiltration, begin to break down in parts whilst they extend in the periphery. The time within which this breakdown begins to occur immensely varies in individual cases. In most it will become apparent within a few months from the onset of the disease; in others, however, and I have seen several such cases, the growth, having attained a certain size, remained apparently stationary for a much longer time, the maximum I remember being a year and a half, before ulceration occurred. From that period onwards the laryngoscopic aspect usually is that of an irregular ulcerating tumefaction, covered in part with greyish mucopus, whilst in other parts reddish fungating granulations may be seen to be springing up one day, and to have practically disappeared the next. In still more advanced stages, and particularly when the perichondrium has become involved, there is often a very considerable amount of acute œdema to be seen round the new growth, and this œdema may not very rarely completely obscure the original disease. Often enough, between all these changes, it is extremely difficult, if not impossible, to recognise the preformed parts of the larynx.

It will have been seen from the foregoing description that no uniform picture must be expected in these cases; practically every case shows some individual differences.

When once the disease has advanced to that stage in which practically the whole larynx has become involved, and changed into a partly ulcerating, partly luxuriantly proliferating tumefaction, whilst at the same time the glands underneath one or both jaws form very large, very hard, or even externally ulcerating masses, there can be, as a rule, not much difficulty about the differential diagnosis of malignant disease from other laryngeal affections.

In the earlier stages, however, these difficulties sometimes are very considerable, and indeed so great that even the most experienced observers are not exempted from occasional diagnostic errors. Apart from benign neoplasms, the differential diagnosis from which has already been dealt with, the affections with which malignant disease of the larynx is most likely to be confounded are chronic laryngitis, syphilis, tuberculosis, lupus, perichondritis, pachydermia, laryngis, and paralysis.

The differential diagnosis from chronic laryngitis comes, of course, into question only in the early stages of malignant disease, and is as a rule facilitated (as already mentioned) by the fact that the congestion preceding actual tumefaction in malignant disease is *uni*lateral. Thus, if a vocal cord should be the primary seat of the disease, the much congested colour of the affected cord strikingly contrasts with the normally white one of the other. At least one case, however, has come under my notice in which, after a preliminary stage of apparently simple *bi*lateral congestion of the vocal cords, malignant disease of the larynx developed. In that case unusual complications occurred, inasmuch as, trusting to the non-dangerous character of what appeared to be simple chronic catarrh of the larynx, no objection had been medically raised to the patient's insuring his life for a large sum shortly before the real character of the disease declared itself. The possibility, remote though it be, of malignancy ought therefore to be kept in view, if an apparently simple chronic laryngitis, even though *bi*lateral, does not yield to the usual remedies.

With regard to syphilis the differential diagnosis often offers very considerable difficulties. Of course, the previous history of the patient, the coexistence of other syphilitic lesions in other parts of the body, or the traces of old syphilitic disease in the forms of scars, etc., will help in the decision; but none of these are absolutely to be relied upon, as malignant disease not rarely affects persons who have suffered from syphilis. Tertiary syphilis of the larynx manifests itself either by a distinct gummatous tumour, or by a more general gummatous infiltration. The former, which, as a rule, is red or yellow, usually solitary, occasionally multiple, and surrounded by a zone of inflammation, as a rule breaks down very rapidly, often within a few days, whilst a malignant ulcer requires, as a rule, weeks for its development. When a gumma has broken down, a large, deep crateriform ulcer results; whilst in a malignant ulcer tumefaction often remains round the ulcer, and fresh thickening appears in the periphery. The size of the carcinomatous ulcer usually is larger than that of a syphilitic, and when once the boundaries of the larynx have been transgressed by malignant disease, the infiltration of the cervical lymphatic glands as a rule is much more considerable than that observed in syphilis. All these signs, however, do not positively protect against occasional mistakes, and in a good many

cases the use of iodide of potassium will have to solve the doubt. It will, indeed, be found a good rule to begin in *every* case of malignant disease, whether there be any doubt as to the correctness of the diagnosis or not, with the administration of iodide of potassium in large doses for a week or a fortnight's time. The initial dose ought to be 10 grains three times a day, and this may be pushed to 30 grains or even more three times daily. No conclusions as to the efficacy of that drug, however, must be drawn from a mere *subjective* improvement. Often enough, even in cases of cancer, iodide of potassium has a transitory beneficial influence by resorbing the œdema surrounding the actual growth, and the patient in such cases feels and swallows better, without the disease being actually arrested. It is only from the occurrence of *actual* changes for the better in the patient's larynx that a conclusion can be drawn as to the syphilitic, as against the malignant, nature of the ulcer.

In the great majority of cases the differential diagnosis of malignant disease of the larynx from tuberculosis is much easier than that from syphilis. The general constitutional symptoms, the almost always concomitant pulmonary affection, the patient's age, the bacteriological examination of the sputum, the characteristic pallor of the larynx, the pseudo-œdematous infiltration of the mucous membrane over the epiglottis and arytenoid cartilages; the slow development of the ulcers—their large number and generally small size; the absence of considerable infiltration of the glands in the neck;—all these signs will, in the great majority of cases, easily enough show the tuberculous nature of a laryngeal ulceration.

Still there are some cases in which the differential diagnosis is enormously difficult. Thus a case of my own, observed in a gentleman, aged over fifty, in which an ulcer surrounded by œdematous tissue occupied the posterior end of a vocal cord, whilst the rest of the larynx was perfectly normal, and the lungs quite free, offered such diagnostic difficulties that an exploratory thyrotomy had to be performed. Even after the larynx had been opened, the nature of the disease remained doubtful, and only the histological examination made by Mr. Shattock whilst the operation was proceeding revealed the tuberculous nature of the mischief.

In another case just now under observation, more than one-half of the epiglottis of a gentleman, aged sixty-three, had been destroyed by ulceration, whilst the remaining portion was changed into a shapeless red tumefaction, covered with greyish muco-pus. From the sides of this, more ulceration extended towards both the arytenoid and epiglottic folds. There were no signs whatever, locally or constitutionally, of tuberculosis in the case, and everything, except that there was no infiltration of the cervical lymphatic glands, seemed to be in favour of malignant disease. The probatory removal, however, of a piece of the stump of the epiglottis definitely established, by means of the microscope, the presence of giant cells, and of very characteristic tubercular tissue. The ulcerated portion was removed by sub-hyoid pharyngotomy by Mr. Victor Horsley, and the patient is now making an excellent recovery. Several cases are known in which the whole larynx was removed, and this by good and competent observers, under the mistaken impression that the disease was carcinomatous, whilst in reality the disease was tuberculous. Still, such cases are very rare, and in the majority of cases the differential diagnosis between malignant disease and tuberculosis seldom offers any serious difficulties. In connection with this subject it ought, however, to be borne in mind that undoubtedly occasionally laryngeal carcinoma coexists with pulmonary tuberculosis, so that even the discovery of bacilli in the sputum cannot be looked upon as an infallible test.

Exceedingly rarely will there be any danger of mistaking cancer or sarcoma of the larynx for lupus, or *vice versâ*. To begin with, primary lupus of the larynx is very rare, and almost always there are concomitant signs in the nose, pharynx, and on the external integument. Secondly, the particularly worm-eaten appearance of the lupoid ulcers is very different from the deep and destructive ulcer, combined with a proliferating tumefaction, which characterises the later stages of malignant disease of the larynx. Extirpation of a fragment and microscopical examination will, in doubtful cases, help to establish the diagnosis.

As repeatedly stated in previous paragraphs, the differential diagnosis of malignant disease of the larynx, when appearing in the form of a general infiltration, from a perichondritis due to various causes, sometimes is very difficult; and additionally, it must not be forgotten that in the later stages of malignant disease of the larynx perichondritis forms one of the most regular symptoms.

It has also been already stated that in some cases the symptoms of perichondritis so entirely mask the original disease, that the differential diagnosis as to the causes of the perichondritis may become a matter of the greatest difficulty, and sometimes altogether impossible. In some of these cases, nothing but prolonged observation after the failure of a course of iodide of potassium will settle the diagnosis; a few cases have been described in which, not only during life, but even at the post-mortem examination, it was impossible to decide the actual nature of the case, and in which only the microscopical examination of the diseased organ finally settled the doubt. Under any circumstances the observer will do well, if he sees a case of otherwise inexplicable perichondritis of the larynx in a

grown-up person, with enormous tumefaction of the part, and œdematous swelling of the mucous membrane, to remember, amongst other possibilities, that he may have to do with cancer or sarcoma of the larynx. An exploratory thyrotomy may help to settle the doubt; but this means is, of course, not one to be indiscriminately recommended.

Very considerable difficulties are sometimes experienced in making a differential diagnosis between Virchow's pachydermia laryngis and malignant disease of the larynx, if the latter should start from the neighbourhood of the vocal process of the arytenoid cartilage. The laryngoscopic appearances of both diseases are sometimes extraordinarily similar to one another, and even the most experienced observer may, in cases in which the affection is unilateral, and the tumefaction at the posterior end of the vocal cord much developed, be very doubtful for a while as to what he has to deal with. Under these circumstances I have always found the question of the mobility of the affected cord a most valuable aid in the diagnosis.

Pachydermia, in my experience, never causes, however much the tumefaction may be developed, impairment of the mobility, whilst malignant disease, when originating in that situation, usually leads from its neighbourhood to the crico-arytenoid articulation, to a distinct sluggishness of the affected vocal cord, even if the tumour be still small. I am well aware that cases of pachydermia have been reported in which an impairment of the mobility of the affected vocal cord was stated to have been present; but I have, in a rather large experience of that disease, never seen such a case, and can only recommend to look upon the question of the mobility of the affected vocal cord as a very valuable differential diagnostic sign. In later stages, *i.e.* when a second pachydermia has developed on the corresponding part of the *opposite* vocal process, and when its most prominent part fits into the cup gradually arising in the middle of the original pachydermia, the difficulty of a differential diagnosis is but small; but it ought not to be left altogether out of consideration that in very rare cases, such as I have once described together with Mr. Shattock, a secondary carcinoma *by contact* may develop on the opposite vocal cord.[1]

The differential diagnosis between malignant disease of the larynx and various forms of paralysis will in very rare cases only come into question. If so, the diagnosis usually, for a time at any rate, is extremely difficult. Thus I have seen two cases in which the appearances were completely those of bilateral paralysis of the glottis-openers, the vocal cords lying close to one another in the middle line of the larynx. The subsequent course, however, proved that this appearance was due to subglottic malignant growth—in one case to an epithelioma, in another to sarcoma.

Finally, I may mention that, from my own personal experience, I do not think it possible to make a clinical and differential diagnosis between sarcoma and carcinoma of the larynx. I know perfectly well that directions describing different appearances of these two forms of growth may be found in almost all handbooks of laryngology; but I confess that I personally never have been able, from mere laryngoscopic examination, to distinguish between them clinically, either in their early or more advanced stages, and that in all my own cases the differential diagnosis has been arrived at by means of microscopic examination, either of fragments intralaryngeally removed before radical operation, or of the entire growth after this had been performed.

Prognosis.—Whilst there is, unfortunately, even now a but too universal belief that malignant disease of the larynx is necessarily a fatal disease, the progress made in both the diagnosis and the operative treatment of carcinoma and sarcoma of the larynx in the course of the last fifteen years is such that, in reality, matters are very different. As a matter of fact, the prognosis of malignant disease of the larynx varies enormously in individual cases according to (1) the original starting-point of the growth; (2) the period at which the patient comes under observation; (3) his general health. From personal experience, I have no hesitation in stating that if an intrinsic laryngeal cancer in a middle-aged or, at any rate, not too old and otherwise healthy person, comes under observation at an early stage, and if the patient agrees to radical operation without delay, the prognosis is equally good, if not better, than in any other form of malignant disease in any other part of the body. I make this statement on the strength of the fact that my own percentage, not merely of successful operations, but of lasting cures in this class of cases, at present amounts to 83·3 per cent. The prognosis, therefore, in this variety can unhesitatingly be pronounced to be very favourable.

On the other hand, if the patient, even though the affection originally belonged to the intrinsic variety, comes under observation at a time when the disease has become very extensive, when the cervical lymphatics have become involved, when his general health has already begun to suffer, the prognosis, needless to say, is, even now, a very grave one. The same applies, to an even higher degree, when the growth is primarily extrinsic, and particularly when it starts from the posterior surface of the cricoid plate. It is true that the progress of surgery has enabled us to save a good many even of such cases by more perfect methods of

[1] "Three Cases of Malignant Disease of the Air-Passages," *Transactions of the Pathological Society of London*, 1888. Case 2.

operation; still this can only be done by means of very serious and mutilating operative interference, and the risk of recurrence in this class of cases is extremely great. It need hardly be said that the prognosis will greatly depend also upon the age and general health of the patient, very old persons, and such afflicted with albuminuria and chronic affections of the respiratory passages, being *a priori* not nearly such suitable subjects for the operation as younger and generally healthy individuals. The general outcome of the foregoing observations is this, that in every case of malignant disease of the larynx one will have to strictly individualise with regard to the prognosis of life and the chance of operation.

TREATMENT. — The treatment of malignant disease of the larynx at the present moment can be only of a surgical character, and it has already repeatedly been stated that its prospects nowadays are much better than they used to be only a few years ago. There are, however, two dangers with regard to the selection of the method of surgical interference, which have become developed during the last few years, and against which a note of serious warning ought to be sounded. The aim of the practitioner in malignant disease of the larynx ought to be *to recognise the malady whilst it is still a purely local affection, and to remove it in that stage so thoroughly as to preclude, if possible, the danger of recurrence.* Two extremes ought to be equally avoided, viz., doing *too little*, and doing *too much*. The first of these two extremes is, in my opinion, represented by the intralaryngeal method, which has of late years been warmly and repeatedly recommended by German authorities whose names justly command respect. But the selection of this method in cases of malignant disease appears to my mind to militate against the very nature of cancer and sarcoma. It is their characteristic that they do not merely grow from the surface, but that they infiltrate the mother-soil from which they spring. Quite in accordance with this is the fact, which I have stated years ago, and which since then I have over and over again had the opportunity of corroborating, viz., that when the larynx is opened in a case of malignant disease, it is almost always found that the infiltration is much more extensive than one would have thought from laryngoscopic examination. Now, whilst the intralaryngeal method is excellently suited for the removal of excrescences from the surface, it does not give the least guarantee for a really radical and complete removal of infiltrated deeper tissues; and there is not the least certainty than even if all that appears suspect has been removed, the disease should not, all the while, progress without let or hindrance in these deeper structures which have not been reached by the intralaryngeal operation, without, for a considerable time, manifesting its presence in these tissues. The patient must, therefore, keep himself under constant observation for a very long time; and even such observation, if the disease be extending into the subglottic cavity, does not offer any guarantee against dangerous progress of the affection in that laryngoscopically only in part visible region. Thus, the proper moment for more radical operation may ultimately become irretrievably lost. Additionally, when the intralaryngeal method has been employed, the risk of constantly irritating by incomplete operation the affected part, and thereby producing a quicker rate of progress of the disease, is certainly more than theoretical, and ought to be taken into serious consideration. I am fully aware that a number of cases have been cured by intralaryngeal operation, and do not in the least doubt their actuality; but I consider the selection of the intralaryngeal method for that class of cases none the less as dangerous and altogether undesirable.

Equally little in the interest of the patient appears to me the other extreme, which has met with some acceptance in Germany, viz., excision of the whole larynx as soon as the diagnosis of malignant disease of that part has been made. If the disease could not otherwise be eradicated than by such heroic measures, matters would be different; but when, in a large number of early cases, a much less mutilating interference has been positively demonstrated to suffice for effecting a lasting cure, it appears to me hardly defensible to deprive the patient of an important organ, and of the use of his voice, not to speak of his being made a subject, half of pity, half of repulsion for the rest of his life, on account of the theoretical argument that total extirpation gave better chances against recurrence than partial extirpation or simple thyrotomy with removal of the soft parts. So long as the last-named operation was still on its trial in cases of this sort, such an argumentation in favour of total extirpation had some show of reason; but now that the experience of the last ten years has demonstrated by actual facts that all that is necessary can be obtained by means of so infinitely simpler, less mutilating, and less dangerous an operation, as thyrotomy is in comparison to total extirpation, it seems time that the latter method should be reserved for such cases only in which it is indispensable, and that in initial cases of intrinsic cancer or sarcoma thyrotomy should be generally awarded that position which, in the experience of those who have methodically practised it during the last ten years, it fully deserves.

As to the operative methods which ought to be selected in any individual case, they must depend entirely upon the primary situation of the new growth and on the stage in which the case comes under observation. In cases of intrinsic malignant disease, particularly when

it is limited to the vocal cords, or to their neighbourhood, there can nowadays be no doubt that thyrotomy is the proper procedure. Personal experience, extending over twelve years, has convinced me that if, after the performance of tracheotomy, and the protection of the trachea by means of Hahn's sponge-cannula, and of additional sponges, if necessary, against the entry of blood into the lower air-passages during the operation, the larynx be opened, the two wings of the thyroid held asunder, and the new growth with an area of healthy tissue around every part of its circumference be (after previous cocainisation of the part) thoroughly excised, and the basis of the removed part thoroughly scraped with Volkmann's sharp spoon, not only is the operation reduced to a minimum of risk, but also the chances of absence of recurrence, if the disease should have been still limited, are excellent. (See under Prognosis.)

The prospects of the voice after the performance of thyrotomy, if one vocal cord only should have been removed, are also surprisingly good. In the great majority of cases a cicatricial ridge forms in the situation just corresponding to the former place of the removed vocal cord, and on the healthy cord joining this ridge in phonation, a loud and serviceable voice is produced, which sometimes has a hoarse timbre, but in not a few cases is almost normal. Thus one of my patients, a clergyman, from whom eight years ago the whole of the left vocal cord and the front part of the left arytenoid cartilage were removed on account of a fibro-sarcoma, now regularly preaches in a church holding four hundred people. To obtain good results, however, it cannot be too strongly insisted upon that no undue sentimentality with regard to the subsequent preservation of the voice should be allowed to prevail over considerations of safety with regard to recurrence; and it is absolutely necessary to perform the operation *everywhere* in the healthy tissue surrounding the growth, and *not too near* the latter. For further particulars with regard to the technique of the operation I would refer to my various contributions on the subject.[1]

A further point in connection with the question of the selection of thyrotomy in this class of cases is this, that under no circumstances should the operator approach the operation with the fixed intention of performing thyrotomy and nothing else. But too frequently one finds after opening the larynx that the disease is more advanced than one had thought after laryngoscopic examination, and that mere removal of the soft parts under such circumstances was not likely to give a sufficient guarantee against recurrence. In such circumstances resection of parts of cartilages, or even partial extirpation of the larynx, ought to be proceeded with. If the disease should only come under observation at a more advanced stage, if there be already signs of perichondritis, or if there be any doubt as to whether the cartilage itself had become affected, partial extirpation will of course take the place of mere thyrotomy. In still more advanced stages in which both sides of the larynx are affected, or in which the disease, unfortunately, be situated on the posterior wall of the larynx, nothing short of total extirpation of the organ, combined, if necessary, with removal of the already affected cervical lymphatic glands, may become imperative. In these cases the principle of commencing the operation by cutting the trachea horizontally and sewing the lower end into the edges of the skin wound, has recently led to a very considerable diminution of the danger of sepsis after the operation, and to a much greater saving of life. But it need not be said that total extirpation means a grave mutilation, that an artificial larynx is but a sorry substitute, which, additionally, can apparently be worn at length by a few patients only, and that in those cases in which the lower end of the trachea has been sewn into the external wound a weak, toneless, whispering sound, produced in the pharynx, is the best that can be expected unless an artificial larynx, such as Professor Gluck's new contrivance, be always used. Still, life is valued so highly by many patients, and the surgical progress in treating even much advanced cases of laryngeal cancer, complicated by infiltration of the cervical lymphatic glands, has been of late years so great, that one ought not to dissuade patients from undergoing the operation, but leave the decision to them.

In cases, finally, in which the disease starts from the epiglottis or an aryteno-epiglottidean fold, supra-hyoid pharyngotomy would seem to be the least serious operation, and at the same time to completely suffice to remove the growth *in toto*. This operation has not hitherto been extensively practised, probably because the cases suitable for it are, on the whole, rare, and in those cases in which it has been performed a curious fatality has followed; but it may fairly be hoped that by perseverance in it better results will be obtained in the near future.

Finally, in such cases in which the patient either refuses to undergo a radical operation or in which he comes under observation too late for such to be recommended, or in cases in which the disease is situated on the œsophageal aspect of the cricoid cartilage,

[1] "On the Results of Radical Operation for Malignant Disease of the Larynx," *Lancet*, December 15, 22, 29, 1894; "Zur Frage der Radikaloperation bei bosartigen Kehlkopfneubildungen mit besonderer Berucksichtigung der Thyreotomie," *Archiv für Laryngologie*, Bd. vi. Heft 3; "Die Thyreotomie bei bösartigen Kehlkopfneubildungen," *Therapie der Gegenwart*, April 1899; "Einige Bemerkungen zu der neuen Sendziak'schen Statistik uber die operative Behandlung des Larynxkrebses," *Monatschrift für Ohrenheilkunde*, No. II., 1899.

extending from there downwards, so that not only total extirpation of the larynx, but also resection of a large part of the œsophagus, would be required—a class of cases, moreover, almost always complicated by early and very considerable implication of the cervical lymphatic glands—palliative measures will have to be resorted to to maintain as long as possible the patient's general health and strength. Should there be much difficulty in respiration, tracheotomy ought to be performed at not too late a period. The relief given by that operation is much greater if it be not postponed till the very last, when, often enough, its first result is an acute bronchial catarrh, which takes away still more of the patient's strength. Trecheotomy in these cases ought to be performed low down, so that, if possible, the tracheotomy wound may not be reached by the disease in its further progress. Often enough, considerable subjective improvement will be noticeable if the patient permits of the tracheotomy being performed in time. Should there be great pain from the ulcerating surfaces, cocaine in the form of a spray, or orthoform by means of insufflations, will do palliative service, and in the more advanced stages injections of morphia may have to be resorted to. Should the growth ulcerate externally, applications of bismuth in powder form are of value.

The diet, of course, particularly if the swallowing be painful, should be of a soft, semisolid, bland kind; and finally, feeding either through an œsophageal tube or by means of nutrient enemata may be required.

### Neuroses of Larynx


| | |
|---|---|
| Innervation of the Larynx | 362 |
| Sensory Neuroses | 363 |
| *Anæsthesia* | 363 |
| *Hyperæsthesia* | 364 |
| Motor Neuroses— | |
| *Inspiratory Spasm in Adults* | 364 |
| *Nervous Laryngeal Cough* | 364 |
| *Phonic Spasm* | 365 |
| *Laryngeal Vertigo* | 365 |
| Paralysis of the Vocal Cords— | |
| *Lesions of Superior Laryngeal* | 366 |
| *Lesions of Recurrent Laryngeal* | 366 |
| The Diagnosis of Laryngeal Paralysis | 367 |
| Hypertrophy of the Lingual Tonsil | 369 |


See also Laryngismus Stridulus, p. 373.

The larynx subserves two main functions, viz., phonation and respiration. Phonation is a volitional act, and the nerve centres for this function are mainly represented in the cerebral cortex. There are, however, phonetic acts which are mainly reflex in character, viz., coughing, sighing, hiccough, etc., and these, like other somatic reflexes, are mainly represented in the bulbar centres. Respiration is essentially a reflex act, and therefore the respiratory centre is mainly represented in the bulb; and the bulbar centres (in the dog) have been shown by Horsley and Semon to suffice for respiration after complete removal of the cerebral hemispheres. For these two essentially distinct functions there are two separate sets of muscles, viz., the adductors of the vocal cords for phonation, and the abductors or glottis-openers for respiration.

Innervation of the Larynx.—The larynx receives its nerve-supply from the superior and recurrent laryngeal branches of the vagus nerve on either side; the former supplies sensation to the whole of the mucous membrane of the larynx, and is also the motor nerve to the crico-thyroid muscle. The recurrent laryngeal nerve contains no sensory fibres, except, perhaps, muscle-sense fibres, and is the motor nerve to all the intrinsic laryngeal muscles except the crico-thyroid. It is probable that the inter-arytenoideus muscle receives motor twigs from both the superior and inferior laryngeal nerves of both sides.

Vaso-motor and secretory nerve fibres are supplied to the whole of the laryngeal mucous membrane by the superior laryngeal nerves.

Without entering on the debated ground as to whether the nuclear centres of the motor fibres of the laryngeal branches of the vagus nerve in the medulla are anatomically associated with the spinal accessory nucleus, or with the common glosso-pharyngeal and vagus nucleus, the nucleus ambiguus, it must be admitted that the weight of evidence is in favour of the latter view. In other words, the lower portion of the nucleus ambiguus corresponding to the accessory nerve roots emerging from the bulb, which may be conveniently distinguished by the term vago-accessory, are in this sense the lower roots of the vagus. No confusion will arise from the employment of the term vago-accessory to the motor roots, and the motor nuclei of the motor nerve fibres to the larynx which are contained in the vagus nerves. In addition to this ventral large-celled nucleus or nucleus ambiguus, there is a dorsal small-celled nucleus, the so-called combined nucleus which lies external to the nucleus of the hypoglossal nerve in the medulla; this is a motor root, and it has been suggested that it is a nucleus for unstriped muscle. The sensory nuclei of the vagus are contained in its root and trunk ganglia, from which the axones enter the bulb and pass to the nuclei in the gelatinous substance in the neighbourhood of the fasciculus solitarius.

The cortical laryngeal centres have been located in the anterior portion of the lower extremity of the ascending frontal convolution, thus on the left side forming a part of Broca's speech centre. Thence their fibres pass down through corona radiata and internal capsule to reach the medulla oblongata.

Semon and Horsley have demonstrated in

the cat, and Risien Russell in the dog also, that there are separate cortical centres for abduction and adduction of the vocal cords, and, moreover, these observers proved that *both in the cortex and in the medulla each centre is bilateral in action.* Hence it follows (1) that destruction of the centres on one side cannot give rise to paralysis of one vocal cord, since the remaining centre continues to act equally on both vocal cords; (2) that irritation of *one* centre, either in the cortex or in the medulla, may cause *bilateral* spasm of the vocal cords. As will be seen, these facts are of considerable clinical importance; for—

(1) In unilateral hemiplegia, *e.g.* right-sided hemiplegia with motor aphasia, the movements of the vocal cords are unimpaired.

(2) Paralysis of one vocal cord cannot be due to a cortical lesion (unless it involves both cerebral hemispheres).

The motor fibres for each set of muscles, though running together, are separable into two distinct strands of fibres, both in the recurrent nerve (Russell) and in the internal capsule (Semon and Horsley).

*Semon's Law.*—It is a remarkable fact of great clinical interest, demontrated by Sir Felix Semon, that "there exists an actual difference in the biological composition of the laryngeal muscles and nerve-endings," rendering the abductors more prone to be affected by conditions resulting in paresis and atrophy than the adductors; "whilst the fact that also in central (bulbar) organic affections, such as tabes, the cell groups of the abductors succumb earlier than those of the adductors, points to the probability that similar differentiations exist in the nerve nuclei themselves." Thus in all progressive organic lesions of the centres or trunks of the motor nerves of the larynx, the more vulnerable abductor muscles are first involved, and, unless the lesion is so gross as to cause total paralysis of the laryngeal nerves from the outset, the abductors are for a time alone affected. This vulnerability of the abductors, as compared with the adductors, is known as Semon's law: the order in which the muscles are involved being (1) the abductors or crico-arytenoidei postici (posticus paralysis); (2) the thyro-arytenoidei interni; and (3) lastly, the adductors of crico-arytenoidei laterales.

The motor fibres to the larynx are contained in the vagus nerve as it passes out of the skull through the jugular foramen, whence it descends within the sheath of the carotid vessels, passing through the neck to the thorax.

In the thorax the course of the nerve becomes different on the two sides of the neck. On the *left* side it enters the chest between the common carotid and subclavian arteries, and crosses the arch of the aorta, where it gives off the left recurrent nerve which winds backwards round the aorta, and then ascends to the side of the trachea to the groove between the trachea and œsophagus, and enters the larynx behind the articulation of the inferior cornu of the thyroid cartilage with the cricoid cartilage. On the right side the vagus nerve passes across the subclavian artery, where it gives off the right recurrent branch, which winds backwards beneath this vessel, and, lying on the apex of the right lung, ascends obliquely to the side of the trachea, whence its course to the larynx is the same as on the left side. The superior laryngeal nerve on either side arises from the inferior ganglion of the vagus, whence it descends by the side of the pharynx behind the internal carotid artery, and then, after giving off the external laryngeal branch to the crico-thyroid muscle, pierces the crico-thyroid membrane, and enters the larynx with the superior laryngeal artery.

Obviously in this long course the motor fibres to the larynx may be irritated or compressed by a large variety of pathological conditions, to which attention will be drawn further on. But it will also be noted that the vagus nerves as far as the inferior ganglion, and, beyond that point, the superior laryngeal nerves, contain both afferent and efferent nerve fibres to the larynx, while the recurrent laryngeal nerve is purely motor nerve. It follows that irritation or compression of one vagus (or of the superior laryngeal nerve) may have a bilateral effect, the peripheral irritation being conducted to the bilateral medullary centre, while irritation or compression of the purely motor recurrent nerve on either side can only affect the corresponding vocal cord. These differences afford an explanation of the alternating bilateral spasm and unilateral abductor paralysis sometimes observed in aneurysm of the aortic arch.

## SENSORY NEUROSES

The neuroses of sensation in the larynx comprise anæsthesia, hyperæsthesia, and paræsthesia.

*Anæsthesia* may be partial or complete, and may involve the whole of the laryngeal mucous membrane, or be confined to the epiglottis or the supraglottic portion, and, further, may be unilateral or bilateral. Anæsthesia may be caused by peripheral lesions, *e.g.* injury to the nerve, diphtheria, etc., or to central lesions, as in bulbar paralysis, tabes dorsalis, epilepsy, and is not infrequently due to hysteria. It is generally associated with motor paralysis of various laryngeal muscles.

*The symptoms* consist mainly in the tendency for food to enter the larynx and produce attacks of choking. It is especially dangerous when the anæsthesia is complete and involves the subglottic region, as then no laryngeal spasm and cough result, so that the food particles are prone to pass into the lower respiratory tract and set up "foreign-body" pneumonia.

*The diagnosis* can only be made with certainty after touching the laryngeal surface with a probe, when the defective sensation can readily be detected.

*Hyperæsthesia.*—A variety of sensations described as rawness, constriction, or tickling, are encountered in anæmic, hysterical, or hypochondriacal patients. Such sensations may be caused by reflex irritations from enlarged faucial or lingual tonsils; but the purely neurotic cases are usually associated with other vague sensations in the region of the pharynx, local causes from the sensations complained of being entirely absent. Of course, sensations of pain or pricking are met with in many organic diseases of the larynx, but such cases do not come under the designation "neuroses."

*The diagnosis* is to be made by the exclusion of organic causes for the sensations, and the concurrent symptoms pointing to a neurotic temperament which are very seldom wanting. The coexistence of laryngeal paralysis involving the abductor muscles would strongly suggest some organic lesion as the real cause of the symptoms of hyperæsthesia.

*Treatment of Sensory Neuroses.*—The exhibition of nerve tonics and the adoption of general hygienic measures is indicated in all these neuroses. When anæsthesia is due to diphtheria, local faradisation and the treatment of any organic affection will demand attention. When laryngeal anæsthesia results in the escape of food into the larynx, it may become necessary to feed the patient by means of a stomach-tube or by rectal enemata.

## MOTOR NEUROSES

The clinical affections comprised in the group of laryngeal neuroses may be conveniently described under three headings: (i.) Spasmodic affections; (ii.) Neuroses of incoördination; (iii.) Paralytic affections.

### *SPASMODIC AFFECTIONS OF THE LARYNX*

#### A. Respiratory Glottic Spasm

1. *Laryngismus stridulus* or "*false croup*," and *Laryngeal stridor*, see p. 372 *et seq.*

2. *Inspiratory Spasm in Adults.*—Laryngismus stridulus is essentially an affection of childhood, and dependent on conditions which are not observed in adult life; the same may be said of the affection congenital laryngeal stridor, which, though not, strictly speaking, a neurosis of the larynx, closely resembles in its clinical aspects laryngismus stridulus. In adult life, spasm of the glottic sphincters is usually a *reflex* phenomenon dependent on morbid conditions in the larynx or in other parts of the respiratory tract —*e.g.* the presence of growths; catarrhal, tubercular, or other affections in the larynx itself; or the pressure of neoplasms, aneurysms on the laryngeal motor nerves. It may be caused by an elongated uvula, adenoid hypertrophy at the base of the tongue, or it may be set up by the excessive irritability resulting in gouty or rheumatic laryngitis.

*Functional* inspiratory spasm, the so-called "hysterical spasm," is liable to arise in hysterical females from slight causes, such as emotional disturbance; it is generally incomplete and transient, but has been known to be prolonged till consciousness is lost. Functional spasm in the larynx is sometimes associated with pharyngeal or œsophageal spasm.

Certain organic affections of the motor nerve centres are liable to be associated with laryngeal spasm, *e.g.* tabes dorsalis with laryngeal crises, hydrophobia, tetany.

*Diagnosis.*—It is important to recognise the existence of any organic disease in patients complaining of laryngeal spasm. Paroxysmal glottic spasm, accompanied by a peculiar and characteristic brassy cough, is often one of the earliest indications of intrathoracic aneurysm; laryngeal crises in tabes dorsalis are usually associated with abductor paralysis of one or both vocal cords, while bulbar crises may follow diphtheria. Further examination of the patient would reveal the existence of such sources of laryngeal spasm. Hysterical spasm may generally be detected by directing the patient to phonate during laryngoscopic examination, the prolonged utterance of a note being usually followed by reflex abduction of the cords as soon as the breath is exhausted. Indications of the gouty diathesis, or of local abnormalities in the larynx, may be held responsible for the occurrence of the laryngeal spasm only after the elimination of the graver conditions to which allusion has been made.

#### Nervous Laryngeal Cough

The "barking cough of puberty," as it was termed by Sir Andrew Clark, or laryngeal chorea, is really one of the convulsive tics, and is not in any way associated with voluntary laryngeal function. It occurs in young persons, both males and females, about the time of puberty. The cough is a single, peculiar, loud, harsh bark; sudden in onset, persisting at irregular intervals throughout the day, but generally ceasing during sleep. The voice is not affected.

The absence of expectoration or of any lung symptoms, coupled with the peculiar character of the cough, renders the diagnosis easy.

*Spasmodic Laryngeal Cry.*—The so-called "hydrocephalic cry," believed by Trousseau to be characteristic of cerebral meningitis, may rarely occur in various conditions associated with cortical irritability, and may be accompanied by spasmodic contraction of other muscles.

*The treatment of respiratory glottic spasm* consists almost entirely in the general treat-

ment of the underlying conditions which have been referred to. For the laryngeal spasms of tabes dorsalis the inhalation of nitrite of amyl may give relief, and sometimes the attacks, if slight, may be kept off by spraying cocaine into the larynx. It is important in all affections attended with laryngeal spasm to avoid, as far as possible, everything which may cause laryngeal irritation, such as smoking, etc.

### Phonatory Glottic Spasm

Phonic Spasm, in which spasm of the adductors and tension of the vocal cords occurs only during vocalisation, is a somewhat rare affection usually met with in professional voice-users. It is essentially an occupation neurosis, and is rarely seen except in those of a highly nervous temperament. In some cases ordinary conversation is not interfered with, the impairment or loss of voice only occurring during attempts at public speaking or singing.

In its earliest manifestation there is weakness or loss of voice commencing soon after the patient begins to read, speak, or sing. In course of time the difficulty increases, until every attempt to use his voice only results in futile endeavours to force a current of air through the spasmodically closed glottis—the glottic closure, however, ceasing as soon as he desists from his attempts to phonate.

*The treatment* of this affection is often disappointing, as is the case with all occupational neuroses. Any faulty method of producing the voice should be corrected, and the patient should abstain for a time from all the conditions associated with the occurrence of the spasm. Prolonged rest and the exhibition of nerve tonics will sometimes result in curing the less pronounced cases.

Laryngeal Vertigo.—An affection characterised by a series of coughs, followed by glottic spasm, and transient, partial, or complete loss of consciousness, which is not followed by stupor or other indications of epilepsy, was originally described by Charcot as "laryngeal vertigo." The term is unfortunate, inasmuch as true vertigo is hardly ever present in this disease.

The precise nature of the attack has not been definitely settled; it has been regarded by different observers as a form of epilepsy (*petit mal*), as due to syncope, or as the result of forced expiration with a closed glottis. M'Bride has put forward the last-named theory, and Weber has shown that a somewhat similar condition can be produced voluntarily by forced expirations with a closed glottis. In support of the "petit mal" theory, it may be said that the sudden partial or complete loss of consciousness, with rapid, complete recovery, is sometimes attended with indrawing of the thumb on the palms, or with epilepsy, and that the attacks are often indistinguishable from petit mal. Getschell, who collected reports of forty-one cases, considered that the average age of the patients was opposed both to the "epileptic" and the "forced expiration" theories. Of the 41 cases, loss of consciousness during bad attacks was reported in 32 cases and falls in 26. True vertigo was mentioned in 1 case only; in 5, slight mental confusion and dizziness in sight was noted. Bronchitis is present in some cases. In one case coming under my own observation the attacks were always the result of pressure over the laryngo-tracheal region. A few short coughs were rapidly followed (not suddenly) by partial loss of consciousness, which became complete only after an appreciable period, and then persisted for several minutes. The patient, a boy, was certain that his respiratory embarrassment was expiratory only, not inspiratory.

*Treatment.*—The patient is always of the nervous temperament, but is generally healthy. Any catarrhal condition of the respiratory tract should be corrected by appropriate treatment. General hygienic measures and the administration of bromides may prove beneficial. In my own case, the attacks were cut short by the application of a sponge with very hot water to the throat externally. The inhalation of nitrite of amyl would probably have relieved the glottic spasm, but I have no knowledge of any case of the kind in which it has been tried. The patient usually recovers consciousness so rapidly without any assistance, that it is only in exceptional attacks that any treatment during an attack could be required.

### Paralysis of the Vocal Cords

Vocal cord paralysis obtains a far-reaching clinical significance, not alone on account of the inconvenience or danger that may be caused from the resulting loss of voice or urgent dyspnœa that may result, but also on account of the valuable aid that such paralysis may afford in the diagnosis of many diseases in other regions; indeed, a laryngoscopic examination revealing a vocal cord paralysis may afford the one and only definite physical sign pointing to the existence of some grave organic disease such as tabes dorsalis, aortic aneurysm, etc. The subject will be much simplified by grouping the varieties of laryngeal paralysis together in discussing their etiology and clinical significance. The section will, therefore, be considered in the following order:—

I. The signs and symptoms of the various forms of paralysis resulting from implication (*a*) of the superior, and (*b*) the recurrent laryngeal nerves.

II. The etiology and pathology of laryngeal paralysis.

III. The treatment of the various forms of laryngeal paralysis.

IV. The clinical significance of laryngeal paralysis.

Paralysis of the Muscle supplied by the Superior Laryngeal Nerve.—The only muscle supplied by the superior laryngeal nerve is the crico-thyroid, the action of which is to assist in rendering tense the corresponding vocal cord. Paralysis of this muscle alone is rare; but it obviously must occur in association with the anæsthesia of the larynx resulting from section of the superior laryngeal nerve, and has also been described as resulting from cold, diphtheria, pressure of growths, etc.

When the crico-thyroid muscle is paralysed, the vocal cord presents a wavy outline, bulges up in the centre in forced expiration, and is depressed on inspiration, these phenomena being due to the defective tension of the cord.

*The treatment* is essentially the same as for laryngeal anæsthesia when that is present, and which is due to implication of this nerve. For weakness or paralysis of the muscle, external faradisation is sometimes called for.

Paralysis of the Muscles supplied by the Recurrent Laryngeal Nerves. — As stated above in progressive organic lesions involving the motor nerves of the larynx, the muscles succumb in the following order—abductors of the cords, internal tensors, adductors; and it will be convenient to follow the sequence in the description of the various forms of paralysis of the recurrent nerve fibres.

Abductor or Posticus Paralysis.—*Unilateral Posticus Paralysis.*—The vocal cords are abducted by the crico-arytenoideus posticus muscle on either side, conveniently spoken of as the posticus muscle, paralysis of which results in the vocal cord being maintained in the median line owing to the normal tonus of the adductor muscle not being counterbalanced, so that even on deep inspiration the affected muscle persistently remains in the middle line, *i.e.* the phonatory position. As speech is not interfered with, and the normal abduction of the unaffected cord leaves sufficient space for quiet respiration, there are no symptoms to direct attention to the larynx, and therefore the condition is frequently overlooked.

The left cord is most frequently affected, and is generally the result of pressure on the left recurrent nerve by an aneurysm of the aortic arch. Foreign bodies in the œsophagus, cancer of the œsophagus, mediastinal growths, goitre, and on the right side tubercular disease at the apex of the lung, or aneurysm of the innominate artery, are all possible causes.

*Bilateral Posticus Paralysis.* — When both cords are affected they remain in the median line, the glottic aperture being reduced to an extremely narrow aperture. Respiration, of course, is greatly embarrassed, and during attacks of dyspnœa the vocal cords are liable to be drawn together by the violent inspiratory effects, so that very little air can enter the chest, and urgent or fatal asphyxia may at any time arise. The voice is unaltered, and it may be difficult for the patient to realise that he is the subject of a very dangerous form of vocal cord paralysis. Fortunately, it is rarely that both cords are simultaneously affected with an extreme degree of posticus paralysis, so that with few exceptions the posticus paralysis is incomplete, or the implication of the recurrent nerve has gone beyond posticus paralysis, and has resulted in total paralysis. The chief causes of bilateral paralysis of the abductors are nuclear degeneration in the bulb due to syphilis, diphtheria, or tabes dorsalis, or bilateral enlargement of the thyroid gland. Occasionally aortic aneurysm involves both recurrent nerves.

Paralysis of the Internal Tensors of the Vocal Cords. — The action of the thyro-arytenoideus internus muscle is to make tense and straight the free margins of the cord during phonation, coughing, etc., paralysis of the muscle, causing the edge of the cord to be slack and concave in outline, so that the margins of the two cords are imperfectly approximated, and leave an elliptical space during attempted phonation. The voice is consequently weak, husky, or altogether lost, but respiration is not interfered with.

This is the commonest form of myopathic laryngeal paralysis, and generally results from catarrhal laryngeal conditions, or from overstraining of the voice — except when it is associated with abductor paralysis, and is but one of the series of laryngeal muscles involved in lesions which progress to complete vocal cord paralysis.

Paralysis of the Adductors of the Vocal Cord.—The vocal cords are adducted by the crico-arytenoidei laterales muscles, which cause them to meet in the median line; though for adduction of the cords to be complete, the arytenoid cartilages must be simultaneously approximated by the arytenoideus and thyro-arytenoidei externi muscles.

*Unilateral paralysis of the adductors* alone is extremely rare. It would resemble in appearance a complete paralysis of one vocal cord, but might be distinguished by observing the larynx, not only during phonation and quiet respiration, but also during deep inspiration, when, if the adductor was paretic only, further abduction would take place. In complete adductor paralysis the vocal cord would be completely abducted, and show a concave margin.

*Bilateral Adductor Paralysis.*—This form of paralysis is hardly ever complete. The paretic adductors during voluntary phonation are able to approximate the vocal cords in some measure, but not sufficiently to make them meet, consequently the patient is aphonic, though abduction, and, therefore, respiration, is not interfered with.

The causes are nearly always cortical and

functional, adductor paralysis being due either to hysteria or to general weakness.

*Paralysis of the inter-arytenoideus muscle*, at any rate when apparent, is always bilateral. The action of arytenoideus is to approximate and to rotate outwards the arytenoid cartilages during phonation. Paralysis of this muscle results in a triangular chink being left between the vocal processes during phonation; the voice is therefore very weak, or lost.

It is always due either to hysteria or to catarrhal laryngitis.

Total Recurrent Laryngeal Nerve Paralysis results in complete paralysis of the vocal cords, "*laryngoplegia*," or *unilateral complete paralysis* ("laryngo-hemiplegia"). The vocal cord remains in the cadaveric position during respiration, and, except when it is helplessly pushed aside by the over-adduction of the healthy cord, in phonation also.

The normal abduction of the other vocal cord leaves sufficient space for ordinary respiration; but although the voice is sometimes lost, ordinary quiet conversation is generally possible, and the voice is sometimes almost normal from the over-adduction of the healthy cord causing it to pass across the median line to meet its paralysed fellow.

The laryngoscopic appearance is characteristic, for during deep respiration the paralysed cord remains immobile; while in phonation the healthy cord is over-adducted and passes obliquely across the median line, appearing also to lie on a slightly higher level than the paralysed cord.

Diagnosis of Laryngeal Paralysis.—Two questions arise in connection with the diagnosis of laryngeal palsy; firstly, "What muscles are paralysed?" and, secondly, "What is the cause of the paralysis?"

Bilateral adductor or internal tensor paralysis or paresis, or paralysis of the arytenoideus muscle, is easily recognised by the laryngoscopic appearance, and failure of the vocal cords or arytenoid cartilages to come into apposition in the median line during phonation; while unilateral adductor paralysis is never observed, or only so rarely as to constitute a clinical curiosity.

Posticus or abductor paresis may be simulated in nervous patients by partial adduction of the cords during inspiration under laryngoscopic examination. The patient should be instructed to sound a sustained note as long as he can during the laryngoscopic inspection, for when the breath is exhausted an involuntary deep inspiration will be made, and the vocal cords will then abduct to their fullest extent, and then any appreciable diminution in the abduction of one or both cords will be obvious. Complete unilateral, or pronounced bilateral posticus paralysis, is easily detected; the difficulties should only be possible in the earlier stages when the abduction of the vocal cord is defective, not absent.

The greatest diagnostic difficulty is the differentiation between true total paralysis of a vocal cord and anchylosis of the crico-arytenoid joint.

Anchylosis of the crico-arytenoid joint is nearly always the result of inflammatory infiltration in the tissues in the neighbourhood of the capsule, and the obvious swelling or deformity produced usually suffices to distinguish between a mechanical fixation of the cartilages and a true paralysis. But in many cases the inflammatory exudation has subsided, leaving the joint more or less fixed, yet without obvious deformity or swelling; while in others the joint lesion has been of a more chronic character, the "adhesive" form with inflammatory degeneration rather than exudation. I have often been able to distinguish between such cases of anchylosis and a true paralysis by the fact that in total unilateral paralysis the arytenoid cartilage itself is obviously pushed aside by the over-adduction of the healthy cord during phonation, whereas when anchylosis has occurred the arytenoid cartilage remains absolutely fixed under all circumstances. But, in course of time, a simply paralysed cord becomes more or less fixed from prolonged inactivity.

As regards the pathological diagnosis, the following table, taken from Watson William's *Diseases of the Upper Respiratory Tract*, summarises under these headings the various diseases which may cause laryngeal paralysis:—

1. *Cortical Lesions.*—Hysteria.
   (Very rarely indeed organic lesions involving the cortical centres on both sides.)
2. *Bulbar Lesions.* — Nuclear degeneration due to syphilis, diphtheria, locomotor ataxia, general paralysis, disseminated sclerosis, amyotrophic lateral sclerosis, labio-glosso-laryngeal paralysis.
   Syringomyelia.
   Hæmorrhage and softening.
   Tumours.
3. *Peripheral Lesions.*—Pachymeningitis.
   Intracranial new growths.
   New growths in the neck, involving the vagus at the base of the skull.
   Goitre.
   Pericarditis.
   Aneurysm of the aorta, right innominate, or the subclavian or carotid arteries.
   Intrathoracic tumours.
   Cancer of the œsophagus.
   Pleural thickening at the apex of the right lung.
   Enlarged bronchial glands to various tuberculous lesions.
   Injury to the nerves.

Neuritis, either rheumatic, alcoholic, syphilitic; or due to typhoid fever, lead, arsenic, phosphorus, or other toxic causes.

4. *Inflammatory Infiltration of the Muscles.*

Further, paralysis of the larynx may be simulated by mechanical fixation of the crico-arytenoid joint.

It will be observed that paralysis or paresis of the laryngeal muscle may be due to lesions in any portion of the motor nerve tract from the cortex to the termination of the nerve fibres in the muscles; but numerous as are the possible causes, the particular variety of paralysis that must result in the different pathological conditions may be often determined by simply bearing in mind the facts to which attention has been drawn in the introductory remarks on p. 362. It will be seen that—

(*a*) Lesions of the cerebral cortex or internal capsule producing paralysis must involve the centres of both cerebral hemispheres, and are, therefore, almost invariably functional diseases, such as hysteria, or weakness from exhausting diseases, anæmia, etc.

But we have seen that the laryngeal function of respiration (abduction of the vocal cords) is mainly represented in the bulb, whereas the phonatory function is represented mainly in the cerebral cortex; therefore cortical lesions only result in adductor paralysis or paresis of the vocal cords during phonation. Thus we find that hysterical or other functional paresis of the vocal cords involves the adductors only; moreover, as the bulbar centres are still active, the purely reflex act of coughing is attended with normal adduction of the cords. Thus the cough of a hysterical patient is a phonetic cough.

(*b*) Lesions of the bulbar nuclei involve both the postici or the abductor muscles (respiratory), and also, though later in all progressive lesions, the adductors of the cords. The diseases which are liable to be attended with nuclear degeneration in the bulb are tabes dorsalis, bulbar paralysis, general paralysis, syringomyelia, syphilis, diphtheria, etc. In these progressive nuclear degenerations the muscles are involved in a definite sequence, according to Semon's law; and therefore the larnygeal paralysis involves first the postici (abductors), then the thyro-arytenoidei interni, and finally the adductors, with consequent complete paralysis of one or both vocal cords. The bulbar lesion, and, therefore, the vocal cord paralysis, may be unilateral or bilateral, but it will be gradual in onset. But in bulbar lesions, once the adductors are involved, the paralysis is complete on both sides; there is no phonetic cough.

(*c*) Of peripheral nerve-trunk lesions a basal meningitis is likely to involve both sides. Outside the skull, a goitre or a malignant growth of the œsophagus are the only lesions likely to involve both motor nerves. The most common cause of peripheral nerve paralysis of the larynx is aneurysm of the aortic arch, which by gradual pressure on the left recurrent nerve causes a progressive paralysis of the left vocal cord. Here again the vocal cord muscles are involved in the sequence laid down by Semon's law.

Peripheral neuritis involving one or both recurrent nerves may result in enteric fever, pneumonia, diphtheria, rheumatism, alcoholism, etc.

(*d*) Paralysis due to direct involvement of the muscle fibres or of the nerve-endings in the muscle, the so-called myopathic paralyses, are the result of local inflammatory changes, and as any individual muscles may be implicated, according to the seat of inflammation, there is no definite sequence in the order of paralysis.

The most common form of myopathic paralysis is that due to laryngitis, with paralysis or paresis of the internal tensors of the cords, or of the arytenoideus; or one or both adductors may be involved. Any local inflammatory affection, such as tuberculosis, syphilis, perichondritis, may implicate particular muscles.

*Treatment.*—Inasmuch as the great majority of cases of laryngeal paralysis are the result of pathological conditions in other regions, the treatment of the paralysis very often resolves itself into therapeutic measures directed solely toward these outlying causes, and therefore outside the scope of this article.

But there are two groups of laryngeal palsies for which local treatment is desirable, viz., functional palsy, and palsy due to local inflammatory infiltration or to peripheral neuritis.

Functional adductor paralysis in hysterical or anæmic patients is an indication for general hygienic measures, and the administration of nervine tonic, iron, etc. In most cases it is possible to maintain an immediate and lasting cure of the aphonia by intralaryngeal faradisation with a strong current. For this purpose one pole of the battery is connected with the episternal notch externally, and a special intralaryngeal electrode is passed into the larynx under the guidance of the laryngoscopic mirror, and the circuit completed. With a fairly strong current the resulting spasm of the larynx and the pain produced cause the patient to utter an exclamation, and on withdrawing the laryngeal electrode the voice is usually found to have been restored. Sometimes the aphonia recurs at short intervals for a time; but after the restoration of the voice by the intralaryngeal faradisation on two or three occasions, the cure is generally permanent.

In laryngeal palsy due to diphtheria or other forms of neuritis, or in the more persistent forms of paralysis following catarrhal affections, the intralaryngeal faradic current is often of great service, but in these cases a single application

is rarely sufficient; often enough it must be persisted in for a considerable period. In neuritic palsies the submucous injection of strychnine into the affected muscles may be tried with advantage.

In the more gross inflammatory lesions ice should be sucked, and counter-irritation in the form of mustard leaves, or the application of cold wet compresses, will be helpful.

In bilateral abductor paralysis fatal asphyxia may arise at any moment, and therefore the patient should be either placed under such circumstances that tracheotomy can be performed whenever the necessity arises, or else tracheotomy or intubation should be performed. Intubation is not desirable except in those cases in which, owing to the nature of the lesion, recovery from the paralysis is possible.

When laryngeal paralysis is produced by a section of the motor nerve, either in attempted suicide or in the removal of growths in the neck, the cut ends should be sutured, just as in similar lesions of other nerves.

### Hypertrophy of the Lingual Tonsil[1]

The lingual tonsil resembles the faucial pharyngeal tonsils in its development, anatomical structure, and in the pathological conditions to which it is subject. It is, however, developed later than these other aggregations of lymphoid tissues, and in early childhood is often small and ill-developed; and it is partly due to these facts, and partly to the special factors which result in its hypertrophy, that pathological conditions of the lingual tonsil are more prone to appear in adult life.

Chronic enlargement of this tonsil may be due to previous acute lacunar or parenchymatous inflammatory attacks; but it may arise *de novo* as the result of chronic pharyngitis, or of long persistence of any of the many causes which commonly lead to chronic pharyngitis. Irregular rounded masses of the hypertrophied lymphoid tissue may then be observed by simply depressing the tongue, though better seen by the laryngoscopic mirror. The hypertrophic tonsil may overlap or impinge against the upper surface of the epiglottis, concealing more or less completely the glosso-epiglottic fossæ.

In many individuals very considerable enlargement is unattended with symptoms, and these are without any clinical importance. But various symptoms are liable to arise—especially a constantly recurring troublesome cough, a sense of persistent discomfort, or a dragging sensation in the throat, or vocal impairment.

As in chronic pharyngitis, so in lingual tonsillar hypertrophy, long-standing irritation is liable to result in some measure of congestion, and the veins ordinarily seen at the dorsum of the tongue may become enlarged and tortuous. It has been stated by some observers that this enlargement of the veins, which has been dignified with the name of lingual varix, is itself the cause of numerous local and reflex symptoms and of grave discomfort; but from personal examination of a very large number of patients who complained of no throat symptoms whatever, I am able to assert that pronounced enlargement of the dorsal lingual veins is so frequently present in patients past middle life as to be practically a normal condition, and without clinical importance.

A lingual accessory thyroid gland is occasionally developed, appearing as a smooth, firm, round red swelling in the region of the foramen cæcum. It consists of thyroid gland tissue, whereas simple hypertrophy of the lingual tonsil is composed of lymphoid tissue. The symptoms are very much the same in either form of enlargement, and the two conditions may easily be mistaken for one another.

*Treatment.* — Simple adenoid hypertrophy, when productive of symptoms, should be removed, either by repeated applications of iodine in solution when the hypertrophy is only of moderate dimensions, or by ablation with a lingual tonsillotome when considerable in amount.

Galvano-cauterisation is followed by much pain, and in some cases it has resulted in severe attacks of parotitis. For these reasons its employment for reducing the hypertrophy is generally undesirable.

An accessory thyroid gland may be removed either by galvano-caustic snare or by enucleation. But it is necessary to ascertain whether the normal thyroid gland is absent, in which case the lingual thyroid gland tissue should not be extirpated for fear of causing myxœdema.

## Affections of the Cartilages

1. Perichondritis—
*Etiology and Pathology* . . . 369
*Symptomatology* . . . . 370
*Diagnosis* . . . . . 371
*Prognosis* . . . . . 371
2. Anchylosis of the Crico-arytenoid Joint . . . . . 371
3. Stenosis of the Larynx . . . 372
Treatment . . . . . 372

### 1. Perichondritis

*Etiology and Pathology.*—Perichondritis of the larynx may be defined as an inflammation of the perichondrium covering the laryngeal cartilages, characterised in some cases by suppuration, with necrosis and exfoliation of the cartilage in whole or in part, in other cases by a plastic inflammation, with the formation of a new fibrous connective tissue.

[1] Although in no wise a neurosis of the larynx, it is convenient to describe this condition in the present section (*vide* Symptoms, *supra*).

The cartilages of the larynx, the surfaces of which are covered with perichondrium, are the cricoid, thyroid, two arytenoids, and the yellow fibro-cartilage of the epiglottis. Their various surfaces lie in relation to the interior of the larynx, the œsophagus, the pharynx, and the subcutaneous tissue of the neck. As the inflammation very rarely attacks the whole larynx, and sometimes only part of one cartilage, the symptoms and signs will vary according to the surface thus affected. Perichondritis may spread from one aspect to the other, so that an inflammation of the thyroid cartilage, which may in the first instance be entirely extra-laryngeal, may later involve its deep surface. Further, the disease may spread from one cartilage to another, and even to the upper rings of the trachea. The arytenoid cartilages are most frequently affected, probably from the fact that tubercular ulceration is most common in that region; the cricoid cartilage occupies the second position in order of frequency. It occurs more frequently in males. The disease may be of *primary* origin, or *secondary* to a pre-existing laryngeal lesion. A few cases of primary affection have been recorded to which no definite cause could be assigned. It is probable that some of these cases at any rate were of the nature of a local septic infection. Perichondritis is much more frequently a secondary affection, and occurs in the course of tubercular, syphilitic, and malignant disease of the larynx. It is also the common form of the laryngeal complication which arises in typhoid fever, and it is met with also in smallpox, scarlet fever, and diphtheria. Further, it may be secondary to deep-seated suppuration in the neck, or originate as a metastatic abscess in acute general septic conditions. Perichondritis may also be of traumatic origin, occurring after cut throat or other wounds of the larynx, or as a sequel to scalds and the action of corrosive irritants. It may follow the lodgment of foreign bodies, or the frequent introduction or retention of œsophageal tubes, while more than one author considers that the pressure of the larynx against the bodies of the cervical vertebræ in the prolonged dorsal decubitus of old people may set up an inflammation of this nature.

Inflammation of the perichondrium is characterised in its earlier stages by small cell infiltration and thickening of the fibrous covering, and by serous exudation beneath it, while a considerable amount of œdema may permeate the surrounding submucous tissue. Subsequently pus forms under the perichondrium, and as the cartilage thus becomes deprived of its nourishment, necrosis and separation in whole or in part may result. When the abscess thus formed breaks through the mucous membrane the pus is discharged into the larynx, pharynx, or œsophagus, or even externally under the skin, according to the situation of the perforation. In the latter event a fistula is formed, a condition which may be still further complicated by the occurrence of subcutaneous emphysema. The necrosed cartilage may be coughed up or discharged through the fistulous opening. In milder forms of the inflammation no suppuration and destruction of cartilage takes place, but the perichondrium becomes thickened in consequence of the formation of new fibrous connective tissue. As a result of these inflammatory changes considerable cicatrisation, permanent thickening, and deformity take place with consequent stenosis of the larynx. Another important sequela of perichondritis of the arytenoid or cricoid cartilages, and one of considerable clinical importance, is anchylosis of the crico-arytenoid joint, with impaired mobility or complete fixation of one or both vocal cords; this subject will presently be referred to in more detail.

*Symptoms and Signs.*—The local symptoms met with are hoarseness and aphonia, cough, pain, difficulty in swallowing, and finally dyspnœa, all of them symptoms which may occur in other conditions. They vary, however, and are considerably modified according to the severity of the attack and the site of the lesion. In the acute cases a considerable amount of constitutional disturbance occurs. If the inflammation attacks the laryngeal surface of the thyroid cartilage, interference with the voice is an early symptom; but if the arytenoids are affected, dysphagia in addition is complained of. If the lesion is confined to the posterior surface of the cricoid cartilage or the epiglottis, difficulty in swallowing may be the only symptom. Dyspnœa usually occurs in the later stages of perichondritis, when the swelling becomes marked; but it must be borne in mind that sudden dyspnœa may supervene even in the early stages of thyroid and cricoid perichondritis. The detection of fragments of cartilage in the sputum renders the diagnosis certain. If the inflammation is confined to the external surface of the thyroid and cricoid cartilages, as may be the case in the early stages of certain cases, swelling in the neck and pain with increased tenderness on palpation may be the only indications of the local condition.

The laryngoscopic appearances also vary considerably. If the arytenoid cartilage is affected there is considerable swelling in that region, which in some cases closely resembles the pear-shaped mass seen in tubercle of the larynx. If the posterior part of the cricoid is at the same time involved, thickening of the posterior laryngeal wall is especially noticeable. There may be impaired movement or complete immobility of one or both vocal cords. A small yellow spot upon the mucous surface is an indication that the abscess is pointing. Should this already have burst, the pus may be visible. If a probe can be successfully introduced into the

sinus, the denuded cartilage may be felt. Involvement of the cricoid cartilage, either along with the arytenoid or alone, may be evidenced by swelling of the laryngeal posterior wall, of the aryepiglottic folds, or of that surface of the larynx which is directed outwards to the pyriform sinus. One or both vocal cords may be fixed, perhaps, in the middle line as the result of destruction of one or both of the posterior crico-arytenoid muscles. In some of the early cases swelling may be detected beneath the cords, the movements of which are somewhat impaired. When the inflammation attacks the laryngeal surface of the thyroid cartilage swelling may be observed either above or below the anterior commissure of the cords, and tending to occlude the glottic chink. In the former case the true cords may be more or less concealed from view. Should the external surface of this cartilage be affected, and present those signs already indicated above, examination with the mirror may assist the diagnosis by disclosing the fact that the mucous membrane on the affected side is reddened, and the mobility of the vocal cord impaired. Should the epiglottis be affected its posterior surface may present considerable swelling, which is seen to extend downwards on to the aryepiglottic folds and false cords, simulating the œdematous infiltration observed in tubercle. The abscess may point and rupture near the free margin of the epiglottis, or it may burst at a more dependent part, and the sinus thus be invisible by laryngoscopy.

*Diagnosis.*—From the foregoing description, it is evident that the diagnosis of perichondritis of the larynx is sometimes beset with difficulties. The clinical picture is not a distinctive one. Neither the symptoms nor the local appearances can be described as characterisic of the condition. In the majority of cases they are identical with those of the primary disease of which the perichondritis is merely a secondary complication. The ulceration and infiltration of tubercle, syphilis, and malignant disease may later be marked by the onset of this complication, while a considerable amount of acute œdema may obscure not only the original disease but also the perichondritis. If it can be ascertained from the history that laryngeal symptoms have existed for a space of time, and if those symptoms have become somewhat suddenly aggravated and possibly accompanied by difficulty in respiration, the existence of this complication must be suspected. If the mirror reveals at the same time considerable swelling and a yellow area on the surface of the mucosa, signifying the existence of pus, or if necrosed cartilage can be detected with the probe or discovered in the sputum, the diagnosis can no longer be a matter of doubt. The diagnosis between true paralysis of a vocal cord and the fixation following the more chronic adhesive form of perichondritis, or an anchylosis of the crico-arytenoid joint, is sometimes very difficult. In some cases, again, the diagnosis may only be cleared up by observing the result of treatment, while in others the exact condition is not ascertained until a post-mortem examination has been made.

*The prognosis* as regards life must depend to a considerable extent upon the nature of the primary affection. In tuberculosis and malignant disease it is grave, while in syphilis or following traumatism it is more favourable. Death, however, may occur suddenly from asphyxia quite independently of any dyscrasia; marked increase in the swelling, perhaps the result of œdema, the rupture of an abscess, or the lodgment of a piece of cartilage in the glottis, may cause sudden death. A fatal termination from septic pneumonia may follow the introduction of pus into the bronchi. In those cases in which the patient's life is not threatened, the prognosis as regards the function of the larynx must be extremely guarded. In some cases the resulting stenosis may be so marked that respiration through the glottis is no longer possible, and the constant wearing of a tracheotomy tube becomes necessary. In others, again, the voice remains affected, some degree of hoarseness or aphonia bearing witness to the permanent deformity which has resulted.

### 2. Anchylosis of the Crico-arytenoid Joint

Impaired movement or complete fixation of this important joint may occur from a variety of causes. The anchylosis may be true or false according to the existence of changes within or external to the joint capsule. Sometimes the fixation results from a luxation of the joint surfaces. As we have already shown that anchylosis may follow perichondritis of the arytenoid and cricoid cartilages, it follows that the various conditions already enumerated as etiological factors of the former must also be regarded as causes producing anchylosis. To the different affections enumerated above we must add as further causes the changes met with in and around the joint in gouty individuals, and the neuropathic and myopathic paralyses which produce secondary joint changes resulting from disuse.

As a result of the anchylosis the movements of one or both vocal cords, as the case may be, are impaired or lost. A varying amount of infiltration and swelling in and around the joint exists in most cases as a sequel of the previously existing inflammatory process. The position of the cord varies according to the position in which the joint has become fixed, and this will vary from that of full adduction to that of complete abduction. Where the anchylosis is produced by cicatricial contraction (**false**), these extreme positions of the cords are more frequently found, while in true anchylosis the cord more frequently is fixed in an intermediate position (cadaveric).

*The symptoms* of this affection must therefore vary considerably; they consist mainly in alterations in the voice and in some degree of dyspnœa, both being determined by the position of the affected cord or cords. The voice may be unaltered; it may be husky or completely lost. Dyspnœa, which may be marked, results from the fixation of both vocal cords near to each other.

*The diagnosis* is sometimes difficult, in other cases impossible, especially when from the absence of any thickening about the arytenoid cartilage a differentiation from true nerve paralysis is practically impossible. Semon lays considerable stress upon the following diagnostic points: the presence of tumefaction round an immobile arytenoid cartilage or an abnormal position of the same; the presence of cicatrices or cicatricial distortion; and lastly, fixation of the vocal cord in the abducted position.

### 3. Stenosis of the Larynx

After what has already been written upon perichondritis and anchylosis of the crico-arytenoid joint in the two previous sections, little remains to be added upon the subject of laryngeal stenosis. In addition to the many laryngeal affections there enumerated, which may lead to some degree of narrowing of the lumen of the larynx, we must mention a few in which no antecedent perichondritis is found. To these must be added congenital webs or adhesions between the vocal cords; the false membrane of diphtheria; the acute œdema complicating septic inflammations, and the presence of suspected foreign bodies; and lastly, bilateral abductor paralysis of the vocal cords of neuropathic origin.

Treatment.—The treatment of perichondritis and its sequelæ must be considered under three heads:—

1. The treatment of the acute stage of the inflammation.
2. The relief of dyspnœa.
3. Treatment of the resulting deformity (stenosis).

1. During the stage of acute inflammation the patient must remain in bed, and absolute rest of the voice must be insisted upon. Cold may be applied to the larynx externally by means of a Leiter's coil or by an ice bag, while further relief may be obtained by the sucking of ice. Some recommend the application of leeches over the larynx. If the pain is severe, opium is necessary; the food should be soft, non-irritating, and cold. Sometimes all the nourishment must be given by means of enemata.

In the syphilitic cases potassium iodide should be administered internally in conjunction with mercurial inunction. If an abscess bursts and continues to discharge, tonics and a nourishing diet become necessary.

2. If dyspnœa threaten, scarification of the swelling may afford the necessary relief; or in the event of the abscess pointing, incision should be practised. Intubation may be possible; but if these methods fail to give relief, or if the case has become an urgent one, tracheotomy must be performed.

3. The treatment of the resulting stenosis, although a subject of great importance, can only be briefly dealt with here. Dilatation with intubation tubes or bougies, thyrotomy or the permanent use of a tracheotomy tube, are the means at our disposal for such treatment. Gradual dilatation by means of O'Dwyer's tubes has been successfully practised in those cases of stenosis which have resulted from chronic cicatricial contraction of the glottis, if the commencement of such treatment has not been too long delayed. The size of the tube introduced is from time to time increased. Similar results have been obtained by the temporary introduction of Schroetter's tubes and specially devised cannulæ. In a large number of cases, however, dilatation does not prove satisfactory, and the patient is subjected to considerable discomfort and annoyance without any advantage accruing. Sometimes the thyroid cartilage is split (thyrotomy or laryngo-fissure), the parts being thus thoroughly exposed, and the infiltrated tissue dissected off with the object of enlarging the glottic aperture. In spite of care taken in the after-treatment to maintain the lumen of the larynx by the passage of bougies, a relapse to the former condition follows in a number of cases. The wearing of a tracheotomy tube permanently becomes in many instances a necessity, which gives, however, to the patient the greatest possible amount of comfort under the circumstances. Not only is the risk of respiratory difficulty in this way overcome, but he is able, by placing his finger upon the outer end of his tube, to converse with those about him often with considerable success.

## Congenital Laryngeal Stridor

Synonyms: *Infantile Laryngeal Spasm, Infantile Respiratory Spasm, Respiratory Croaking, Congenital Laryngeal Obstruction.*

*Definition.*—A condition of noisy breathing, due to interference with the free entrance of air into the larynx, which begins at or soon after birth, lasts more or less continuously for many months, and disappears spontaneously before the end of the second year.

*Clinical Features.*—In a typical and uncomplicated case of congenital stridor, the infant who appears normal in other respects is noticed immediately, or within a week or two after birth, to have noisy breathing. The noise consists of a crowing sound accompanying inspiration, which rises to a high-pitched crow when a longer or more vigorous breath is taken. Expiration is often accompanied by a short crow

when the stridor is loud, but at other times it is noiseless. Even in the most severe cases there are occasional brief intervals during which there is no sound audible; but with this exception the stridor goes on constantly when the child is awake, and sometimes even when he is asleep. Any emotional excitement or any physical cause of deeper breathing, such as exposure to colder air or exertion on sucking, is apt to intensify the sound. The child's power of crying and coughing is quite unaffected. Although the breathing is noisy it is not accompanied by the slightest distress, and there is no cyanosis. There is, however, always marked inspiratory indrawing of the thoracic abdominal walls, except in the very slightest cases.

The stridor increases in loudness during the first few months, and after remaining about the same for a few more months, gradually lessens and disappears spontaneously in the course of the second year. Long after it ceases to occur habitually, however, it is apt to be set up by emotional causes.

*Etiology.*—Great difference of opinion has been expressed as to the causation of congenital stridor. A peculiarity of form of the upper aperture of the larynx is present in most if not in all the cases. This consists in an exaggeration of the normal peculiarities of the infantile larynx. The epiglottis is more folded on itself, and the aryepiglottic folds consequently more closely approximated. They may even be found to be almost touching in cases where the patient has died of respiratory disease accompanied by dyspnœa. Some (Lees, Sutherland and Lack, Variot, Refslund) have regarded this condition as a congenital malformation, and thought it sufficient to cause all the symptoms. By others the symptoms have been attributed to posticus paralysis (Robertson), or to adductor spasm due to adenoids or some other source of irritation (Löri, E. Smith). One writer has even blamed enlargement of the thymus (Avellis).

It is probable, however, that the essential elements of the causation of the condition are two—(1) an arrest of development of the cortical structures which control the coördination of the respiratory movements leading to a choreiform respiratory spasm (not a spasm of the larynx only); (2) the extremely soft collapsible character of the laryngeal structure naturally present in the young infant. These act in the following way:[1]—The ill-coördinated and spasmodic character of the breathing gives rise to a constantly repeated sucking-in of the sides of the upper aperture of the soft larynx, and leads very soon to its remaining indrawn and deformed, exactly as the thorax assumes the form known as pigeon-breast when indrawing of its lower segment is constantly repeated for a long period of time. The stridor is probably produced partly at the abnormally approximated aryepiglottic folds and partly in the larynx proper.

[1] For a fuller account, see a paper by John Thomson and Logan Turner, *Brit. Med. Journ.*, vol. ii., 1900.

*Diagnosis.*—The diagnosis is generally easy. The chief points to be attended to are the time of onset of the symptoms, the evidence of laryngeal obstruction (stridor and chest retraction) without any apparent distress, and the presence of a loud, clear cry and cough.

In cases of compression of the trachea by caseous bronchial glands, the stridor is mainly expiratory, the larynx does not move up and down as in cases of intralaryngeal obstruction, and there is much greater respiratory distress.

*Prognosis.*—Uncomplicated cases tend to complete and spontaneous recovery. The presence of respiratory spasm, however, constitutes a serious complication to inflammatory diseases of the respiratory organs, so that if bronchitis or pneumonia occur the prognosis must be guarded.

*Treatment.*—No form of treatment has usually any effect on the continuation of the stridor, although cases have been recorded (E. Smith) where removal of adenoids was followed by improvement. The child should, of course, be carefully guarded against chills.

## Laryngismus Stridulus


ETIOLOGY . . . . . 373
CLINICAL FEATURES. . . . . 374
DIAGNOSIS AND PROGNOSIS . . . 374
TREATMENT . . . . . 374


SYNONYMS: *Child-crowing, Spasm of the Glottis.*

*Definition.*—Laryngismus has been defined as "a sudden arrest of respiration followed by a long-drawn crowing sound due to inspiration through the narrowed glottis" (Barlow). While this is a good description of the usual type of attack, the arrest of breathing may occur with the thorax in the position of inspiration instead of in that of expiration, and then there is no crowing heard. The most noticeable phenomenon in an ordinary attack is a spasmodic closure of the glottis, but if the seizure is at all severe the other muscles of respiration participate in the spasm to a varying degree.

ETIOLOGY.—1. *Of the Tendency to Laryngismus.*

*Rickets.*—In the great majority of cases laryngismus occurs in rickety children, and it is certain that rickets is far the most important element in its causation from a practical point of view. Whether it is as closely connected with an active rickety process in the cranial bones, as Kassowitz believes, is very doubtful; but its almost invariable association with rickety phenomena, and its rapid recovery under treatment which cures rickets, is beyond dispute.

*Age, Sex, etc.*—There are, however, several other most important etiological factors. The

disease generally sets in between the sixth and twenty-fourth months of life, and it comparatively rarely begins before or after these ages. It is commoner in boys than in girls. Several cases are apt to occur in the same family.

*Reflex Causes.*—Such sources of reflex irritation as painful gums from teething, and adenoid growths, are often thought to have something to do with its causation. Enlargement of the thymus is no longer regarded as a cause; but it is probable that enlargement of the bronchial glands may be, and the presence of hydrocephalus certainly is so.

*Time of Year.*—As Gee and others have shown, the disease is much more prevalent during the first half of the year than in the latter six months. Thus in a 100 consecutive cases seen by the writer, 81 occurred between January and June inclusive, and only 19 between July and December. This seasonal distribution has been attributed to the children having been kept much in the house during the preceding months. It is also probable that the greater prevalence of cold winds (E. and N.) during the spring months has something to do with it.

2. *Of the Seizure.*

Any shock to the nervous system, however slight, and any exertion on the child's part, may bring on an attack in those who are predisposed. Thus, the child very often has a paroxysm on awaking from sleep, if exposed to a draught of cold air, if frightened or annoyed in any way, and during swallowing or straining.

Clinical Features.—Laryngismus does not often begin suddenly in a severe form in children who are perfectly well. In most cases the patient has been out of sorts for a week or two at least, and the attacks are at first very slight, only becoming severe after the condition has lasted for some time. The attacks themselves set in with great suddenness. The child, who has been sleeping quietly or playing in a natural happy way, suddenly stops breathing, looks scared, and throws his head back with the mouth open. The chest is fixed and the body and limbs become stiff; the hands are clenched and the feet rigid; the face turns at first cyanotic and afterwards ashy pale. There may be apparently a short loss of consciousness, although this is not common, and a general convulsion may often come on. After a few seconds of arrested breathing, the glottic spasm relaxes and there is a long inspiration, accompanied by a loud crow which is intermediate in character between the whoop of whooping-cough and the stridor of croup. This is what happens in a severe seizure. There are, however, great differences in the severity of the attacks as well as in their duration and in the frequency of their occurrence. In very many cases a few laboured inspirations, accompanied by crowing, are all that is to be observed; while, on the other hand, the spasm is occasionally so severe and continued that the child dies in it. In some cases there are only a few seizures in the course of the day; in others there may be twenty, thirty, or more. They are generally more frequent during the night.

As has been already mentioned, rickets is present in practically all the cases. We very rarely find laryngismus in advanced rickets with great deformity; but it is common in the comparatively well-nourished, fat, and often rosy-cheeked children in whom the disease, although not far advanced, is actively progressing. The children are very frequently also subject to other nervous manifestations, especially to facial irritability (Chvostek's symptom), tetany, and general convulsions. Thus, in 100 consecutive cases, 69 showed facial irritability, 12 had symptoms of tetany, and no less than 60 were said to have had general convulsions. Convulsions were much more commonly observed in boys than in girls. In many cases in which there is no regular tetany, Trousseau's symptom can be elicited (see "Tetany").

Diagnosis.—The disease with which laryngismus is most commonly confounded is false croup or laryngitis stridula. From this it may be distinguished by the absence of a croupy cough and other signs of laryngeal catarrh, and of any fever; also by the age of the patient, false croup being comparatively rare in children under two years. The history of recent convulsions and the presence of facial irritability, or tetany, are strongly in favour of the condition being laryngismus.

The glottic spasm which is set up by a foreign body in the larynx resembles closely in character that of laryngismus; but the history of the case will usually render the diagnosis easy.

Prognosis.—The prognosis is generally very good in uncomplicated cases, as the great majority rapidly and completely recover under treatment. It must, however, always be guarded, because so long as the child is subject to even a mild form of the disease, a fatal seizure may possibly occur at any moment. Should the child acquire any inflammatory disorder of the respiratory organs, the presence of laryngismus constitutes a dangerous complication.

Treatment.—As rickets is such an important cause of laryngismus, its treatment naturally forms an essential part of the management of the case. The diet is to be revised, and the proteids and hydrocarbons in the food increased if they are deficient. Cod-liver oil is generally indicated, and phosphorus (gr. $\frac{1}{200}$ thrice daily) is also useful. The child must be taken into the open air as much as possible. Rapid and striking improvement almost always follows the regular use of the cold douche, and often this acts like a charm. The douche may be given in the following way:—The child is made

to sit in a bath containing a small quantity of hot water and, immediately a jug of cold water (60° F.) is emptied over his back and shoulders; he is then taken out and thoroughly dried before the fire, and rubbed till he is warm. This may be done once, twice, or even oftener in the day, and is very beneficial even in cases where the child is frightened by it. Sedatives, of which antipyrin and phenacetin are the most useful, may also be given. Should there be constipation or an unhealthy character of the motions, it is well to begin the treatment with a dose of calomel. The inhalation of smelling salts sometimes cuts short a paroxysm.

**Lasanon.**—A bed-pan, night-stool, or obstetric chair (Gr. Λάσανον).

**Lasègue's Law.**—The fact that there is an increase in the reflexes of an organ affected by functional troubles or superficial lesions, while there is a suppression of them in organic lesions.

**Lasègue's Sign.**—A hysterical phenomenon; the person affected is unable to move a part of the body unless she sees it; it is usually associated with tactile and muscular anæsthesia of the affected part. *See* Hysteria (*Symptoms, Motor Disorders, Amyosthenia*).

**Laserol.**—A resinous substance ($C_{14}H_{22}O_4$) obtained from *Laserpitin* (the crystalline substance, $C_{15}H_{22}O_4$, contained in the root of *Laserpitium latifolium*, laserwort or herb frankincense).

**Lassar's Paste.**—A paste or ointment used in the treatment of eczema and other skin eruptions; it consists of salicylic acid, zinc oxide, starch (powdered), and vaseline.

**Lassitude.**—Exhaustion and fatigue, such as are in health produced by exertion, unassociated with any preceding exertion. *See* Typhoid Fever (*Symptoms, Nervous System*).

**Latah.**—A curious mental affection met with in the Malay Peninsula, Java, and in certain parts of Russia, characterised by symptoms which depend on an increased susceptibility to the influence of suggestion. It is a peculiar emotional disease closely allied to those known as dancing mania and the various religious psychopathies. "Under ordinary circumstances the subjects of latah appear in no way different from their neighbours. But on the occurrence of some sudden and startling impression, such as a loud sound or anything calculated to produce a vivid impression, or on witnessing particular movements, or on hearing peculiar sounds, or in response to some overt suggestion by word, movement, or facial expression on the part of an experimenter, they pass into a peculiar mental state in which they involuntarily utter certain sounds or words or execute certain movements. In other instances they will imitate words and movements, or yield themselves to suggestions coming from others, or even from the phenomena of external nature. During their hypnotic-like state, which in some may last for a few moments, in others for an indefinite time, or until removed by a contrary suggestion, although consciousness and intellect are clear, and although strenuous efforts may be made to resist suggestion, the victim is at the mercy of his prompter, and will inevitably follow any lead indicated, no matter the consequences." (Manson.) This extract briefly indicates the leading features of the disease. For further information regarding this the reader is referred to the literature.

LITERATURE.—Clifford Allbutt. *System of Medicine*, vol. viii., with bibliography.

**Latent.**—Concealed or masked; existing, but not evident or fully developed; thus we speak of latent empyema (*see* Nose, Accessory Sinuses, Inflammation of, *Chronic Suppuration or Latent Empyema*), latent hypermetropia (*see* Refraction, *Hypermetropia*), latent peritonitis (*see* Peritoneum, Acute Peritonitis, *Symptoms*), latent strabismus (*see* Strabismus, *Superable Squint*), and latent typhoid fever (*see* Typhoid Fever, *Forms of, Ambulatory or Latent*). The *latent period* in the development of a disease is the incubation time, when the malady is really present in the system but has not yet manifested itself by symptoms. In Physiology, the latent period is that which intervenes between the stimulation and the resulting contraction of a muscle (*see* Physiology, Tissues, *Muscle, Course of Contraction*). Latent is opposed in meaning to *patent*.

**Laterad.**—Towards the side or lateral aspect of anything; similarly formed words are cephalad (towards the head), caudad (towards the tail), dextrad (towards the right side), and dorsad (towards the back).

**Lateral Chain Theory.** *See* Immunity (*Side Chain Theory*).

**Lateral Curvature.** *See* Spine, Surgical Affections of (*Scoliosis*).

**Lateral Deviation.**—Deflection to the side, especially applied to displacement of an organ, like the uterus, which is situated in the middle line. *See* Labour, Protracted (*Lateral Obliquity of the Uterus*).

**Lateral Fillet.** *See* Auditory Nerve and Labyrinth (*Localisation of Seat of Lesion in Nerve-Deafness*).

**Lateral Operation.**—The operation of lithotomy, in which the incision is made on one or the other side of the perineum. *See* Bladder, Injuries and Diseases of (*Calculus, Lithotomy*).

**Lateral Sclerosis.** *See* PARALYSIS (*Primary Lateral Sclerosis, Amyotrophic Lateral Sclerosis*).

**Lateral Sinus.** *See* BRAIN, PHYSIOLOGY OF (*Venous Circulation*); BRAIN, SURGERY OF (*Abscess, Diagnosis, Lateral Sinus Phlebitis*).

**Lateral Ventricles.** *See* BRAIN, PHYSIOLOGY OF (*Lymphatic Circulation*); HYDROCEPHALUS (*Internal*); MENINGITIS, TUBERCULOUS AND POSTERIOR BASIC (*Treatment, Drainage of Ventricles*).

**Late Rickets.**—Rickets developing after the third year but usually before puberty. *See* RICKETS (*Late Rickets*).

**Lateritious Deposit.**—The "brick-dust" sediment or deposit (Lat. *later*, a brick), consisting of amorphous urates, found in the urine in cases of the lithic acid diathesis. *See* URINE, PATHOLOGICAL CHANGES IN (*Urinary Sediments, Amorphous Urates*).

**Latero-.**—In compound words *latero-* means relating to the side, as in *latero-abdominal* (relating to the side and the abdomen, *e.g.* the Sims' position in gynæcology), *latero-dorsal* (relating to the side and the back), *latero-deviation* (displacement to one side), *latero-flexion* and *latero-version* (flexion or version to one side), and *latero-torsion* (twisting to one side).

**Latex.**—Milky fluid, especially that which exudes from various plants, but also the chyle.

**Lathyrism.**—A disease, somewhat resembling beriberi, due to the ingestion of meal from various kinds of vetches or chick-pea (*Lathyrus cicera, L. sativus*, etc.), characterised by spastic paraplegia (and tremors) like that which follows transverse myelitis (with hæmorrhage); lupinosis. It occurs in Italy, India, Kabylia, and France. *See* TOXICOLOGY (*Food-Stuffs, Vegetable, Lathyrism*).

**Lathyrus.** *See* LATHYRISM.

**Latissimus Dorsi.**—The broad muscle of the back. *See* MUSCLES, DISEASES OF (*Congenital Absence*).

**Latitude.** *See* METEOROLOGY (*Temperature as influenced by Latitude*).

**Latrines.**—Public water-closets, in which the closets are arranged side by side and open into a common trough containing water. They are used in schools, barracks, factories, and in the poorer districts of large towns; they have the disadvantage of being somewhat offensive, and they are flushed with difficulty. *See* SEWAGE AND DRAINAGE (*Water-Closets, Trough Closets*).

**Laudable.**—Healthy, in the sense that in the healing of wounds the pus thrown out was regarded as praiseworthy and an efficient agent in the reparative process; no longer used in this sense.

**Laudanum.**—Tincture of opium. *See* OPIUM; MORPHINOMANIA; TOXICOLOGY (*Opium and Morphine*); etc.

**Laughing-Gas.** *See* ANÆSTHETICS (*Nitrous Oxide Gas*).

**Laugier's Hernia.**—A femoral hernia in which the sac protrudes through an opening in Gimbernat's ligament. *See* HERNIA (*Femoral*).

**Laumonier's Ganglion.** — The carotid ganglion.

**Laundries.**—According to the Factories and Workshops Act of 1901, laundries are regarded as factories if mechanical power be used in them, as workshops if it be not. Women and young persons must not be employed in them for more than sixty hours per week, and children for more than thirty hours; the same hygienic provisions must be made for them as for factories or workshops. Public laundries are to be recommended, especially for the washing of infected articles so that these may be kept apart and dealt with separately.

**Laurocerasi Folia.**—The fresh leaves of *Prunus laurocerasus*, the Cherry Laurel, "Bay Leaf." It contains *laurocerasin*, a glucoside, a compound of amygdalin and amygdalic acid, by the decomposition of which in the presence of moisture small quantities of prussic acid are formed. *Preparation*—Aqua Laurocerasi. Contains about $\frac{1}{10}$ per cent of prussic acid. *Dose*—½-2 ʒ. It is not often employed because the amount of hydrocyanic acid present is very uncertain. It may be used as a flavouring agent.

**Laurvik.** *See* BALNEOLOGY (*Norway*).

**Lavage.**—The washing out of the stomach. An india-rubber stomach-tube with a glass funnel and some tubing are needed; the funnel is filled with fluid (water, saline solution, etc.) which passes through the tubing into the stomach tube and so into the stomach; the funnel is then lowered and the fluid flows out again by syphon action; the process is repeated several times till the water comes back clear. Lavage is useful in chronic dyspepsia (especially in the fermentative form), and in dilatation of the stomach, but is not to be employed if there is the suspicion of the presence of a gastric ulcer or thoracic aneurysm. The intestine may also be washed out, but this is usually called *irrigation*. *See* ENEMATA; INDIGESTION (*Treatment, Instrumental Measures*).

**Lavatories.** *See* SEWAGE AND DRAINAGE (*Lavatories, Baths, and Sinks*).

**Lavender.**—The flowers of *Lavandula vera* or *L. officinalis* yield an oil (*Oleum Lavandulæ*), which contains an alcohol (*Linalool*, $C_{10}H_{17}OH$), *Linalool acetate*, and the oil *Cineol* (isomeric, with hydrate of cajuputene), and is given in doses of ½ to 3 m.; official preparations are the *Spiritus Lavandulæ* (dose, 5 to 20 m.) and the *Tinctura Lavandulæ Composita* (dose, ½ to 1 fl. dr.), while the oil of lavender is contained in *Linimentum Camphoræ Ammoniatum*, and the compound tincture is found in *Liquor Arsenicalis*. It has the carminative and stimulating actions of all the aromatic volatile oils, and the compound tincture is used as a red colouring agent (it contains red sanders-wood).

**Laveran's Bodies.**—The crescent- or sickle-shaped bodies found in the blood of malarial patients by Laveran, in 1880. *See* MALARIA (*History*).

**Lavey.** *See* BALNEOLOGY (*Switzerland, Canton Vaud*).

**Law.** *See* BEAUMÉS' LAW; COLLES' LAW; COURVOISIER'S LAW; LASÈGUE'S LAW; PROFETA'S LAW; etc.

**Laxative.**—A mild purgative medicine, such as honey, fruits (figs, prunes, stewed apples), sulphur, treacle, nux vomica, and olive oil. *See* PHARMACOLOGY.

**"Laxoin."**—A synthetic laxative (dihydroxyphthalophenone) recommended in place of the various vegetable drugs whose composition is apt to vary (Oppenheimer).

**Layman.**—The term layman was first used of a non-clerical person, but it now includes those who are outside any of the professions, such as medicine, law, and art; with regard to any one of these professions the layman is an "outsider," one of the laity.

**Lazaretto.**—A leper house or hospital, but more often used now for a quarantine building.

**Lead.** *See also* AMBLYOPIA (*Toxic*); BRAIN, TUMOURS OF (*Diagnosis*); COLOUR VISION (*Acquired Colour Blindness*); CONSTIPATION (*Causes, Drinking Water*); GENERAL PARALYSIS (*Diagnosis*); INSANITY, ETIOLOGY OF (*Toxic Action*); INSANITY, NATURE AND SYMPTOMS (*Etiological Varieties*); LEUCOCYTOSIS (*Toxic*); NERVES, MULTIPLE PERIPHERAL NEURITIS (*Lead Paralysis*); NEPHRITIS (*Renal Cirrhosis, Etiology*); STOMACH AND DUODENUM, DISEASES OF (*General Etiology*); TOXICOLOGY (*Lead*); TRADES, DANGEROUS (*Lead Poisoning*); UNCONSCIOUSNESS (*Lead Poisoning*); VISION, FIELD OF (*Limitation in Lead Poisoning*). — Plumbum; symbol, Pb. The metal itself is of no value therapeutically, and is represented officially by the following salts. 1. *Plumbi Oxidum*, lead oxide, known as "litharge." It exists as red scales made by roasting lead in air; it is insoluble in water but soluble in nitric and acetic acids. When boiled with olive oil it yields an oleate. *Preparation*—Emplastrum Plumbi, consisting of lead oleate or "lead soap." It is contained in Emplastra Hydrargyri, Plumbi Iodidi, Resinæ, and Saponis. 2. *Plumbi Carbonas*, really a mixture of carbonate and hydrate. It is known as "white lead." It is a heavy white insoluble powder. *Preparation*—Unguentum Plumbi Carbonatis. It may be used as a dressing for burns, scalds, and ulcers provided that too large a surface is not affected. An ointment made up with starch and cold cream is recommended for severe sunburn. 3. *Plumbi Acetas*, known as "sugar of lead." It is prepared from the oxide by the addition of acetic acid and water. It deposits in large, porous, tangled masses, with a sweet astringent taste. It is soluble 10 in 25 of water. *Dose*—1-5 grs. *Preparations*—(1) Unguentum Plumbi Acetatis. (2) Suppositoria Plumbi Composita. Each contains 3 grains of lead acetate and 1 grain of opium. (3) Pilula Plumbi cum Opio; lead acetate 6, opium 1, syrup of glucose ⅔. *Dose*—2-4 grs. In the following preparation made from the acetate the lead exists in the form of the *Subacetate*. (4) Liquor Plumbi Subacetatis Fortis (Goulard's extract). A clear, colourless liquid, containing 24 per cent of the subacetate. (5) Liquor Plumbi Subacetatis Dilutus (Goulard's lotion or water). Contains 1 part of the strong liquor in 80. (6) Glycerinum Plumbi Subacetatis. (7) Unguentum Glycerini Plumbi Subacetatis. Externally, the lotions and ointments derived from acetate of lead are used for their astringent and sedative effect. They are applied in cases of moist eczema and in some forms of ulceration. The weak liquor is employed as an injection for gonorrhœa, vulvitis, leucorrhœa, and otorrhœa. It is also used in cases of sprain, and in painful affections of muscles. For this purpose it should be mixed with opium, as in the commonly employed lead and opium lotion, made by mixing 5 grs. of extract of opium, ℥j of the dilute liquor, and ℥j of water. Internally, lead acetate is widely prescribed as an astringent in severe diarrhœa, as in dysentery; and in hæmorrhage from the stomach and intestine, as in gastric ulcer and tuberculosis. For these purposes the pill with opium, and the suppository, are specially valuable. The suppository is also useful in painful hæmorrhoids and in ulcerative conditions of the rectum. 4. *Plumbi Iodidum*, a bright yellow powder almost insoluble in water. *Preparations*—(1) Emplastrum Plumbi Iodidi; (2) Unguentum Plumbi Iodidi. These are sometimes applied in cases of glandular enlargements, especially if there is present at the same time some irritation of the skin; but it is doubtful if they are of any real value.

**Lead Colic.**—Abdominal pain due to lead poisoning, resembling true colic in its characters, often preceded by constipation. *See* LEAD; TOXICOLOGY (*Lead*).

**Lead Palsy.**—A result of lead poisoning, in which wrist-drop is a prominent symptom. *See* LEAD; TOXICOLOGY; TRADES, DANGEROUS; etc.

**Lead Poisoning.** *See* TOXICOLOGY (*Lead*); TRADES, DANGEROUS (*Lead Poisoning*); etc.

**Leamington.** *See* BALNEOLOGY (*England, Muriated Waters*); MINERAL WATERS (*Muriated Saline, without Carbonic Acid Gas*).

**Leaping Ague.**—A form of dancing mania or choreomania.

**Leash Ulcer.** *See* CORNEA (*Phlyctenular Ulcer, Fascicular Keratitis*).

**Leather Dressing.** — An offensive trade, like fell-mongering, in the carrying on of which the pelts are limed, then washed, then immersed in "puer" (a mixture of water, dogs' dung, and pigeons' manure), then bleached, and then tanned; the odours are disagreeable. The law requires cleanliness of the buildings, an ample water supply, and frequent emptying of the lime-pits.

**Leber's Atrophy.** — Hereditary optic atrophy. *See* RETINA AND OPTIC NERVE (*Atrophy of Optic Nerve, Etiology*).

**Lecanopagus.**—Symmetrically united twins in which the union is by the pelves (Pygopagus or Ischiopagus). The word *lecano-*, from the Greek λεκάνη, a dish, means relating to the pelvis; thus lecano-terata are monstrosities in which the malformation affects most markedly the pelvis.

**Lecithin.**—A fat in which one of the acid radicles is replaced by phosphoric acid linked to cholin, hydroxyethyl-trimethyl-ammonium-hydroxide ($C_{44}H_{90}NPO_9$); it is found in the yolk of egg (Gr. λέκιθος, yolk of egg), blood, bile, brain, nerves, semen, etc., and it is now frequently employed as a therapeutic agent in the treatment of anæmia, neurasthenia, rickets, etc. *See* LIVER, PHYSIOLOGY OF (*Regulation of Supply of Fat*); PHYSIOLOGY, TISSUES (*Nerve*); PHYSIOLOGY, BLOOD (*Red Cells, Chemistry*); PHYSIOLOGY, FOOD AND DIGESTION (*Eggs*); PHYSIOLOGY, EXCRETION (*Milk Secretion, Phosphorus Compounds*).

**Lecitogen.**—A powder with a pleasant taste, consisting of a combination of lecithin and cocoa; it has been recommended in doses of three to four tea-spoonfuls daily in the treatment of secondary anæmias.

**Ledesma.** *See* BALNEOLOGY (*Spain*).

**Leeches.** *See also* BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Vascular Lesions, Treatment*); BRAIN, SURGERY OF (*Concussion, Treatment*); KIDNEY, SURGICAL AFFECTIONS OF (*Traumatic Nephritis, Treatment*); MEDIASTINUM (*Growths, Treatment*); NOSE, FOREIGN BODIES, ETC. (*Parasites*); SKIN DISEASES OF THE TROPICS (*Caused by Animal Parasites, Leeches*).—Hirudo, the leech. Two kinds of leech are official—the speckled leech, *Sanguisuga medicinalis*, and the green leech, *Sanguisuga officinalis*. The belly of the former is greenish yellow with black spots, while that of the latter is plain olive green. The body of each is about two inches long, wrinkled transversely, and of a dark green colour with reddish stripes longitudinally. At each end of the body there is a muscular disc, the anterior one being provided with a triradiate mouth by means of which the animal bites. In the pharynx is secreted an anti-coagulating substance which prevents the flow of blood stopping too soon.

Leeches are applied to the skin for the extraction of blood in inflammation or congestion of deep-seated organs — *e.g.* one or two may be placed behind the ear in inflammation of the tympanic cavity, or four or five over the liver when that organ is enlarged and congested. The part of skin selected should be thoroughly washed, and, if necessary, shaved; after which a little milk is dabbed over it. The leech is cleansed by being allowed to swim about in water, and is then applied to the skin in a test-tube or wine-glass. A good specimen should extract from ½-2 ʒ of blood, and when full it will drop off of its own accord. If it is desired to remove it before it is satisfied, the application of strong salt solution will usually make it leave go its hold. When a large amount of blood is to be removed, the flow may be prolonged by applying a hot fomentation after the leeches have fallen off. Should the bleeding continue too long, firm pressure with a pad will usually stop it; but if not, the bite must be touched with a silver nitrate pencil, or perchloride of iron solution. When applied to mucous membranes, as in the mouth, rectum, or vagina, the leech must be introduced in a leech glass—a tube narrowed at one end so that only the head can protrude.

**Leeks.** *See* INVALID FEEDING (*Vegetables, Celery, Sea Kale, and Leeks*).

**Leg.** *See* AMPUTATIONS (*Leg*); ANEURYSM (*Lower Limb*); ARTIFICIAL LIMBS (*Leg—Bucket, Anglesea, Palmer, and Bly Leg*); DEFORMITIES (*Bow-Legs*); FRACTURES (*Leg*); LABOUR, OPERATIONS (*Version*); LYMPHATIC SYSTEM, PHYSIOLOGY AND PATHOLOGY (*Leg*); PUERPERIUM, PHYSIOLOGY (*Œdema of Legs*); PUERPERIUM, PATHOLOGY (*Phlegmasia Alba Doleus*); ULCERS AND ULCERATION (*Leg Ulcers*).

**Legal Medicine.** *See* MEDICINE, FORENSIC.

**Legal's Disease.** — Tenderness and pain on the part of the scalp supplied by the auriculo-temporal nerve, with catarrh of the pharynx and middle ear; cephalalgia pharyngotympanica.

**Legitimacy.**—The fact of being legitimate, *i.e.* in accordance with law; thus we speak of the legitimacy of a child born in wedlock or within a number of months after the death of the father. *See* ILLEGITIMACY.

**Legumens.**—The seeds of the *Leguminosæ*. *See* PHYSIOLOGY, FOOD AND DIGESTION (*Vegetable Food-Stuffs*).

**Leguminosæ.** — A natural order of plants, the pea and bean tribe, containing many seeds used as food; some of them contain a proteid, legumin, or vegetable casein; the vetches also belong to this order. *See* LATHYRISM.

**Lehman's Sign.**—If, during the giving of chloroform, the patient's eyelids keep opening although closed by the anæsthetist, the anæsthetisation will be difficult.

**Leio-.**—In compound words *leio-* (Gr. λεῖος, smooth) means smooth or glossy, *e.g.* leiodermia, leiomyoma.

**Leiodermia.**—A smooth or glossy condition of the skin due to atrophic changes; a form of xerodermia.

**Leiomyoma.**—A tumour composed of unstriped (smooth) muscular tissue; a fibroid, *e.g.* of the uterus.

**Leishman-Body.** — A parasitic body found by Leishman in the spleen in cases of *Kala-azar* or *Piroplasmosis*. *See* DONOVAN-LEISHMAN BODIES; MALARIA; etc.

**Leiter's Coil or Tubes.**—Flexible metal tubes which can be applied closely to any part of the body (*e.g.* the head), and through which cold water can be made to flow; an apparatus for reducing the temperature of a part.

**Lembert Suture.**—A method of closing an opening (wound or incision) in the intestine by bringing peritoneum into relation with peritoneum. The peritoneal and muscular coats are transfixed by the needle transversely on one side of the opening; the needle is then brought out and carried across the opening to transfix the same coats on the opposite side, and in this way the wall is doubled in peritoneum being brought into contact with peritoneum. A modified form of this suture is used to close the uterus in cases of Cæsarean section. *See* INTESTINES, SURGICAL AFFECTIONS OF (*Treatment*).

**Lemniscus.** — A band of nerve fibres passing from nuclei in the medulla upward through the pons and crus cerebri to the cerebrum, where it divides into the lateral and median fillets; the fillet.

**Lemon.**—Lemon Peel, Limonis Cortex; obtained from the fruit of *Citrus medica*, var. *Limonum*, grown in the south of Europe. It contains a volatile oil and a bitter principle. *Preparations* — 1. Syrupus Limonis. *Dose*—½-1 ʒ. Lemon juice is also used in its preparation. 2. Tinctura Limonis. *Dose*—½-1 ʒ. 3. Oleum Limonis. *Dose*—½-3 m. Lemon peel is contained in Infusum Aurantii Compositum and Infusum Gentianæ Compositum. The preparations of lemon peel are used as flavouring agents. *Succus Limonis*, Lemon Juice, is the freshly expressed juice of the ripe lemon. It contains citric acid, both free and in the form of salts, chiefly salts of potassium. *Dose*—½-4 ʒ. Lemon juice is very valuable in the treatment and prevention of scurvy; it is more efficacious than citric acid itself, probably because of the large quantity of potassium salts contained in it. It is also used to relieve thirst, usually in the form of an effervescing drink. *See also* HYDROPATHY (*Water-Drinking*); PHARMACOLOGY; etc.

**Lemonade.** *See* INVALID FEEDING (*Beverages, Lemonade*); LEMON; TRADES, DANGEROUS (*Lead Poisoning, Preventive Treatment, Acidulated Drinks*).

**Lenigallol.**—Triacetate of pyrogallol; used, as an ointment, in eczema and psoriasis (strength, ½ to 5 per cent).

**Lenirobin.**—Tetracetate of chrysarobin, employed in skin diseases.

**Lenitive.**—Soothing, emollient, demulcent, or gently laxative (*e.g.* a lenitive electuary).

**Lenk.** *See* BALNEOLOGY (*Switzerland, Canton Bern*).

**Lennhoff's Sign.**—In cases of hydatid cyst of the abdomen, a furrow is seen to form between the tumour and the edge of the ribs on deep inspiration; this is called Lennhoff's sign.

## Lens (Crystalline).

ANATOMY AND PHYSIOLOGY . . . 379
DISPLACEMENTS . . . . . 380
LENTICONUS . . . . . 381
MICROPHAKIA . . . . . 381
COLOBOMA . . . . . 381

*See also* ACCOMMODATION (*Condition of Lens*); CATARACT; EYE, CLINICAL EXAMINATION OF; EYEBALL, INJURIES OF (*Dislocation of Lens*); GLAUCOMA (*Causes of Secondary Glaucoma, Dislocation of Lens*); REFRACTION.

THE crystalline lens and the cornea are the

principal parts of the eye which have to do with the formation of retinal images. For this reason they are transparent. In addition, the lens is capable of altering its focus, so as to admit of retinal images being obtained of objects lying at different distances from the eye. For this purpose (accommodation, as it is called) the lens is so constructed as to admit of its shape altering in accordance with the state of contraction of the ciliary muscle (see "Accommodation," vol. i.). The lens is an epithelial structure, and as such continues to grow throughout life, though only comparatively slowly after the end of fœtal life, and still more so after adult life is reached.

It develops from a thickening of the ectoderm which comes to lie in contact with the primitive optic vesicle on either side. This thickening becomes involuted and cut off at its neck, so as to form a sac which is eventually filled up mainly, and at first wholly, by the proliferation of the cells of its posterior wall. The portion of ectoderm, on the other hand, which closes over the front of the lens epithelium afterwards becomes the epithelial layer of the cornea.

The cells of the posterior wall of the lens sac become gradually more and more elongated into the so-called lens fibres, whilst those of the anterior wall develop into the single layer of cubical cells which line the inner surface of the anterior capsule, and which exercise an important influence in afterwards maintaining the transparency of the lens.

The more peripheral of these cells, however, those which lie towards the equator of the lens, also undergo an elongation, but not until the lens has become solid. The resulting fibres, the shorter ones of which are nucleated like the cubical cells, have to do with the subsequent growth of the lens, and constitute what is known as the transitional zone.

The development of the lens, so far as its transparency and position goes, is complete before the end of fœtal life. During life the healthy lens remains transparent; at first, too, it is perfectly colourless, though in advanced age it develops a more or less marked yellowish colouration.

It is approximately, though not accurately, centred with the cornea, its axis deviating usually about 5° from that of the cornea, and is held in position by the suspensory ligament or Zonule of Zinn, the fibres of which are firmly incorporated with its capsule mainly at its anterior and posterior peripheral portions. The suspensory ligament has also a firm attachment to the ciliary body and its processes, so that the state of contraction of that muscle influences the degree of tension which the capsule exerts on the lenticular fibres (*vide* "Accommodation").

The lens capsule is a homogeneous transparent membrane whose histogenesis is not altogether clear. Though continuous, it is commonly divided by anatomists into an anterior and posterior position. The anterior capsule is considerably thicker than the posterior. The lens capsule is strong and elastic.

The body of the lens, though elastic, is of much less firm consistency than the capsule. At first its consistency is pretty much the same throughout; but before the age of thirty there has developed in its centre a portion, the so-called nucleus, which always increases in size, and forms, therefore, a larger proportion of the whole lens as age advances. There is, however, no sudden transition between the *nuclear* and the surrounding *cortical* portion.

The formation of the nucleus is due to loss of liquid, and probably also to the absence of metabolic changes. Its consequent greater density causes it to be more highly refractive than the cortex. Owing to this, some reflection of light takes place at its surface; this gives rise to the grey appearance of the pupil in elderly people so different from the pure black which characterises the pupil of the young. The hardening process also causes a diminution of elasticity, and a consequent diminution in the range of accommodation.

Helmholtz has shown that, owing to the gradual change in the density of the successive layers of the crystalline lens, its focal power is greater than if it possessed throughout the same density as it has in its nuclear portion.

The lens exercises a considerable power of absorption of both actinic and heat rays. Its nutrition is supplied by liquid from the ciliary processes which enter the capsule mainly in the region of the equator.

*Displacements* of the lens can only occur where, from accident [1] or disease or faulty development, the suspensory ligament is wholly or partially defective in structure or attachments. Congenital displacement of the lens, ectopia lentis, which is due to defective development of a portion of the ligament usually in the neighbourhood of the fœtal slit, is generally bilateral. It is hereditary, and frequently met with in several members of the same family. In one instance known to the writer seventeen members of a family (in three generations) were affected in this way.

*The diagnosis* of ectopia lentis is easily made. The iris quivers on movements of the eye (iridodonesis), and on examination with the ophthalmoscope mirror part of the margin of the lens is seen to cross the pupil. When the displacement is very slight this may only become visible if the pupil is dilated. In many cases vision is improved by the use of glasses. In some cases concave glasses which correct the myopia existing in that part of the pupil which lies in front of the lens are the most suitable; in other cases the greatest improvement is got by the use of convex glasses correcting the area from opposite

[1] See "Eyeball, Injuries of," vol. iii.

which the lens is displaced. There is often, too, a difference of refraction according to the position of the head, the myopia being greater owing to the falling forwards of the lens when the head is held down. There is a tendency on comparatively slight injuries to the eye, in these cases, for the lens to become dislocated into the anterior chamber.

Traumatic dislocation of the lens is not an infrequent occurrence. The lens may be dislocated forwards into the anterior chamber or backwards into the vitreous. When the blow causing the displacement also leads to rupture of the sclera, the lens may be dislocated out of the eye altogether and lie under the conjunctiva.

*Lenticonus* is a curious and rare anomaly. Most commonly the conical projection occurs in the posterior surface of the lens (lenticonus posterior). These cases are probably connected in some way with developmental changes. The effect of the conical protrusion is to cause a high degree of central myopia. Less frequently a conical projection of the anterior surface has been met with; the pathology of this condition is unknown.

*Microphakia* is an abnormally small lens. In this condition, though otherwise well developed, the lens is very much smaller than normal.

*Coloboma* of the lens is a fairly common congenital effect. Most frequently only a slight flattening or notch is found to exist in the lower or lower and inner portion of the lens equator Sometimes, however, the notch is deep, and is then generally associated with coloboma of the iris, or with ectopia lentis. Different views are entertained as to the cause of this condition. Apparently all cases, at all events, are not due to localised defects of nutrition caused by imperfect closure of the fœtal fissure. It seems likely that in a number of cases a delay in the absorption of the vascular tissues found in the fœtus may cause notching by pressure.

**Lenses.** *See* REFRACTION (*Lenses*).

**Lenticonus.** *See* LENS, CRYSTALLINE (*Lenticonus*).

**Lenticular.**—Having the shape of a lens or lentil (bean), *e.g.* the lenticular muscles of the corpus striatum. *See* ATHETOSIS (*Morbid Anatomy*); BRAIN, PHYSIOLOGY OF (*Lenticular Nucleus*); PHYSIOLOGY, NERVOUS SYSTEM (*Cerebrum*).

**Lentigo.**—A pigmentary affection of the skin, consisting in the formation of small freckles or yellowish spots on the face and hands. *See* SKIN, PIGMENTARY AFFECTIONS OF (*Epithelial Pigmentation*).

**Lentils.** *See* PHYSIOLOGY, FOOD AND DIGESTION (*Vegetable Food-Stuffs, Legumens*).

**Lentin.** — Meta-phenylenediamine hydrochlorate, a white crystalline powder, soluble both in water and alcohol, recommended in the treatment of diarrhœa of children (dose, $\frac{1}{6}$ grain) and adults (*Merck*).

**Leontiasis Hystrix.**—The grave form of ichthyosis or porcupine skin. *See* ICHTHYOSIS (*Ichthyosis Hystrix Gravior*).

**Leontiasis ossea** (megalocephaly) is the disease, first recognised by Virchow, which is characterised by hyperostosis of the facial and cranial bones. The cause of the disease is unknown, and there is no definite evidence that either trauma, rickets, or syphilis are causal factors. The onset is, as a rule, noticed in early life—from the tenth to the thirtieth year—and the disease progresses very slowly, as the patient may live for twenty or thirty years after the first appearance of the disease. A somewhat similar affection is said to occur in monkeys. There are two main forms of leontiasis ossea, but some cases represent conditions intermediate between the two extremes.

1. *Localised hyperostosis* with the formation of "bosses" resembling osteomata, and composed either of cancellous or compact bone, whilst the bone for a varying distance around the bosses shows a diffuse hyperostosis. The bony masses attain a large size, are often symmetrical, affect chiefly the maxillæ, less frequently the nasal and frontal bones; cause great deformity of the skull; encroach on the cavities of the orbit and mouth, on the nose and its accessory sinuses, and exert pressure on various peripheral nerves. The first sign of the disease is usually the development on the upper or lower jaw of one side of a swelling, which very slowly enlarges. The further symptoms and deformity vary according to the site and bulk of the osteomatous masses; exophthalmos is the result of partial obliteration of the orbital cavity, whilst epiphora and interference with nasal respiration and with the taking of food may also occur. After the disease has lasted for a variable time the patient suffers from the effects of pressure on various nerves, as in diffuse hyperostosis.

2. *Diffuse Hyperostosis.*—The skull is large and heavy; the skull bones, though unequally affected, are all involved, their surface is uneven, but there are no large exostoses. The diploë is either preserved or replaced by compact bones, and the fossæ, bony cavities, nerve channels, and also their foramina, are constricted. It is very seldom that other bones than those of the skull have been also affected. There are only twelve cases in the literature where the clinical symptoms are recorded. Deformity of the skull has usually been an early sign, and varies according to the bones mainly involved—*e.g.* the frontals may become very prominent; exophthalmos gradually

develops, and neuralgic pains, facial paralysis, blindness, and involvement of other special senses, occur in the course of time owing to compression of the various nerves. The death of the patient has usually been due to cerebral pressure, marasmus, or some intercurrent affection.

*Diagnosis.*—Bony deformity of the skull is the most important sign. The localised form in its early stages might easily be mistaken for syphilis or sarcoma, and in one instance an ossifying myxoma of the nasal septum was recorded as leontiasis ossea. Diffuse hyperostosis has to be distinguished from acromegaly, myxœdema, and osteitis deformans (Sternberg).

*Treatment* is applicable only when prominent bony masses can be removed, or when surgical means may be expected to yield relief from the effects of pressure on peripheral nerves.

**Lepanto.** *See* BALNEOLOGY (*Greece*).

**Lepcophtheirus.**—A variety of fish-lice. *See* SNAKE-BITES AND POISONOUS FISHES (*Poisonous Fish*).

**Leper.** *See* LEPROSY.

**Lepido-.**—In compound words *lepido-* (Gr. λεπίς, a husk or scale) signifies relating to a scale, *e.g. lepidosarcoma* (a fleshy tumour covered with scales), *lepidoid* (scaly), *lepidosis* (ichthyosis), *lepothrix* (a disease of the hairs of the axilla or scrotum which are ensheathed in sebaceous matter), and *leposteophyton* (a thin scale of bone).

**Le Pita.**—Tinea imbricata or Bowditch Island ringworm. *See* SKIN DISEASES OF THE TROPICS (*Caused by Vegetable Parasites*).

**Lepra.** *See* LEPROSY; PSORIASIS (*Synonyms*).

**Leprelcosis.**—The ulceration of leprosy.

**Leprology.**—The study of the pathology, symptoms, and treatment of leprosy (*q.v.*).

## Leprosy.

GEOGRAPHICAL, ETC. . . . . 382
CLINICAL FEATURES—
*Lepra Tuberosa* . . . . 383
*Lepra Maculo-Anæsthetica* . . 385
PATHOLOGY—
*Lepra Tuberosa* . . . . 387
*Lepra Maculo-Anæsthetica* . . 388
ETIOLOGY . . . . . 388
DIAGNOSIS . . . . . 389
TREATMENT . . . . . 389

*See also* AINHUM (*Etiology*); ALOPECIA (*Varieties*); BALNEOLOGY (*Historical*); CONJUNCTIVA, DISEASES OF (*Exanthematous Conjunctivitis*); CORNEA (*Striated Keratitis*); LARYNX, CHRONIC INFECTIVE DISEASES (*Leprosy*); NERVES, MULTIPLE PERIPHERAL NEURITIS (*General Etiology*); NOSE, CHRONIC INFECTIVE DISEASES (*Leprosy*); OSTEO-ARTHROPATHIES (*Mode of Production*); OVARIES, DISEASES OF (*Leprosy*); PALATE (*Diseases, Leprosy*); PHARYNX, CHRONIC INFECTIVE DISEASES (*Leprosy*); POST-MORTEM METHODS (*Bacteriological Investigations, Leprous Nodules*); SCROTUM AND TESTICLE, DISEASES OF (*Leprosy of Testis*); SKIN, BACTERIOLOGY OF THE (*Leprosy, Bacillus Leprœ*); SKIN DISEASES OF THE TROPICS (*Constitutional Infective Diseases with marked Skin Lesions*); SYRINGOMYELIA (*Morvan's Disease and Leprosy*).

THERE is evidence to show that leprosy existed in Egypt and India in times of great antiquity. In an old papyrus, which was transcribed in the fifteenth century B.C., reference is made to the remedies for the cure of a severe disease named "uchetu" which caused pain, deformity, and often death. The Coptic name for leprosy, "ouseht," is considered identical with "uchetu," and is the Coptic word in the Pentateuch for leprosy. As this papyrus professes to be a copy of a much older one, it carries the evidence of the existence of leprosy in Egypt to a very remote antiquity, possibly to 4000 years B.C.

The evidence of the existence of leprosy in India in remote periods is of a more definite character, the vernacular terms for leprosy in India now being practically the same word as that used in the ancient Sanskrit. In the Sanskrit writings allusion is made to leprosy in the fourteenth century B.C., and very definitely in the sixth century B.C.

Probability points to Africa as the original site of the disease, from whence it may have spread to India through Arabia. It existed in China two thousand years ago.

There is evidence to show that the disease found its way to Greece through Asia Minor in the fourth century B.C., and it has been suggested that the spread of leprosy in south-eastern Europe is associated with the hosts led by Xerxes from Asia to Europe.

There is no doubt that at these times leprosy was not accurately separated from other severe cutaneous diseases, and the leprosy of the Bible undoubtedly included other skin affections as well as leprosy. The first good account of the symptoms of the disease was given by Aretæus in the first century of our era, whose account for accuracy and fulness leaves little to be desired.

Lucretius stated that leprosy was confined to the valley of the Nile, and Celsus at the beginning of the Christian era remarks that it was almost unknown in Italy. Pliny the elder relates that the disease was brought from Egypt and Asia Minor in the time of the first emperors, and that it was unknown until the return of Pompey's soldiers from the East—*Ægypti peculiare hoc malum est.*

Leprosy soon spread through Europe. Ac-

cording to Galen, a few cases had already appeared in Germany in the second century. Its diffusion was rapid until in the Middle Ages it was universal, as is evidenced by the number of leper houses and legislative enactments in all the countries of Europe. It extended from England into Ireland and Scotland, where King Robert the Bruce died of the disease.

With the establishment of leper houses came the fear of contact with the leper, and with the isolation of the lepers in the twelfth, thirteenth, and fourteenth centuries the disease began rapidly to diminish. This diminution began in England in the fourteenth century, and the last leper in Great Britain died in the Shetland Islands in the end of the eighteenth century. At the present time leprosy has disappeared from most parts of Europe, but there are still a few cases in certain parts of Spain and Portugal, and on the coasts of Provence; except in Spain, however, it is rapidly disappearing from these parts. There are still cases to be found in Greece, European Turkey, and most of the Mediterranean Islands. There are still many cases in Norway, Iceland, Lapland, and the Russian shores of the Baltic, in most of which places, however, and particularly in Norway, the numbers are diminishing. As the Norwegian law of isolation is to be put in force in Iceland, it is probable that the disease will be stamped out there.

The following numbers, taken from the Reports of the International Leprosy Congress at Berlin in 1897, as reported in the *Annales de dermatologie de syphiligraphie* of that year, refer to the prevalence of the disease in different parts of the world at the present time:—

"P. Kubler, in his review of the geographical distribution of leprosy, remarked that in Asia there is an immense centre (which includes the Indies, South China, and Japan), from which the disease spreads to the north as far as Siberia and Kamtschatka, westward to Persia, Turkestan, and Turkey in Asia, eastwards to the Sunda Islands and to the Moluccas. Australia and Oceania have many centres, mostly of emigrant Chinese. In Africa, where it is endemic on the mainland, he stated that the disease had invaded Madagascar, Mauritius, and Réunion.

"The east side of South America, opposite Africa, is much more severely affected than the west side, with the exception of Columbia. North America is comparatively free; in the United States there are only about 200 lepers. In Europe he laid stress on the importance of the Balkan Peninsula as a centre of the disease.

"In Norway, where isolation is compulsory, the number has decreased from 2833 in 1856 to 321 in 1895. In Iceland there are 158 lepers, in Russia 1200, of whom 800 belong to European Russia. In Germany there have been 34 cases noted, all in the district of Memel, and of whom 19 have died. In Roumania there are noted 208 cases. In Turkey it is not possible to estimate even approximately their number, and at Constantinople alone there are not less than 500 to 600. In Egypt we find more than 3000 cases. In South Africa there are 600 in the Cape, 250 in Basutoland, 150 in the Orange Free State, more than 650 in East Griqualand and Transkei, 105 in the Transvaal, and 200 in Natal; in all, nearly 3000 cases. In the West Indies there is a large number of lepers, several thousands in Japan, and 4000 in the Sunda Islands. The ravages of the disease in the Sandwich Islands, Tahiti, Marquesas, and New Caledonia are well known. It is endemic in Mexico, Central and South America, and more particularly in the Antilles, Guianas, and Brazil, but above all in Columbia, where it is estimated that there are 30,000 in the 4,000,000 of inhabitants."

Dr. Thomson described six cases of leprosy amongst the natives of New Zealand in 1854, whence it was probably introduced from some of the Polynesian Islands.

Clinical Features. — The symptoms of leprosy, taken broadly, may be stated to depend on the localisation of the bacillus, and the localisation depends upon the circumstance that certain tissues afford a suitable soil for its development, whilst other tissues of the body are entirely or comparatively immune. In some individuals the nerves are chiefly affected by the development of the bacillus, the other tissues being spared. This difference in individual cases has led to the clinical distinction of two forms of leprosy, which in their typical development contrast greatly in the outward manifestation of the disease. The two forms of leprosy thus recognised are tubercular leprosy and anæsthetic or nerve leprosy. It is proposed by Hansen and Looft that these two forms might be distinguished as lepra tuberosa and lepra maculo-anæsthetica, the latter especially being a very suitable definition. We shall use the terms tubercular leprosy and nerve leprosy.

Some authors describe a form of mixed leprosy in which the symptoms of tubercular leprosy and of nerve leprosy are coincident; but as sooner or later in all cases of tubercular leprosy the nerve trunks become affected, the cases of so-called "mixed" leprosy may be included amongst cases of tubercular leprosy.

*Symptoms of Lepra Tuberosa.* — There is evidence to show that after the bacillus has established itself in the human organism, it produces toxic symptoms before it has increased to such an extent as to produce local manifestations. These symptoms are often overlooked, and when present are very apt to be attributed to another cause. It is certain, however, that before the development of tubercular leprosy there is in many cases a history of occasional rigor, and pains and stiffness of the limbs,

with lassitude and debility. Vertigo, drowsiness, dyspepsia, febrile attacks associated with much sweating, and occasional epistaxis, are symptoms that have been noted. Dr. Hillis noticed in British Guiana that profuse sweating and vertigo constantly preceded the development of leprous erythema. This erythema is the first distinct local manifestation observable, and can only be explained on the hypothesis that the bacilli on their first development in the cutis exercise a toxic effect on the vessels of the skin, leading to hyperæmia.

The erythema is observed in the form of small patches or in areas of considerable size, sometimes well-defined and sometimes with indistinct borders. The colour is best seen in sudden changes of temperature, and that it is a true erythema is shown by its disappearing under pressure. In the negro the erythematous rash is red or brownish, in the white races of a crimson or reddish mahogany colour which gradually becomes darker. It is frequently seen in the face, and is also found in the extremities. With the development of the rash the patient's general health improves, and he is for a time comparatively well. The skin may gradually resume its natural colour or remain slightly pigmented, but after a shorter or longer period the outbreak is renewed, and after a certain number of these attacks the erythematous patches remain, the colour remaining stationary and the skin thickened. At this stage the erythema no longer disappears under pressure. Finally, with renewed attacks of fever the stage of unmistakable lepromatous infiltration becomes permanent.

*Lepra-tubercles.* — The mode by which the bacillus multiplies by local infection leads to the formation of tubercles or lepromes, which vary in size from a small pea to that of a small nut. Their form is rounded, and they may be isolated or confluent, of a colour varying from violet to dark brown or yellow, flattened in parts which are subject to pressure, and harder on the face and extremities than on the trunk. The swelling is localised in the cutis, the epidermis which covers them being stretched. Although they may occur on every part of the skin except the palms or soles or scalp, they are found more frequently in certain parts of the body than in others. They are usually first observed on the face, on the backs of the hands, and on the wrists (parts exposed to the atmosphere), and afterwards on the extensor surfaces of the limbs. They are rare on the back of the neck, and are not often seen on the back or nates. They are exceptional on the flexor surfaces of the limbs. Hillis, in British Guiana, found that the face, ears, nasal mucous membrane, extremities, nipple, mammary glands, scrotum, prepuce, margins of the anus, vagina, and the armpits are their most frequent sites.

The parts most frequently found affected probably vary in different climates. Hansen and Looft state that in Norway, where people often go barefoot, wading through streams, marshes, and rivers, the backs of the feet and the under parts of the calves are frequently the seat of the first leprous eruption, not so often in the form of nodules as of a dense regular infiltration. The characteristic *facies leonina* is caused by the manner in which these nodules are situated in the face. They develop early and extensively in the skin of the eyebrows, causing them to project over the eyes. The growth in the skin of the forehead above the eyes may be either nodular or take place as a thickened infiltration, but in either case it is deeply furrowed. After the disease has lasted for some time the hairs drop out of the eyebrows. The persistent change of colour, the reddened and usually greasy appearance of the skin, and especially the thickening and change of colour over the eyebrows, are important diagnostic symptoms. As the infiltration progresses the skin of the forehead becomes thicker, the cheeks uneven, the lips protuberant, the skin of the nose thick, and the ears large, rough, and inelastic from the leprous deposit. Particularly in the limbs the leprous infiltration may be in the form of simple diffused thickening with a characteristic dark erythematous colour, without the development of special nodules. In sections from this discoloured skin leprosy bacilli are found. After a time the tubercles remain stationary, but the patient becomes subject to fresh attacks of fever, which are often coincided with their absorption; but whilst the old tubercles absorb or disappear, fresh ones may develop at another part of the body, or during these attacks the tubercles may become red, swollen, and tender. The tubercles may disappear during the course of an acute disease. The leproma may persist a very long time without the epidermis being affected, but it desquamates slightly, and is the seat of excessive sebaceous secretion. The natural elasticity of the skin is lost.

The natural course of the leprosy tubercle is to soften: the epidermis falls off, the tubercle is then discharged, and a scar remains. If not properly treated they may take on necrotic action: the bones may be exposed, and parts become destroyed, particularly the fingers and toes may fall off. The cicatrices which follow ulceration are harder and wider than those which follow absorption.

There is no perspiration in the skin which covers the tubercles, and the sensibility is diminished.

The leprosy bacilli may develop in such a way that, instead of the formation of nodules, large flattened plaques of infiltrated skin may be found, especially in the limbs. This skin may break down in points, which may enlarge and coalesce, and form irregular ulcers with

hard, raised, abrupt borders. The ulceration may extend round the whole limb.

During the development of the disease the lympathic glands in the groins, axillæ, and neck become swollen, sometimes to a considerable extent. The swellings are indolent; in the neck they may produce difficulty in breathing or swallowing.

Sooner or later the nerve-trunks are attacked by the leprous infiltration. Hansen and Looft state that the facial, radial, ulnar, median, and peroneal nerves are always diseased, and they have found that the nerves of the extremities are affected throughout their whole length. "The affection is severe only at certain places, namely, where the nerves run superficially over bones or joints, as the median at the wrist, the ulnar at the elbow, and the peroneal where it crosses the fibula."

The early stage of affection of the nerves is characterised by much pain, and, as the infiltration leads to atrophy of the nerve tubules, to anæsthesia. There may be repeated attacks in the nerves as they become the seat of fresh infection, and the patient may suffer from frequent painful attacks through the course of years. The nodules in nerves are often painful when first developed, but sensation is deadened later. The testicle, liver, and spleen are stated by Hansen and Looft to be always affected in tubercular leprosy.

The duration of the eruptions and the intervals between them vary greatly in different patients. There may be sometimes several in the course of a year, or only one or two in the whole course of the disease. The more frequent the eruptions the more severe is the disease. The mucous membrane of the tongue, cheeks, hard and soft palate, uvula, and tonsils are in time affected, the patient becomes hoarse, and if bronchi catarrh develops, respiration is much interfered with.

Tubercles form in the mucous membrane of the nose, particularly over the part which covers the septum, causing destruction of the nostrils and of the cartilage. Dr. Hillis, who gave much attention to the condition of the throat in this disease, states that in tubercular leprosy the first throat symptoms occur during the febrile attack. The fauces, uvula, and back of the throat become uniformly red and congested, or glazed looking, and the patches seen at the back of the pharynx and roof of the mouth have raised, crescentic edges. Such patches are pathognomonic of leprosy, and, when combined with the thickened condition of the mucous membrane of the nose, explain the epistaxis. After a varying period of some months the interior of the mouth is found to present a dull white, pallid appearance, extending not only to the larynx (see p. 347), but even to the bifurcation of the trachea.

The pharynx becomes the seat of extensive ulceration which may destroy the uvula, and lead to hypertrophy of the submucous connective tissue of the epiglottis and ulceration of the vocal cords. Leprous tubercles of the mucous membrane may begin as white or opaline spots, but are usually of a pale red or livid colour. On the tongue the disease may appear as isolated tubercles, or simply as opaline spots. Eventually this organ becomes thickened, raspberry-looking, and lobulated, the mouth of the patient emitting a fœtid odour.

The leprous affections of the eye have been well described by Dr. C. F. Pollock in a book entitled *Leprosy as a Cause of Blindness*. He states that the disease in the eyeball is largely ciliary in origin, the infiltrations spreading through the cornea. The anterior chamber is invaded from the angle between the cornea and the iris, the iris is attacked from its periphery, and the ciliary body is then involved, the disease passing to the neighbouring portion of the choroid and the ora serrata of the retina, causing blindness.

Both sexes would appear to be equally liable to leprosy, but from their different habits of life men appear to be more exposed to contagion than women, and some statistics show a considerably larger proportion in the male than in the female sex.

In tubercular leprosy there is nearly always nephritis present, and amyloid degeneration of the kidneys, liver, spleen, and intestine are frequent.

Hansen and Looft state that in many examinations of the blood they have never noticed anything remarkable in the form and relation of blood corpuscles.

Recovery is possible, and there are well-authenticated cases of recovery in tubercular leprosy, but the disease is usually fatal. The average duration of life is said to be eight to twelve years, although in some instances the patient may be carried off quickly by acute leprosy, or may live twenty or more years. The cause of death is usually some complication, lepers being particularly liable to tuberculosis.

*Symptoms of Lepra Maculo-Anæsthetica.*—In the cases of leprosy in which the manifestations of the disease are chiefly confined to the nerves, the affection runs a milder course. Either the bacillary poison is less active in this form, or there is a stronger constitutional resistance on the part of the patient; but even in nerve leprosy the first infection of the system by the toxin frequently produces appreciable effects, rigors, pallor, and depression being observed. Frequently, however, these symptoms are either not present or are so slight that they are overlooked.

It will be convenient to describe separately the chief characteristic symptoms. These are spots, bullæ, anæsthesia, motor paralysis, and absorption of tissue with mutilation. The spots

usually appear early in the disease. At first they are simply erythematous and become gradually pigmented. Their usual size is from that of a sixpence to that of the palm of the hand or more. In course of time the reddish colour changes to a yellowish shade, becoming eventually dark. At first the redness disappears under pressure. They are either flattened or may be slightly elevated, free from sensation, or the seat of slight itching or burning. They may remain stationary in size, or, in increasing, they coalesce, forming large irregular surfaces in which are found patches of unaffected skin. In course of time the pigment becomes absorbed, leaving the skin pale or livid, the margin retaining its colour longest. The borders of the spots are nearly always raised, and small vesicles may be observed in them. The colour of the spots is influenced by race. Hillis found that in negroes they are almost invariably yellow, while Norwegian writers call special attention to the erythematous nature of the spots. Whilst hyperæsthetic in the peripheral hyperchromic margin, the centre (where the terminal branches of the nerves have been destroyed) becomes anæsthetic. The corresponding lymphatic glands are always swollen.

In distribution the spots appear to follow that of the nodules in tubercular leprosy, except that they are frequently found on the back and in the intercostal spaces. They are not found on the scalp or palms or soles; and although when there is much eruption present they are apparently symmetrical, this symmetry is by no means absolute—it is often entirely absent.

Coincident with the development of the spots symptoms of neuritis present themselves. Before the anæsthesia is developed the patches are usually hyperæsthetic, the ulnar and peroneal nerve-trunks are found to be thickened and sensitive, and the peripheral finer branches may be detected by the finger. Hansen and Looft state that in one case they were able to feel the cutaneous nerve branches in a patch growing daily more and more thickened. The large nerve-trunks become thicker near joints where the nerves pass superficially over a bone.

The affection of the nerves causes neuralgia and pain in the regions which they serve, the anæsthesia not being confined to the patches, and progressing gradually from the periphery to the centre till the whole limb and often parts of the trunk become anæsthetic. There is usually more or less anæsthesia on the face.

The appearance of bullæ is very characteristic of nerve leprosy, and it is assumed they are a direct result of the neuritis. These bullæ may appear suddenly, and within months or years after the premonitory symptoms. They vary in size from a hazel-nut to a hen's egg, are somewhat transparent, and are filled with a sticky yellowish fluid. They leave slightly reddened ulcerated surfaces, the secretion from which gives rise to a succession of brownish crusts. They may heal in a few days without a scar, but months usually elapse before they close. The scars which follow the healing of these ulcers are white, slightly depressed, and often less sensitive than the surrounding skin; they are sometimes surrounded by a light brown border; they are generally free from hairs, and where hairs are found they are fine and colourless.

The formation of bullæ may go on for years. Danielssen and Boeck have seen it last for five years, the patient being free from them for very short intervals. They are usually solitary, but sometimes several come at once.

Danielssen and Boeck have only once seen leprous bullæ on the face. They occur very frequently on the palms and soles, but they may come on any part of the body except the scalp. Leloir has seen them three times in mucous membrane.

As a rule the early bullæ are small, numerous, and hyperæsthetic or even normal in sensation, whilst the later ones are large, solitary, and may be anæsthetic. With further development of the neuritis excessive hyperæsthesia, limited to certain parts or extending over the extremities or a large part of the face, may develop. It often occurs at first in the extensor surfaces. Danielssen and Boeck state that the slightest contact produces a "sensation like that of an electric shock." Movement causes violent pains from which the patient only obtains relief by remaining in bed. He loses hope and appetite, emaciates, and, perspiring little, his skin is disagreeably dry. The hyperæsthesia, which may last a long time, is succeeded by anæsthesia, occurring usually at first in the parts supplied by the ulnar and peroneal nerves. The skin becomes parchment-like and inelastic at places, the secretions of sweat and sebum being entirely arrested. The anæsthesia in the feet leads to uncertainty in gait.

As a consequence of the neuritis there is muscular wasting, and frequently the first symptom which a patient recognises is loss of power. Hillis mentions that amongst the negroes in British Guiana the field labourer often has his attention first directed to his condition by the difficulty he finds in holding his cutlass. This muscular shrinking often begins in the hands, the shrinking of the muscle over the metacarpal bone between the forefinger and the thumb being characteristic; then the muscles of the hand, forearm, and upper arm atrophy. A similar atrophy occurs in the corresponding muscles of the legs. The anæsthesia is so complete that the flesh may be burned or amputated without pain being caused, but a sense of contact is experienced when the bone is sawn or scraped.

The changes in the muscles cause the fingers to be permanently flexed, leading to the charac-

teristic clawing. After some time the fingers cannot be straightened. Similar changes occur in the toes; perforating ulcers form on the sole, particularly in persons who go barefoot. After the disease has lasted some time the bones of the fingers and toes may disappear by caries and by interstitial absorption, the interstitial absorption of bone without inflammation often being a special characteristic of nerve leprosy.

The neuritis of the nerves of the face produces striking effects. The paralysis of the orbicularis palpebrarum muscle leads to incapacity to close the eyelids, and the lower lid falls downwards, particularly at the inner corner. Tears from the lachrymal duct flow over the cheek. From the injury sustained by the cornea by remaining uncovered, particularly during sleep, small vesicles form, leading to opacities. Complete ectropion is established; the ulceration of the cornea may lead to rupture and prolapse of the iris and atrophy of the eyeball. Paralysis of the orbicularis oris leads to dropping of the under lip, difficulty in closing the mouth, and dribbling of saliva. In later stages, smell and taste are diminished or lost. Dyspeptic symptoms, heartburn, pyrosis, acidity, constipation, drying of the mouth, and great thirst occur. The patient complains of sensation of cold, and in the later stages of the disease Hillis states the temperature is several degrees below normal.

The kidneys are liable to amyloid degeneration, and death is frequently caused by diarrhœa accompanied by cramps.

If the disease occurs before puberty, menstruation does not occur. If it begins in adult life it is usually irregular, and sometimes ceases.

The progress of nerve leprosy may become arrested. The spots nearly always disappear when the disease has lasted long, and sensibility may be re-established. The general health may improve, but the anæsthesia of the skin and the atrophic condition of the muscles remain, although even these conditions may greatly improve in young persons. In some cases the disease may be considered completely cured.

The mean duration of the disease is stated by Bidencap to be eighteen or nineteen years, and many of these persons may attain a relatively great age. In tropical countries cases of nerve leprosy outnumber those of tubercular leprosy, whilst in cool damp climates the reverse is the case.

Pathology of Lepra Tuberosa.—When a section is made from a leprous nodule, the substance of the cutis is found to be replaced by an accumulation of cells of various sizes packed together in enormous numbers. The cells vary in size, many of them being not larger than a white blood corpuscle, while some are considerably larger. These cells contain the leprosy bacilli, which were discovered by Hansen in 1871.[1] The smallest of these cells contain few bacilli, but the larger cells contain great numbers, often arranged in groups. The majority of the cells have the appearance of white blood corpuscles, and we have found in small capillary blood-vessels of a leprous larynx white corpuscles containing bacilli. But there is evidence to show that the connective tissue cells also contain bacilli.

The leprous nodule is well supplied with blood-vessels; and as it grows, and the number of cells containing bacilli increase, the connective tissue is absorbed. In the skin, for a long time, a thin layer of connective tissue immediately under the *rete mucosum* remains entire.

Bacilli are very rarely found in the epidermis, but they have been occasionally observed; and the author of this article has described and figured them in cells in the *rete mucosum* (possibly cells which have migrated from the cutis), and Dr. Unna has shown clearly that many lepra bacilli may be found in the hair follicles between the sheath and the hair-shaft.

The leprous cells in the spleen, liver, and testicle, nerves, lymphatic glands, and eyes, and in the pharynx and larynx, also contain bacilli similar to those in the nodules in the skin.

Hansen and Looft have never found bacilli in the liver-cells, but have found in the hepatic vessels white corpuscles containing bacilli.

In old nodules it is found that the leprosy bacilli have broken down into granules. Although the bacilli are mostly contained in cells, collections of them are also found in lymph spaces. They develop in the protoplasm of the cells, the nucleus being long spared, vacuolation of the protoplasm eventually resulting. The bacilli are rarely found in the blood, but that they may be found there is shown by the fact that Hansen and Looft have described and figured them lying between red corpuscles in the vessels.

The order of development of the leprous nodule would seem to be that white corpuscles containing bacilli are deposited in the tissues; the toxin generated by the bacilli acting on the blood-vessels leads to emigration of leucocytes, which in their turn become infected by the previously infected corpuscles with which they come in contact—this process going on slowly and persistently until we have the large accumulation of cells contained in the leprous nodule. That this development requires special conditions within the body is shown by the fact that in many tissues it does not take place.

[1] *Medico-Chirurgical Transactions*, vol. lxvi. p. 315: "The first notice of the bacillus of leprosy is contained in a report made to the Medical Society of Christiania in 1874 by Hansen. In his paper on the subject in the *Quarterly Journal of Microscopical Science*, New Series, vol. xx., 1880, this report is referred to as containing the statement that he had 'often, indeed generally found, when seeking for them in the leprous tubercles, small rod-shaped bodies in the cells of the swelling.'"

The leprosy bacillus closely resembles the tubercle bacillus in size and staining properties. Yet in form they are not absolutely identical, successful photographs showing that the leprosy bacillus is slightly club-shaped. It also shows a tendency to develop in groups, even within one cell, a quality which is not shown by the bacillus of tubercle.

Certain distinctions are also made regarding the capacity for staining and for retention of the stain, but these differences can hardly be considered as well established.

It has been maintained that an essential difference between the leprous and tubercle degenerations is to be found in the presence and absence of so-called "giant" cells, these appearances being held to be absent in leprosy; but this is not quite exact. Although much more frequent in tubercle than in leprosy, they are also found in the latter disease. Until, however, a definite understanding is come to as to what a "giant" cell actually is, and how it is formed, the point is not one on which great importance can be laid.

Pathology of Lepra Maculo-Anæsthetica.—For some time it was considered that bacilli were not found in nerve leprosy. Dr. Arning was the first to show the presence of bacilli in a portion of an excised nerve, and since then other observers have confirmed his statement. Darier has recently given an account of the changes found in the erythemato-pigmentary patches of nerve leprosy. The first change is an infiltration of cells in the sheaths round the blood-vessels. In some parts these infiltrated sheaths become confluent and form layers of cells. The majority of these cells are small connective tissue cells, with which are mixed in varying proportions white corpuscles, plasma cells, a few mast cells, and, in rare cases, giant cells. In eight out of nine cases which he examined he detected the presence of bacilli. Whether the spots were old or recent, erythematous or purely pigmented, there were sometimes very few present, at other times they were very numerous.

The first pathological changes, therefore, are identical in kind though differing in degree from those found in nodular leprosy. Similar changes are described by Hansen and Looft, who, in an old anæsthetic patch, found only very slight infiltration along the vessels. "The cells were mostly spindle-shaped, only a few were round or epithelial. In most of the sections no bacilli were found; in some, one or two distinct bacilli and some granules which took the same stain were present."

Until recently it was considered that leprous affections of the spinal cord did not exist. This opinion has, however, been recently modified. Looft has found in two cases of nerve leprosy degeneration of the posterior columns, atrophy of the posterior roots and fibres, degeneration of the spinal ganglia, with disappearance of the medullary fibres and changes in the nerve-cells.

In these two cases the affection appeared to be primary in the ganglia and secondary in the cord. Leprosy bacilli were not found in these cases; but Chariotti found them once in the cord, and Suderkowitsch in a spinal ganglion. Babes found bacilli nine times in the spinal cord, three times in the anterior horns, and often in the spinal ganglia. Generally they are found in the protoplasm of the nerve-cells, which are sometimes vacuolated and altered, and sometimes normal. It would appear that sometimes the centres and sometimes the peripheral nerves are primarily invaded.

Whilst the pathological changes in the skin and nerves in this form of leprosy are caused by the direct infection of the bacillus lepræ, the tropho-neurotic changes in muscles, bones, and joints appear to be secondary, as the bacillus has not been found in these tissues. The atrophy of the muscles regarded by Neisser as a specific leprous process is regarded by G. and E. Hoggan as secondary, and due to neuritis, an opinion supported by Hansen and Looft.

The following views regarding the relations of tubercular and nerve leprosy were expressed at the Berlin Conference:—

Neisser considered that the difference between tubercular and nerve leprosy is not simply one of quantity of the bacilli, but in the nature of the morbid process which they produce. In the one case the change leads to proliferation, whilst in the other it is an atrophic one. Hansen considered that climate has an influence on the forms; Blaschko, that it is only a difference in the quantity of the bacilli. Dehu and Gerlach had proved that the bacilli can affect the nerves, beginning at the peripheral cutaneous extremity. Arning considered the difference fundamental: in the tubercular form the nerves may be stuffed with bacilli, with yet few nervous changes; whilst in nerve leprosy there may be few bacilli in the nerves and in the skin, and yet anæsthesia, amyotrophic sweat troubles, and neuralgia are present.

Etiology.—The etiology of leprosy is much simplified since the discovery of the bacillus. As tuberculosis is dependent on the development in the tissues of the tubercle bacillus, so leprosy in all its forms is dependent upon the changes in the tissues which are selected by the leprosy bacillus. Although the proof in the case of the leprosy bacillus is not logically complete, inasmuch as the disease has not been communicated to a healthy individual by the cultivated bacillus, yet the universal presence of the organism in this disease, and its absence in persons free from leprosy, warrants the assumption that it is the true cause of the malady.

The grounds on which it is held that leprosy is a bacillary disease are that the bacillus is always present in cases of the malady, that its

localisation is associated with those changes that are symptomatic of leprosy, and that the cells of the organism undergo changes in proportion to the number of bacilli which they harbour. The bacillus itself has certain specific characters by which it can be distinguished from all other known bacilli. The inference is strengthened by the fact that the progressive changes in the tissues of a leper, and the manner in which the disease is propagated, harmonise with what is known of other bacillary diseases, and confirm this view.

The bacillus has not yet developed in inoculated animals, and its cultivation in artificial media is not yet accepted. Campana, however, who has devoted much attention to this matter, considers that, if the bacilli which it is attempted to cultivate are taken from an early stage of the eruption, appearances which warrant the assumption that growth has taken place in the media may be obtained. After the first stage of the disease the bacilli are dead, and are incapable of propagation.

The history of leprosy shows that the disease is conveyed from man to man. Its long period of incubation and slow development are obstacles to the discovery of the means by which infection is propagated. Clothes, shoes, bandages, etc., are suspected of being the media by which leprosy is spread. In Java, India, and Tonquin, where the people walk barefoot, the disease begins in the feet in one-half of the cases, the presumption being that the bacilli are contained in the soil, in which they have been deposited from leprous discharges.

Sticker inferred, from an examination of four hundred lepers, that the initial lesion is usually in the nasal mucous membrane, in the cartilaginous portion, beginning as a simple ulcer, which sometimes precedes for several years the nerve symptoms and nodules. This ulcer was only missed in 13 cases in 153, and in 9 out of the 13 there were an abundance of bacilli in the nose; but Arning's successful inoculation at Honolulu shows that the bacilli may enter by other parts. The chief means by which the bacilli are spread abroad from the patient are through the mouth and nose in coughing and sneezing. Schaeffer calculated that a leper has only to speak aloud for two minutes to eject for a distance of a metre and a half and more 40,000 to 185,000 bacilli.

Saliva, the mammary glands, the sperma, the female genital passages, often contain bacilli in large quantities, which are thus conveyed outside. It has been stated that in Honolulu the common tobacco-pipe, which is passed from mouth to mouth, conveys the disease.

That leprosy may be conveyed by direct contagion from the leper to a healthy man is proved by the record of several cases in which no fallacy was possible; that, for example, recorded by Dr. Hawtrey Benson of Dublin. In this case a leper who acquired the disease in a tropical country shared the same bed with his brother, who had never been out of Ireland, and who afterwards became a leper.

Diagnosis.—In a developed case of tubercular leprosy the diagnosis presents no difficulty to any one who has ever seen a case of the disease, or who has even seen good pictures of it; but in the early stage the diagnosis might be for a time less easy. There might for a time be a difficulty in determining whether the disease was leprosy or syphilis, but this difficulty should not last long. The development of the eruption differs greatly. Whilst in the early stages of a syphilide the eruption is distributed over the trunk and less markedly on the limbs, and gradually fades within the usual time, in the early stage of tubercular leprosy some part of the body is usually selected, and, after a time, the characteristic brown colour removes the doubt. In suspected leprosy the eyebrows should be especially examined, as the hairs are early lost, and the skin above the eyebrows soon thickens, giving rise to the well-known expression of the leper.

The diagnosis of nerve leprosy in the early stage is often by no means easy, and errors are in these cases not uncommon. The writer has known the patches of early nerve leprosy mistaken for body ringworm and for lupus. The anæsthesia, however, which can always be detected if carefully looked for, is distinctive. The fingers, toes, wrists, and dorsum of the feet should be carefully examined for evidence of anæsthesia, and in some parts of the spots themselves sensation will be found to be absent or perverted. Hansen and Looft suggest that in examining for anæsthesia calipers or very slight stroking should be used, as deeper pressure can be at once detected. They also call attention to the fact that in nerve leprosy the lymphatic glands will be found to be swollen. Nerve leprosy may also be mistaken for syringomyelia, and a case has been published in France in which this disease was diagnosed, but in which its true nature was shown by leprosy bacilli being demonstrated in an excised portion of the ulnar nerve. There are other examples on record of the difficulty of diagnosing between these two diseases. It should be borne in mind that in syringomyelia, though there is loss of painful and thermal sensibility, tactile sensibility remains, and that in nerve leprosy there ought to be found the remains of the characteristic spots.

Treatment.—The treatment of leprosy should consist of measures which are likely to enable the organism to resist the effects of the toxin, and to repair the tissues which have been damaged by the direct and indirect effects of the bacilli. Just as in recent years the effect of constant fresh air, hygiene, and good diet have had unlooked-for effects in enabling

patients to throw off the results of tuberculosis, so the same measures should be used to support the patient while undergoing the effects of the leprous poison. Lepers should have frequent baths, should be well clothed to promote the cutaneous circulation, and should be made to spend a considerable time in the open air. This should be combined with the systematic administration of an abundance of highly nutritious food.

Of the many drugs that have been tried in leprosy it is certain that many of them are of no value.

Dr. Dougall's treatment by gurjun oil was tried in Norway, but without the good results described by him. On the other hand, Dr. Hillis considered that gurjun oil greatly relieved the symptoms of the disease. Dr. Dougall recommended the oil to be given internally, 15 grains night and morning, and made into an emulsion with lime water; patients rubbing the whole body for two hours forenoon and afternoon with a mixture of 1 part of oil to 3 parts of the lime water. Every morning they rubbed themselves with dry earth and took a bath to remove the oil. Dr. Dougall states that in India this yielded good results.

Chaulmoogra oil has also been recommended in India. It is given internally in half-drachm doses—best in capsules—and applied externally in a mixture of 1 part to 16 parts of olive oil. This is rubbed into the skin, and a bath taken some hours afterwards. A trial which was made in Norway did not give encouraging results.

Carbolic acid, creasote, phosphorus, arsenic, and ichthyol were found by Dr. Danielssen in Norway to be inefficacious. Mercury was found by Dr. Danielssen to make the patient worse rather than better; but recently Dr. Radcliffe Crocker believes that he has found benefit from subcutaneous injections with the perchloride.

Iodide of potassium has a peculiar effect, producing in lepers new eruptions of nodules or patches. Dr. Danielssen, therefore, used it as a test of the cure of a patient. If after iodide no new eruption appeared, the cure was considered complete.

Dr. Unna has recommended an application of a plaster consisting of chrysarobin, salicylic acid, and creasote, which certainly produced favourable results for a time. Dr. Unna has also recommended a 10 per cent ointment of pyrogallic acid and lanoline, and the writer can testify to the good results of this treatment in early nerve leprosy. Amelioration is said to have been effected by the administration of salol. Danielssen considered salicylate of soda as very useful in the treatment of leprosy; he found that under its use the fever was lessened, the period of eruption shortened, and that newly-formed nodules disappeared. It did not affect old nodules.

There is no uniformity in the testimony as to the action of drugs in the treatment of leprosy, and in the cured cases it is probable that the cure takes place spontaneously. There is no doubt, however, that drugs are very useful in alleviating symptoms.

Hansen and Looft call attention to the benefit derived from surgical treatment, section of the cornea being practised in the case of tubers growing into it, the cicatrix of the section barring the way to the further penetration of the growth. Iridectomy is often performed when the pupil has been obliterated by adhesions of the iris or by exudation. Tracheotomy is necessary when the larynx is occluded by leprous growths or by cicatrices. Necrotomies should always be performed when there is necrosis of the bones of the hands and feet. The wounds heal well in the anæsthetic parts, and the patients are spared from long-standing suppuration by the removal of the necrosed bones.

If leprosy is little amenable to treatment, preventive measures have produced most favourable results. If every leper is looked upon as a source of possible infection, and sufficient means are taken to prevent healthy persons being contaminated by discharges from his body, it is beyond doubt that the disease will diminish, and that it is capable, under favourable circumstances, of being exterminated. Unclean habits and overcrowding favour the development of leprosy where lepers exist, whereas personal cleanliness and a separate room, or, at least, a separate bed, leads to diminution of the number of cases. In the clean surroundings in North America the Norwegian lepers have ceased to propagate the disease; and in Norway itself, since isolation has been instituted, the number of lepers has diminished from 2833 in 1856 to 321 in 1895. If the same precautions that are now considered essential in the case of a tubercular and syphilitic person are practised with lepers, new cases would soon cease to occur. In regard to isolation, the International Congress on Leprosy at Berlin, 1897, accepted the following resolution:—"In all countries in which there are centres of leprosy, or in which the disease extends, isolation is the best means of preventing its propagation.

"Compulsory notification, inspection, and isolation, as they are practised in Norway, are recommended to all nations in which there are autonomous municipalities, and in which there are a sufficient number of medical men. It should be left to the administrative authorities to determine, on the advice of the sanitary committees, measures in detail, having regard to the social conditions of each country."

**Leptismus.**—Emaciation (Gr. λεπτύνω, to grow lean, to make thin).

**Lepto-.**—In compound words *lepto-* (Gr. λεπτός, slender or thin) means thin or slender.

**Leptocephaly.** — A malformation of the head, which has a narrow shape due to synostosis of the frontal and sphenoid bones.

**Leptochrous.**—Having a thin or delicate skin.

**Leptomeningitis.**—Inflammation of the pia and arachnoid mater ("piarachnoid") of the brain and spinal cord. *See* Meninges of the Cerebrum (*Inflammation of the Pia Arachnoid*).

**Leptomicrognathus.**—A malformation characterised by smallness of the lower jaw (micrognathus) due to simple shortness of the same (*Taruffi*).

**Leptomitus Lacteus.**—A fungus, allied to the salmon fungus, which blocks up streams, and is due to distillery effluents.

**Leptophonia.**—Weakness or thinness of the voice.

**Leptoprosopous.** — Having a long, thin face (Gr. λεπτός, thin, and πρόσωπον, a face); dolichofacial.

**Leptorrhine.**—Having a thin or slender nose; with a nasal index of less than 48°.

**Leptothrix.**—A micro-organism consisting of thin, unbranched, hair-like cells, straight or spinal. There are several varieties: leptothrix buccalis, l. buccalis maxima, l. epidermidis, l. lacrimalis, l. racemosa, l. vaginalis, and leptothrix of puerperal fever. *See* Pharynx, Examination of (*Pharyngomycosis*); Septicæmia; Teeth (*Causes of Dental Caries*); Teeth (*Oral Sepsis*); Tonsils, Diseases of (*General Considerations*).

**Leptus Autumnalis.**—The harvest bug. *See* Scabies (*Other Acari*); Stinging Insects (*Harvest Bug*).

**Le Ragle.**—"A name given by d'Escayrac de Lauture in 1885 to hallucinations, mostly visual . . . which not unfrequently happen to travellers in the desert, especially to such as are in a debilitated state from previous illness, or who have suffered from great fatigue, want of food, anxiety, terror, etc." (*Hack Tuke*).

**Lerema.**—Loquacity or silly childish talk, such as is met with in senile dementia.

**Leresis.** *See* Lerema.

**Les Avants.** *See* Therapeutics, Health Resorts (*Switzerland*).

**Leschenoma.**—Loquacity, garrulity, or the silly chattering of mental disease and hysteria.

**Lesion.**—An injury or damage to any part, or a morbid change in the structure or in the performance of the functions of an organ. There are various kinds of lesions: thus a *molar* lesion is a gross one; a *molecular* lesion is one not recognisable by the microscope or by chemistry; a *histological* lesion is one in which the changes are sufficiently marked as to be recognisable by the microscope; a *discharging* lesion is a morbid change in the brain causing a sudden discharge of nervous motor impulses; and a *lesion of continuity* is a solution or break of continuity. The *initial* lesion of syphilis is the chancre.

**Lesser's Triangle.**—The space in the neck bounded above by the hypoglossal nerve and at the sides by the bellies of the digastric muscle.

**Leste.** — The east wind of the Canary Islands and Madeira. *See* Air, Examination of (*Moisture*).

**Lethal.** — Causing death, fatal, mortal, *e.g.* a lethal wound. *See* Medicine, Forensic (*Wounds*).

**Lethargy.**—Drowsiness or torpor which the patient cannot overcome by will. *See* Adolescent Insanity; Insanity, its Nature and Symptoms (*Stupor*); Sleeping Sickness or Negro Lethargy; Unconsciousness.

**Letheomania.**—A morbid longing for narcotic drugs or anæsthetics (Gr. λήθη, forgetfulness).

**Letheon, Compound.** — Sulphuric ether.

**Lethin.**—A proprietary preparation containing chloroform, camphor, acetic acid, etc.

**Lethrinus Mambo.**—A poisonous fish of the South Pacific. *See* Snake-Bites and Poisonous Fishes (*Poisonous Fish*).

**Lettuce.** *See* Invalid Feeding (*Vegetables, Boiled Lettuce*); Lactuca.

**Leucæmia.** *See* Leukæmia; Leucocythæmia.

**Leucæthiopia.**—Albinism in the black race; a negro albino.

**Leucine.**—Amido-caproic acid ($C_5H_{10}NH_2CO{\cdot}OH$), found normally in various organs (liver, spleen, salivary glands, thymus, thyroid), and under abnormal conditions in the urine. *See* Bronchi, Bronchitis (*Fœtid Bronchitis, Dittrich's Plugs*); Expectoration (Microscopical Examination of Sputa, *Crystals*); Physiology, Food and Digestion (*Pancreatic and Intestinal Secretions*); Trades, Dangerous (*Phosphorus Poisoning, Urine*); Urine, Pathological Changes in (*Abnormal Nitrogenous Constituents, Sediments*).

**Leucinuria.**—The presence of leucine in the urine.

**Leucoblasts.**—Young leucocytes. *See* PHYSIOLOGY, BLOOD (*Sources of the Blood Constituents*).

**Leucocidine.**—A poison which can be separated from pus (*Denys* and *Van der Velde*). *See* SUPPURATION (*Circumscribed Abscess*).

**Leucocytes.** *See* BLOOD (*White Corpuscles, Leucocytes, Physiological and Pathological Variations*); IMMUNITY (*Phagocytosis*); INFLAMMATION (*Escape of Blood Corpuscles*); LEUCOCYTOSIS; PHYSIOLOGY, BLOOD (*Characters and Constituents*); URIC ACID (*Relation of Leucocytosis to Uric Acid*); URINE, PATHOLOGICAL CHANGES IN (*Sediments, Leucocyte Casts*).

## Leucocythæmia.


DEFINITION . . . . . 392
VARIETIES . . . . . 392
CHANGES IN THE BLOOD—
*Myelæmia* . . . . . 392
*Lymphæmia* . . . . . 393
*Mixed Forms* . . . . . 394
*Effect of Intercurrent Affections* . . 394
SYMPTOMS . . . . . 394
COURSE AND PROGNOSIS . . . . 395
MORBID ANATOMY . . . . . 395
CAUSATION AND PATHOLOGY . . . 396
DIAGNOSIS . . . . . 398
TREATMENT . . . . . 399


*See also* ANÆMIA; AUDITORY NERVE AND LABYRINTH (*Hæmorrhage into the Labyrinth*); CHLOROSIS (*Diagnosis*); LEUCOCYTOSIS; OXYGEN (*Uses*); PURPURA (*Cachectic*); RETINA AND OPTIC NERVE (*Retinal Hæmorrhages, Causes*); STATUS LYMPHATICUS; THYMUS GLAND; X-RAYS.

DEFINITION.—Leucocythæmia or leukæmia is a disease of the blood and blood-forming organs, in which there is a great increase in the number of leucocytes or white corpuscles present in the blood and an alteration in their characters and relative proportions. The bone-marrow, lymphatic apparatus, and spleen, or any one or more of them may be converted into nurseries for the varieties of leucocytes present in the blood, and in addition collections of these corpuscles may be found in various other organs. These changes may give rise to enlargements of organs. Anæmia, more or less severe, always accompanies the condition, and after a short or long course it almost always terminates fatally.

VARIETIES.—The nomenclature of the varieties of the disease has undergone several changes, and is likely to pass through more in the future. The first cases observed by Hughes Bennett and Virchow, in 1845, were associated with great splenic enlargement; and when Virchow later met with a case in which the lymphatic glands were mainly affected, he distinguished a "splenic" and a "lymphatic" form, according to the organs from which he believed the increased numbers of leucocytes to be derived. Later still, Neumann pointed out that the bone-marrow was also affected in many cases, and a "medullary" form was distinguished. Further research showed that most cases were of a mixed kind, and the terms in use till quite recently were "spleno-medullary" for those cases where the spleen was enlarged and the marrow hypertrophied, and "lymphatic" for those in which enlargement of lymphatic glands was the principal feature. Ehrlich's studies on the varieties of leucocytes, as determined by their staining reactions, gave a fresh impetus to the investigation of the blood in these conditions, and the tendency at the present day is to classify cases entirely according to the varieties of leucocytes present in excess in the blood, without reference to the enlargements of organs. The names of "myelæmia" or "myelocythæmia" and of "lymphæmia" or "lymphocythæmia" are often used to express respectively the varieties in which the cells in the blood resemble those normally found in the bone-marrow, of which the myelocytes are specially characteristic, and those found in the lymph-glands, the lymphocytes. If, however, we accept the view of Löwit, recently put forward, that both conditions are due to a blood-parasite, we are desired by him to use for the former condition the term "polymorphocytic leukæmia," for the latter "homoiocytic leukæmia," cumbrous terms which are not likely to be accepted. I shall use in this article "myelæmia" and "lymphæmia" for the two varieties, as they are short terms and sufficiently accurate, but do not commit us to the acceptance of any theory as to the causation of the disease.

CHANGES IN THE BLOOD.—When the ear is pricked the blood very often shows no special naked-eye change; it may look opaque, however, or may be pale if there is great anæmia, but it does not look pink unless the increase in leucocytes is very great indeed. When a fresh specimen is examined the leucocytes are seen to be greatly increased in number; but it is, of course, impossible to distinguish it from a leucocytosis until counts have been made and stained films examined, unless the specimen is examined on a warm stage, when the great majority of cells in leucocytosis will be found to be amœboid, while in leucocythæmia of either variety most cells are non-amœboid.

*Myelæmia.*—There is a greater actual increase in the number of leucocytes than in any other condition. Cases have often been recorded with 1,000,000 per cubic millimetre, and the average is about 400,000. The actual number varies greatly, however, from day to day, and even from hour to hour, and in exceptional

cases where remissions occur, the number may fall to normal, so that the condition would not be suspected unless films were carefully examined, when a certain proportion of the abnormal corpuscles, especially the myelocytes, will generally be found to be present.

When films stained with a mixture of basic and acid stains, such as Ehrlich's triacid, or eosin and methylene blue, are examined, it will be found that the special character of the blood is the presence of large numbers of myelocytes, which form usually about 30 per cent of the total number of leucocytes. These cells may occur in other conditions, usually towards the close either of a long-continued leucocytosis, as in cancer, or of a short very extreme leucocytosis, as in pneumonia; but they never appear in anything like the same number as in this disease. At first sight their faint neutrophile granules and pale nuclei seem to fill up the whole film, as they are mostly large cells, and are apt to lie together in large groups. Normally they do not appear in the blood at all, but are found only in the bone-marrow, and are believed to be the precursors of the polymorpho-nuclear neutrophile cells, which form the majority of the normal blood leucocytes. These are also absolutely increased in myelæmia, though they are relatively diminished; and in this disease more than in any other, and more even than in the normal bone-marrow, transition forms between the myelocyte and its descendant are to be seen, and forms also which, by the pale staining of their granules, the homogeneous staining of their nucleus, or their small size, give the impression of being degenerated forms. The eosinophile cells are also much increased absolutely, and usually they show a slight relative increase as well. They may consist of several different forms—the form with polymorphous nucleus which is usually found in the blood, a form not larger than a small lymphocyte, and the form known as eosinophile myelocytes; large cells with a pale rounded nucleus and just like a myelocyte, except that the granules are eosinophile instead of neutrophile. These cells are the most numerous form of the three, are normally found only in the marrow, and except in this condition only appear in small numbers in some cases of pernicious anæmia.

The lymphocytes are usually increased absolutely, but relatively are always greatly diminished. Basophile cells are always present in varying numbers, and are always both absolutely and relatively increased. Ehrlich regards this increase as specially characteristic of myelæmia. They may be either finely or coarsely granular, and have either a round or polymorphous nucleus. They are of importance in relation to Türck's objections to Löwit's theory.

The red corpuscles in the early stages of the disease are not diminished; but later there is generally some diminution in their number, with a corresponding diminution in hæmoglobin, and, if the anæmia is marked, with corresponding changes in the shape and size of the corpuscles. The average number in a well-marked case is about 3,000,000. Quite independent of any anæmia, however, is the number of nucleated red corpuscles. These are always to be found, and in far greater numbers than in any other disease in adult life. They are generally normoblasts, but very occasionally megaloblasts may appear. The number of red corpuscles bears no constant relation to that of the leucocytes, though usually they become fewer as the leucocytes increase in number. Blood-plates are usually increased in number.

Each of these different factors is present in every case of myelæmia, but their relation to one another is extremely variable. Sometimes the myelocytes are overwhelmingly numerous, and this is the most common of the varieties, sometimes the eosinophile cells; in one case the nucleated red corpuscles predominate, and in another the basophile cells. Each case presents a different and individual blood-picture, but the general effect is that of an inundation of the blood with marrow-cells. One can indeed produce a very fair imitation of the condition by mixing normal bone-marrow with blood, and making films of the mixture.

*Lymphæmia.*—The number of white cells is not usually so great as in the other variety. It is often under 100,000 per cubic millimetre, and the average of my cases has been about 200,000. Cabot records one case with 1,480,000, but this is quite exceptional.

The striking thing about the films is the enormous increase of lymphocytes, both relatively and absolutely. They usually form more than 90 per cent of all the leucocytes, and either large or small forms may predominate. The remaining cells are always polymorpho-nuclear neutrophiles. Myelocytes do not appear, and eosinophiles are seldom seen. So much is common to both forms of lymphæmia, for this variety is subdivided clinically into acute and chronic forms. In the latter the red cells are about the same in number as in myelæmia, but unless there is marked anæmia, nucleated red corpuscles are not found, and the film contains nothing but ordinary red corpuscles, lymphocytes, and an occasional polymorpho-nuclear cell. If a chronic case becomes acute the anæmia advances, and the leucocytes are practically all lymphocytes. I saw one case where they were over 99 per cent. In cases acute from the first there is usually marked and progressive anæmia, with nucleated red corpuscles proportional in number to the anæmia, and in children and young people often becoming very numerous. The lymphocytes are more usually of the large variety. Blood-plates are diminished in both forms.

In this form of the disease, then, the blood-

picture is that of an inundation of the blood with the elements usually found in the lymphatic glands.

*Mixed Forms.*—In a very small proportion of cases the blood shows an apparent mingling of the myelæmic and lymphæmic characters. I have seen one case, and others have been recorded, where a pure myelæmia showed towards the end a large increase in the absolute and relative number of lymphocytes in the blood, and where post-mortem the organs contained as many lymphocytes as myelocytes.

*Effect of Intercurrent Affections.*—It is important to note the effect upon the blood of those intercurrent conditions, as, for instance, pneumonia and pleurisy, which produce a leucocytosis in normal blood. In rare cases in leucocythæmia such a complication produces no apparent effect on the blood; more frequently the total number of leucocytes remains unaltered, but a much larger proportion of them than before are polymorpho-nuclear neutrophiles. In the greatest number of cases, however, the total number of leucocytes is decreased, and may even descend far below the normal, especially as death draws near. Generally in such cases the proportion of polymorpho-nuclears is increased.

Symptoms.—Except as regards the condition of the blood and the organs usually enlarged, ordinary myelæmia—which, apart from accidents, is always chronic—and chronic lymphæmia do not differ in their symptoms. The patient's attention is usually caught either by the increased girth of the abdomen and the dragging pain from the enlarged spleen, by progressive weakness and dyspnœa, by the enlargement of glands, or by the occurrence of some hæmorrhage, most often from the nose. The disease is almost always thoroughly established by the time patients come under observation, so that its onset must be very insidious. The urgency, or otherwise, of the symptoms depends very largely on the amount of anæmia present. Cases in which this is slight may enjoy fair health, even although the number of leucocytes in the blood is very great, and the enlargement of organs extreme. This enlargement may of course, however, give rise to symptoms by pressure on important organs; and there is a special tendency to dropsy of various forms, and still more to hæmorrhage.

In the alimentary system the main points to note are the frequent occurrence of stomatitis, of gastric and intestinal catarrh, with vomiting and diarrhœa, hæmorrhage from the bowel, enlargement of the liver, and either as a result of this or as part of a general dropsy, the occurrence of ascites. The heart is always enfeebled, and dyspnœa is a marked feature in the disease. All the murmurs and other cardiac changes due to anæmia are usually developed. Thrombosis of capillaries and small vessels from plugs of leucocytes is very common, and is one of the factors which cause hæmorrhages to be so frequent. These occur most often from the mucous surfaces. Epistaxis is the most common, then perhaps hæmorrhage from the bowels, and then follow bleedings from the gums, the stomach, the kidneys, lungs, and uterus. The most serious is, of course, cerebral hæmorrhage, which is not infrequent, while hæmorrhage into joints, into muscles, into serous cavities, or elsewhere, may follow slight injuries or small operative procedures such as tapping the pleura or peritoneum. Hæmorrhage into the retina is often associated with collections of leucocytes visible by the ophthalmoscope during life, or discovered post-mortem. There are no special symptoms associated with the lungs, but bronchitis, pleurisy, pleural effusion, œdema of the lungs, and pneumonia are frequent complications or terminal phenomena. In every case there is at some time fever without apparent cause, very much like that which occurs in pernicious anæmia. There are no constant changes in the skin, though nodules of leucocyte infiltration are not uncommon there, and hæmorrhages may occur. Albuminuria may occur, generally late in the disease, and albumosuria may appear, but the special characteristic of the urine is its constant acidity and the greatly increased amount of uric acid and of the xanthin bases which it contains. These are the result of the increased leucocyte metabolism, and their amount in the urine of a case at any time corresponds generally to the number of leucocytes present in the blood.

Enlargement of the spleen is present to a greater or less extent in all cases of myelæmia, and in a large number of chronic lymphæmic cases. The character of the enlargement is the same in both sets of cases. The organ usually extends downwards and forwards, much more rarely upwards, and its general form is retained, the notches usually persisting. It may pass beyond the middle line, and, as a firm tumour, fill almost the whole of the abdomen, or may be of any smaller size. Generally speaking, the more chronic the case the greater the enlargement. It bears no special relation to the number of leucocytes in the blood; in remissions when the blood becomes nearly normal the organ may diminish somewhat, but more frequently, in my experience, remains unaltered in size. In the acuter cases, again, there may be slight enlargement with a very high leucocyte count. The lymphatic glands are often but little enlarged in myelæmia, though towards the end of a case some of them usually increase in size. I have indeed seen very great enlargement in some cases. In chronic lymphæmia, however, it is the rule to find most of the glands in the body enlarged, especially those of the neck, axillæ, and groins, and usually the internal glands as well. The enlargement is

irregular, painless, and unaccompanied by periadenitis as a rule, and the swellings do not usually give rise to pressure symptoms. The amount of glandular affection varies extremely in chronic lymphæmia. I had the good fortune about a year ago to have four cases of the kind under observation. Two of them showed general and extreme glandular enlargement with but slight increase of the size of the spleen; one had a fair amount of glandular enlargement with a fairly large spleen; while in the fourth the glands were not enlarged at all, and the spleen reached nearly to the pubes. Yet all these cases showed the typical changes in the blood.

The hypertrophy of the bone-marrow cannot be diagnosed clinically, and gives rise to no symptoms.

Acute lymphæmia presents a very different clinical picture from the chronic forms. The fever, hæmorrhages, and anæmia which appear at the end of the chronic cases, usher in the acute ones. The patient passes in a few days from a condition of health to one of extreme prostration, and death usually occurs in from a few days to a few weeks. Curiously enough, the symptoms may be well marked *before* any important changes occur in the blood, but before death there is usually a very great increase in the lymphocytes and a high degree of anæmia. In the very acute cases there is no time for enlargement of either glands or spleen to take place to any great extent; in those less acute the glands enlarge rapidly, the spleen less markedly. The disease is a rare one, occurs usually in young people, and from its rapid onset and course gives one very much the impression of being an infective condition.

Course and Prognosis.—Cases of myelæmia usually live from one to two years from the time they come under observation, but may live much longer; and chronic lymphæmia may last quite as long. Either class of cases may show remissions and exacerbations. The blood may become nearly normal, and the general health improve, or this improvement may occur without any great decrease in the number of leucocytes. Cures have been reported, but all rest either on insufficient evidence or too short a period of observation. As far as we know, the disease is always fatal in the long run. Death is usually preceded by a period of cachexia, and what may be called the normal ending to the disease is by gradual heart failure. Other common causes are by gastro-intestinal symptoms, hæmorrhage into the brain or from mucous membranes, and very frequently by pneumonia. Out of seven fatal cases that I have seen in the last two years four have died of this complication.

The acute cases are always fatal, but sometimes a case may begin acutely and then become rather more chronic and last for a few months.

The prognosis in chronic cases of either variety is of course very grave, but is relatively favourable when the patient is middle-aged, in fair general health, with a fairly vigorous circulation, little or no anæmia, and no enlargement of lymph glands in the myelæmic form. A large spleen means that a case has so far been chronic and may, presumably, remain so. Unfavourable elements in prognosis are youth in the patient, a steady increase in the number of leucocytes in the blood, advancing anæmia, continued fever, hæmorrhages into the skin or large bleedings from the mucous membranes, enlargement of glands in the myelæmic form, dropsy, and of course the presence of any serious complication.

Morbid Anatomy.—The naked-eye appearances of the organs do not differ greatly in the two forms of the disease. The enlargements of organs which were discovered clinically are confirmed, and it is usual to find a much more extensive enlargement of glands than was expected, especially of the abdominal glands. In addition to splenic and lymphatic enlargement, the liver, kidneys, thymus, thyroid, suprarenals, tonsils, and other organs may show more or less enlargement, with a frequency nearly in this order. This enlargement is due mainly to the infiltration of their connective tissue spaces with leucocytes, but partly to the occurrence of infarcts from thrombosis, and hæmorrhages, and to fatty infiltration from the anæmia which is almost always in existence in fatal cases.

The *spleen* is usually firm, and firmer the more chronic the case. Its capsule is often thickened or rough from local peritonitis. On section it is generally of a uniform red colour; the Malpighian bodies are indistinct. Microscopically the pulp is found to be packed with leucocytes like those in the blood, while in chronic cases the trabeculæ and stroma generally are often thickened.

The *lymph glands* vary greatly in size from that of a pea to that of a large plum, are generally oval in shape, and white or pink in colour. In cases where they were much congested, however, or where hæmorrhage had taken place into them, I have seen them of a dark purple colour. They are usually embedded in fat to a greater or less extent, and show no trace of periadenitis, unless they have been exposed to injury or irritation. On section they are soft or succulent. Microscopically they differ in the two varieties of the disease. In lymphæmia they show no trace of the normal structure of the gland. The distinction between cortex and medulla, germ centres and lymph paths, is completely lost, and one finds simply a mass of lymphocytes packed tightly together and obscuring the stroma, and occasional blood-vessels traversing the mass, and often old or recent hæmorrhages. Among these lymphocytes one may find mitotic figures; but one cannot, of course, lay any stress on their presence or absence in the organs of persons dying a natural death whose bodies are not

examined for some time post-mortem. In myelæmia it is exceptional to find the glands so packed with cells of the myeloid type, though I have found it so in some very chronic cases. More usually the greater part of the gland retains the normal structure and appearance, and islets of the myeloid cells are to be found in the peripheral parts of the gland, brought there by hæmorrhage or in the same way as they appear in other organs.

The *bone-marrow* shows alteration in *all* cases —in lymphæmia as well as in myelæmia, and nearly quite as much in the former as in the latter, though it is, of course, impossible to examine all the bone-marrow in the way in which one can examine all the spleen or all the lymph glands. The essence of the change is that the fat which is present everywhere in the marrow, but especially in the shafts of the long bones, is absorbed, and its place taken by cells of the same kind as those found in the blood. Thus the marrow in the shaft of the femur, which is usually examined, instead of being fatty and yellow in colour, is usually pink and firm, the so-called "lymphoid" condition. It is exceptional to find it white, soft, and "pyoid." Microscopically in myelæmia it presents very nearly the normal appearance of red marrow, with the differences that no fat spaces are left, that the giant cells are small and few in number, that the nucleated reds are fewer than usual, and further that the tendency seen in the blood in different cases to a preponderance of special kinds of cells is seen also to a certain extent in the marrow. In some cases eosinophiles predominate, in others neutrophile myelocytes, and so on. In lymphæmia a section of the marrow looks very much the same as that of lymph gland. Probably, however, the replacement of the proper marrow tissue by lymphocytes is never quite complete, though this point has not been fully worked out. It is, of course, evident that this leucocytic hypertrophy in the marrow will reduce considerably the area there which is normally reserved for the formation of red blood corpuscles. This is one cause of the anæmia which is always present, and is probably also the reason why nucleated red corpuscles are so commonly found in the blood; they are pushed out of the marrow, and red corpuscle formation goes on in the blood-stream as it does in the embryo.

The *liver* is usually pale, and fatty from the general anæmia. On close inspection pale zones will be found surrounding the portal spaces, which, when examined microscopically, are found to be caused by infiltration of the connective tissue there with leucocytes. This infiltration also extends to a varying distance between the columns of liver-cells, and similar patches are sometimes found under the capsule. The *kidney* is usually in the same state, and so are the other organs mentioned as showing enlargement. In fact, patches or strands of leucocyte infiltration may be found anywhere throughout the body, in the lungs, the heart-muscle, etc. We do not certainly know whether these are due to metastasis, to the ordinary but here exaggerated diapedesis of leucocytes from capillaries, or to the overgrowth of pre-existing lymphatic nodules.

The *alimentary canal* is usually in a condition of chronic catarrh, sometimes associated with atrophy of the mucous membrane. In chronic cases there is often no special enlargement of the lymphatic sheath of the alimentary tube, but it is noteworthy that in very acute cases of lymphæmia, which are rapidly fatal, this may be almost the only part of the lymphatic apparatus to show enlargement.

Causation and Pathology. — The disease occurs with greater frequency in men, and is found at all ages. The acute lymphæmic form is more common in early life; on the whole, the great majority of cases occur between thirty and fifty. No antecedent disease or condition has been proved to be casually connected with it. Malaria was thought by Gowers to be an antecedent in about one-fifth of the cases, but this is probably an overstatement. I have never seen a case which had a previous history of malaria, and that disease can certainly not be more than an auxiliary in causation.

The early views that leukæmia was a suppuration of the blood or a cancer of the blood, have long been given up, in that form at least. The problem which at present is being discussed is whether the increased number of leucocytes in the blood is good or bad for the whole organism; whether it is a measure of defence against some injurious influence, or a useless proliferation of blood-cells; whether, in fact, it is a symptom or a disease. The analogy of leucocytosis in infective conditions is, of course, in favour of the former view, and Ehrlich is the principal upholder of that theory. He takes up the position that myelæmia is a mixed leucocytosis, and is derived from a change in the bone-marrow similar to that which accompanies an ordinary leucocytosis. In order to support this view he accepts some observations by Jolly, who declares that both neutrophilic and eosinophilic myelocytes are amœboid on the warm stage, and Ehrlich lays it to the charge of imperfect methods that this phenomenon is not oftener seen. He is, of course, obliged to take this position in order to show that myelæmia is an "active" leucocytosis, and thus to support his contention that the marrow produces only granular leucocytes and that these only are attracted chemiotactically into the circulating blood and make their way into it by active immigration. The secondary deposits in the spleen, lymph glands, and other organs he regards as metastases from the marrow. Lymphæmia he puts on quite another footing. He regards

it as a primary disease of the lymph glands which leads to increased formation of lymphocytes, and to the mechanical flooding of the blood with these in a passive way, and not as an active immigration—the result of chemiotaxis. He brings it into line with the lymphocytosis which occurs when there is an increased lymph circulation in a greater or smaller area of glands, as in digestion, in irritation of the intestine in children, and so on. According to him, therefore, myelæmia and lymphæmia are processes essentially different in origin. The former would be due to some noxious substance in the blood which acts chemiotactically on the marrow and draws its cells into the circulating fluid, and would therefore be primarily a blood disease to which the hypertrophy of the marrow is secondary. The latter would be due to something in the lymph glands which causes them to hypertrophy and to pour into the blood an excessive number of lymphocytes; it would therefore be primarily a disease of lymph glands, and only by accident, as it were, a disease of the blood at all. Ehrlich, in common with all recent writers, has ceased to believe that the spleen has any causal relation to either disease. The reasons for this view are first, that there are no special splenic leucocytes; second, that evidence is slowly accumulating to the effect that apart from the production of lymphocytes in the Malpighian corpuscles, the spleen acts either simply as a reservoir of blood or as a blood-destroying organ rather than as a blood-forming organ; and third, that enlargement of the spleen does not take place in acute leucocythæmias, but is rather an indication and a measure of chronicity. Muir suggests, indeed, that the enlargement of the spleen may be an attempt to deal with and destroy the excess of leucocytes; but it is difficult to see how this is to be effected without an enlargement of the Malpighian corpuscles, the only structure in the spleen which could produce leucocytes capable of acting as scavengers. Such an enlargement does not take place.

Ehrlich's view is, in my opinion, too artificial, does not explain those cases of lymphæmia where there is little or no enlargement of lymph glands, and quite fails when it is applied to acute lymphæmias; and I am much more inclined to accept the views of Neumann's school, whose most recent exponent is Walz, that the excess of leucocytes in both lymphæmia and myelæmia is derived from the marrow. In all lymphæmias which have been fully observed, no matter how rapidly fatal they were, a great excess of lymphocytes has been found in the marrow. Lymphocytes are normally present there, though not in large number; and all recent work has gone to show that the marrow is the most adaptable tissue in the body, that according to the needs of the organism it may contain in excess either normoblasts, megaloblasts, eosinophiles or myelocytes, and their descendants the polymorpho-nuclear neutrophiles. There seems to be no reason why it should not contain an excess of lymphocytes in turn, except the theoretical opinion to which Ehrlich's name gives weight, that the marrow is reserved for the production of granular cells. To those who, like myself, hold that all leucocytes are derived from the same stock, and that their different varieties are due merely to differences of environment and to specialisation of function, this argument does not carry much weight; and the acceptance of the view that all leucocythæmias of both varieties are myelogenic would explain all the facts of their pathology, would account for their being indistinguishable clinically except for the examination of the blood, and would clear the way for the search for the prime cause of the disease. Frankly, we do not know what this is. It may be something in the blood or in some other organ, but acting through the blood, which attracts the leucocytes from the marrow and causes its hypertrophy, or it may be something in the marrow which causes its cells to proliferate, to take up the available free space and to pass out into the blood. Bacterial organisms have been described as present in the blood, but there has never been any sufficient reason given for us to believe that they are of importance. The most recent, most elaborate, and most plausible attempt to find the cause is that of Löwit, who has described two organisms, one as the cause of myelæmia, the other of lymphæmia, which he calls "hæmamœba leukæmiæ magna" and "hæmamœba leukæmiæ parva vivax" respectively. He considers them to be sporozoa, and nearly related to the malaria parasite. His work with regard to lymphæmia and its supposed parasite is so incomplete that I need not discuss it; his views are meant to stand or fall by their application to myelæmia, which he has studied more fully. He states that the parasites are usually found in the blood in varying numbers, that they occur in the leucocytes as a rule, seldom in the plasma, and never in the red corpuscles. The leucocytes attacked are the small and large mononuclear forms—the lymphocytes and transition forms—never the eosinophiles, and only once a neutrophile cell. The bodies are amœboid, and may be sickle-, crescent-, or spindle-shaped, or rounded. He describes flagellate forms, but these are obviously artefacts; indeed, Löwit allows that they are more numerous when there are many injured or badly fixed leucocytes in the preparation. Inoculation of rabbits with myelæmic blood or parts of organs does not produce myelæmia, but "parasites" are found in the blood and organs which are like those found in the human subject; there is also a lymphocytosis lasting for some months, and albumoses are found in the urine, as is frequently the case with myelæmic patients. Cats, guinea-pigs,

and dogs do not give positive results with inoculation, though it may be remarked that in the dog and cat spontaneous leucocythæmia has been observed. Löwit's method of demonstrating the "parasite" is as follows. Films are thoroughly fixed by heating them for one to one and a half hours at 110°-115° C. (alcohol must not be used, though, curiously enough, in the tissues the parasites show best in alcohol-hardened organs!), then stained for half an hour in a concentrated watery solution of thionin at room temperature, washed, dried, differentiated for 10-20 seconds in iodine 1 part, iodide of potash 2 parts, Aq. dest. 300 parts, washed in water, dried, and mounted in balsam. The parasites are then of a green colour. Löwit points out their resemblances to and differences from all enclosures and plasmolytic products—the distinction is evidently difficult—and he also makes the damaging admission that the stain does not succeed well when it is freshly made, but only when yeasts and other fungi have developed in it! Of course the first essential in a stain used to demonstrate organisms is that it must itself be free from organisms.

Löwit's idea of pathogenesis is that when the organism is once introduced to the blood it lives in the leucocytes, renders them functionless, and ultimately destroys them. They are then replaced by fresh leucocytes from the marrow; but these in turn become provender for the parasites, and so a vicious circle is set up. Curiously, however, he entirely overlooks the fact that, on his showing, all leucocythæmias should be lymphæmias; for the parasite lives in and demands lymphocytes, and he gives no explanation of the presence of myelocytes or eosinophiles. We know definitely, however, that the marrow supplies only those cells which are asked of it.

I am afraid Löwit's views cannot be accepted, partly for the reasons I have interpolated in describing them, but also because the bodies he figures present no common morphological characters, and, incidentally, have no resemblance to the malaria parasite. He seems to have figured everything which stained in the desired way, and has evidently included all sorts of objects. Some of these *may* be parasites, but we have no means of knowing which. At the German congress of physicians, in April of this year, Türck suggested that Löwit's bodies are artefacts, produced from the granules of the basophile leucocytes, or mast-cells, by the method he employs. This view would quite explain their presence in myelæmic blood and organs, where, as I have pointed out, basophiles are numerous; their absence from lymphæmic blood, where no basophiles are found; and their occasional presence in lymphæmic organs. Ehrlich and other speakers supported Türck, and my own observations point in the same direction.

Earlier observers had described amœbæ or other parasites as the cause of leucocythæmia, but none of them have made good their case.

Another question of much importance is that of the relation of leucocythæmia to Hodgkin's disease, or pseudo-leukæmia, as it is called in Germany. This will be discussed when the latter disease has been described (Lymphadenoma).

Diagnosis.—This depends ultimately in every case upon the examination of stained films of the blood, and the careful observation of the kinds of leucocytes present. A fresh film does not enable us to distinguish the condition from a large leucocytosis, and there are many conditions which cause splenic or glandular enlargement which may be associated with leucocytosis. Of course a leucocyte count of 500,000 or so per c.mm. would put the matter beyond doubt, but it is not in cases with large leucocyte counts that doubt is likely to arise. It is in the numerous cases where the leucocytes, either temporarily or permanently, do not rise above, say, 100,000 or less, or where a remission or an intercurrent affection has brought the leucocytes down to normal and altered the general appearance of the film, that difficulty occurs, and there are some cases where the minutest care in examining the films and in weighing the evidence derived from them is necessary to arrive at a correct diagnosis. It must be remembered that the early symptoms of the chronic disease may be gastric, intestinal, respiratory, or cardiac, and that these may appear before the spleen or glands have enlarged sufficiently to attract attention. It ought, of course, to be a rule that the blood should be examined in *all* obscure cases, and it may be borne in mind that while a fresh film will not always enable us to diagnose the condition with certainty, it will at all events give either negative evidence or a warning that a fuller examination is necessary.

Enlargement of the spleen in the malarial cachexia, in splenic anæmia, in tumours and waxy disease of the spleen, tumours of the kidney and suprarenal body, and hydronephrosis may all give rise to error until the blood is examined, and in the same way the enlargement of lymphatic glands in chronic tuberculosis and in lymphadenoma may simulate the lymphæmic form of the disease. One caution that must be given in regard to this is that it is necessary to remember that in children the percentage of lymphocytes in the blood is much higher than in adults; but even in very young children it seldom rises above 50 per cent, while in lymphæmia the percentage is always over 80, usually over 90. The more acute a lymphæmia is, the less likely is it to be diagnosed. The fact that leucocythæmia may be an acute disease, possibly fatal in a few days, is not yet widely recog-

nised, and cases are much more likely to be labelled typhoid, purpura hæmorrhagica, or ulcerative endocarditis, than to be diagnosed for what they are. When the glands enlarge the blood is likely to be examined, and the diagnosis should then be made.

In children there will sometimes be difficulty in making the diagnosis between pernicious anæmia and leucocythæmia. All grave anæmias in children are apt to be accompanied by enlargement of the spleen, and in the pernicious form there are more nucleated corpuscles in the blood than in the adult, and usually a leucocytosis which may include a fairly large percentage of myelocytes. The diagnosis must be made by the preponderating features in the blood, and is often very difficult. The name "anæmia pseudo-leukæmica infantum," given by von Jaksch to these difficult cases, does not seem to me to correspond to any well-defined clinical entity, and should be discarded.

TREATMENT.—As in all blood diseases where the nutrition of the cardiac muscle is likely to be impaired, rest in bed is essential, with careful diet and general attention to ordinary hygienic principles. The bowels must be regulated, but without producing diarrhœa, which is always injurious. The only remedy which can be given with any confidence that it will be of use is arsenic, and this should be given in increasing doses up to the largest that can be borne, and continued for a long time. Iron in various forms, quinine, mercury, and many other drugs have been given, but without any constantly good results. The inhalation of oxygen has been reported to be useful, but one fails to see how its action can be other than temporary; while in the only case in which I have seen Ewart's inhalation of carbon dioxide tried it seemed to me to hasten rather than retard the fatal result. It is useless to try to reduce the size of the spleen by drugs or internal remedies, and probably unjustifiable to remove it in this disease, as the mortality after the operation is so high and the procedure entirely without avail in arresting the disease. Bone-marrow in various forms has been given; but one fails to see how it could possibly be of use, and experience has confirmed this view. It must not be forgotten, in estimating the effect of remedies, that the disease is one in which, as in pernicious anæmia, though not with the same frequency, spontaneous improvement may take place, and this is specially apt to occur when the disease is diagnosed and the patient's surroundings improved. Until we gain a more exact knowledge of the causation of the disease we must be content with the benefit to be obtained from the empiric use of arsenic. One of the main tasks of the physician is the avoidance of complications, especially those associated with the alimentary and respiratory systems. For example, the idiosyncrasies of patients with regard to food must be carefully studied, for if a gastro-intestinal catarrh be allowed to establish itself, it is often a very difficult matter to get rid of it. The same holds good, for instance, with bronchitis. Prevention of complications in these conditions is not only better, but a great deal easier than cure.

[*X-Ray Treatment of Leukæmia.*—The remarkable effects which Röntgen rays exert on sarcomatous and other tumours have naturally suggested them as likely to be serviceable in leukæmia. The earliest cases of this disease so treated were reported in America, and the credit of priority in the introduction of a remedy which, though not curative, is yet more likely than any other to prolong life and mitigate suffering, belongs to Pusey and Senn, who in 1903 and 1904 first brought the subject under the notice of the medical world. Since that time the treatment has been very extensively tested, and its value can now be fairly accurately estimated.

As has been said, the X-rays do not cure leukæmia permanently; but in many instances, nevertheless, splenic and glandular enlargements subside, the blood becomes practically normal, and the patient enjoys a period of respite from symptoms which may be of very considerable duration. The following tables, compiled by Pancoast, give the results of 123 cases treated between 1901 and 1906.

| | Spleno-medullary. | Lymphatic. | Unclassified. |
|---|---|---|---|
| Symptomatic cure . | 33 | 11 | 3 |
| Improved . . . | 22 | 8 | 1 |
| Improved, and still under treatment . | 14 | 1 | 0 |
| Unimproved . . | 13 | 10 | 3 |
| Unknown . . | 1 | 0 | 2 |
| The final results in 63 cases are :— | | | |
| Still living and well . | 2 | 2 | 0 |
| Symptomatic cure, fatal relapse . . | 11 | 5 | 0 |
| Symptomatic cure, serious relapse . | 5 | 0 | 0 |
| Improvement, fatal relapse . . . | 14 | 3 | 1 |
| Unimproved, died . | 5 | 8 | 3 |
| Relapsed and under treatment . . | 4 | 0 | 0 |

The duration of symptomatic cure is variable. Though it often lasts only a few months, four patients at least are reported as alive and well three, four, and six years after treatment. Myelocytic leukæmia appears to respond more readily than the lymphocytic type, and patients suffering from the acute form of the disease derive little benefit, or possibly harm, from the treatment.

*Technique.*—A hard tube should be used, as being more penetrating and less likely to cause dermatitis. The sittings should be brief, from

three to ten minutes thrice a week or oftener. Exposures should be made over the spleen; the bones and enlarged glands have also been rayed. Neighbouring parts may be protected by lead shields.

*Effects of the Rays on the Hæmopoietic Organs and Blood.*—As a rule, the spleen becomes softer and diminishes in size after a few exposures. In many cases it speedily returns to normal dimensions; in a few instances, however, even repeated applications have produced no effect. Enlarged glands become smaller, though the change in them is less marked than in the spleen. Tenderness of the bones disappears. The general effect on the blood is to produce a steady diminution of the leucocytes. There is, it is true, an immediate temporary rise of short duration, due in all probability to the profound changes in the lymphoid tissues causing a flooding of the blood with white cells; but this is followed by a speedy fall, which may amount to nearly 200,000 per c.mm. after a single exposure. Generally, however, the diminution is more gradual—perhaps twenty or thirty exposures spread over five or six weeks being required to bring the leucocytes to an approximately normal number. The leucocytes, moreover, are altered qualitatively as well as quantitatively, the abnormal elements practically disappearing. Arneth and others have worked out the changes which occur. The lymphocytes, particularly the larger forms, steadily diminish; the neutrophile myelocytes dwindle and may vanish altogether, while the polynuclears correspondingly increase. Mast cells fall, while the eosinophile group becomes normal. There is, at the same time, a rise in the red corpuscles, and a disappearance of abnormal forms. Broadly, then, the effect of the rays is to produce an aleukæmic phase. Myelocytes are more susceptible than lymphocytes, and in lymphocyte leukæmia, though the count may become normal, the percentage of lymphocytes usually remains high.

With the symptomatic cure, the general health is correspondingly improved; relapse, however, occurs sooner or later, and the treatment must be repeated with, as a rule, less prospect of success than before.

Satisfactory on the whole, considering the nature of the malady, as the treatment is, it is not entirely devoid of risks. The rapid destruction of large numbers of leucocytes is associated with the liberation of toxic products, and it is not surprising that some cases of fatal toxæmia have been reported. Sudden death has occurred from this cause in not a few instances, and in some of these nephritis has been found. The treatment, therefore, should not be entered upon when there is any evidence of renal disease, or any suspicion of toxæmia—cautions which practically exclude acute leukæmia from the scope of X-ray treatment. Among minor inconveniences, dermatitis, palpitation, vomiting, and diarrhœa have been reported.

*Nature of the Action of X-Rays.*—Theoretically, the diminution of leucocytes produced by the rays may be due either to inhibition of the function of the blood-forming organs, or to stimulation of leucolysis. There is reason to suppose that the latter view is correct. In animals subjected to exposure to the rays, Heinecke, whose work has been confirmed and is generally accepted, found marked evidence of destruction of the lymphocytes throughout the body—in the splenic pulp, marrow, glands, and follicles of the intestines. The change begins within three hours of exposure, and is complete in from thirty-six to forty-eight hours. Probably the leucocytes in the circulation are also destroyed. The red corpuscles, on the contrary, resist the action of the rays. In man, accompanying the diminution of the leucocytes, there is an excessive output of urinary phosphorus and nitrogen.

X-ray treatment has also been employed in pseudo-leukæmia, in which the results are said to be better than in leukæmia (about a quarter of the patients treated remain alive and well for three or four years—*Pancoast*), in polycythæmia (without benefit), and in pernicious anæmia. In the last-named disease the liability to the occurrence of toxæmia, and the frequency of kidney lesion, seem to contraindicate the remedy.]

## Leucocytosis.


DEFINITION . . . . . 400
PHYSIOLOGICAL LEUCOCYTOSIS . . . 401
PATHOLOGICAL LEUCOCYTOSIS . . . 401
FORMS—
1. ORDINARY (FINELY GRANULAR) LEUCOCYTOSIS . . . . . 401
*Leucopenia* . . . . . 402
*Presence of Myelocytes* . . . 403
2. LYMPHOCYTOSIS . . . . 403
3. EOSINOPHILE LEUCOCYTOSIS . . 404
NATURE OF LEUCOCYTOSIS . . . 404


*See also* ANÆMIA; ANÆMIA, PERNICIOUS; BLOOD (*Physiological and Pathological Variations*); CHEST, CLINICAL EXAMINATION OF (*Blood Counts*); CHLOROSIS (*Pathology*); MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*Symptoms, Blood*); PNEUMONIA, CLINICAL (*Complications*); RHEUMATISM, ACUTE (*Symptoms, Changes in the Blood*); STOMACH AND DUODENUM, DISEASES OF (*Special Symptomatology of Cancer of the Stomach, Diagnosis*); SUPPURATION (*Leucocytosis*); WHOOPING-COUGH (*Complications, Leucocytosis*).

THE term is applied to an increase above the normal of the leucocytes per c.mm. in the circulating blood; but it does not embrace the increase of leucocytes met with in leuco-

cythæmia. In conditions of health the average number of leucocytes varies considerably in different individuals, but may be said to be as a rule between 6000 and 10,000 per c.mm. It is impossible to state exactly at what point the variation of the leucocyte number becomes abnormal, but it may be stated that it is rare for the number to rise above 12,000 or to fall below 5000 without some abnormal condition being present. The average normal number may be increased under certain physiological conditions, and such a change is accordingly spoken of as *physiological leucocytosis*. It occurs to a slight extent after a meal — digestive leucocytosis—being usually most marked three or four hours afterwards. The increase is said to be more pronounced after a diet rich in proteids, but in any case it rarely exceeds 20 per cent of the normal number. The number of leucocytes may also be increased during the later months of pregnancy, and to a rather more marked degree after parturition. It is important that these variations should be known and borne in mind, otherwise slight rises in the leucocyte number may sometimes be misinterpreted In infancy also, especially in the few weeks following birth, the leucocyte number is increased, and at this period the proportion of lymphocytes is unusually high, being often about 50 per cent (see article on "Blood"). In these various conditions of physiological leucocytosis, with the exception of the increase in infancy, it has usually been found that the various forms of leucocytes are uniformly increased; but fuller details on this point are still desirable.

It is, however, with the leucocytosis occurring in disease that we have to do in this article, and we shall also treat here of the converse condition, namely, a fall in the leucocytes—*leucopenia*. In the great majority of cases of pathological leucocytosis, the increase in the number is due mainly, and often exclusively, to an increase of the finely granular (neutrophile) polymorpho-nuclear leucocytes. This is a well-established fact, and accordingly the term *ordinary leucocytosis* is frequently used with the significance that the increase is on the part of these cells. The term *lymphocytosis* strictly means an increase of the lymphocytes per c.mm., but is often used as indicating a percentage increase in the number of these cells. The latter use of the term is somewhat unfortunate for scientific purposes, as in many cases where the leucocyte number is diminished, the relative proportion of lymphocytes is increased, whilst their total number is not so. It is therefore advisable that the term should be only employed to signify an actual increase of the lymphocytes. It may be stated here that such actual increase is comparatively rare, if we except the lymphatic form of leucocythæmia, and in some cases of tumours of the lymphatic glands. The term *eosinophile leucocytosis* or *eosinophilia* is used to signify an increase in the percentage number of the eosinophile leucocytes. In this case the percentage increase practically always indicates an actual increase. We shall now consider more in detail these variations in the leucocytes as they are met with clinically.

1. Ordinary (finely granular) Leucocytosis.—The ordinary leucocytosis, due to an increase of the polymorpho-nuclear neutrophile leucocytosis, occurs in a great many different conditions. Such conditions, which have been variously classified, may be placed for convenience in the following groups; but we shall have to consider afterwards whether in the different groups the leucocytosis is not in nature essentially the same: — (*a*) Leucocytosis in inflammatory and infective conditions; (*b*) Toxic leucocytosis; (*c*) Post-hæmorrhagic leucocytosis; (*d*) Cachectic leucocytosis, especially associated with malignant diseases.

(*a*) The first group embraces a great many diseases, many of which have now been proved to be due to special micro-organisms, whilst in others the nature of the causal agent is still unknown. A well-marked leucocytosis is common in pneumonia, erysipelas, diphtheria, scarlet fever, plague, smallpox (in the suppurative stage), etc.; in acute inflammatory processes, especially when they are attended by suppuration, *e.g.* in peritonitis, arthritis, appendicitis, abscesses in internal organs, in most gangrenous inflammations, in pyæmia, and in most cases of septicæmia. It will be noted that in the majority of such conditions there is present a local lesion with emigration in large numbers of the finely granular leucocytes into the tissues, and it may be stated as a general rule that up to a certain point the leucocytosis is proportional to the severity of the affection or the extent of the local lesion. This rule, however, must be taken in a very general sense, as several factors are involved. An empyema will be attended by a greater leucocytosis than a small local abscess; but the degree of leucocytosis in pneumonia does not vary strictly with the amount of lung tissue affected, just as the severity of the disease does not depend on this alone. Further, there are variations depending upon individual peculiarities, and when the affection becomes very severe and marked toxæmia is present, the leucocytosis may diminish, and the leucocyte number may even fall below the normal. The actual number of leucocytes in such conditions as those mentioned, of course, varies greatly, but 15,000 to 30,000 may be said to be the common upper limit. Leucocytosis in which the number rises above 40,000 must be considered extreme. Of the diseases mentioned, pneumonia is that in which the condition of leucocytes has been

most fully worked out, and we may state the chief facts by way of illustration. The number of leucocytes rises within an hour or two after the rigor; in fact, in some cases has been found to be raised even at the time of rigor. The number rises with comparative rapidity, and during the period of fever remains high, showing variations of irregular type. It usually commences to fall a short time before the crisis, and falls rather more gradually than the temperature does; hence on the day after the crisis the number may still be a little above normal. During the leucocytosis period the increase, as we have said, is on the part of the finely granular neutrophile leucocytes. In delayed resolution the leucocytosis persists, though usually diminished in degree, their proportion often rising to 90 per cent or even more. The lymphocytes may show a slight actual decrease, and one striking and well-authenticated fact is that the eosinophiles may practically disappear from the peripheral circulation; at least, it may be impossible to find a single example on examining a number of films. At or shortly after the crisis the hyaline leucocytes may show a slight relative increase, whilst the eosinophiles reappear in the blood, and a day or two afterwards may show a percentage above the normal. A fall in the temperature without amelioration in the condition, a "pseudo-crisis," is usually unattended by diminution in the leucocytosis. Furthermore, in very grave cases leucocytosis may be absent and leucopenia may be present throughout the case; or a leucocytosis, present at first, may gradually disappear and give place to leucopenia, even although the temperature remains high; sometimes also a few myelocytes may appear in the blood. These conditions are always to be regarded as of grave significance. The blood examination in pneumonia is undoubtedly of considerable value; it may aid the diagnosis in cases of deep-seated pneumonia: it enables one to judge of the significance of variations of the temperature; and from what has just been stated, it will be seen that it affords valuable assistance in the matter of prognosis.

In the other diseases mentioned above the leucocytes show somewhat analogous changes to those in pneumonia, though they may not be so pronounced. The increase of the finely granular leucocytes is again the prominent feature, whilst the eosinophiles are in most cases diminished and not infrequently absent. The fall of the temperature by crisis is attended by a disappearance of the leucocytosis, and a rise in the number of the hyaline cells, and it may be of the lymphocytes, is not uncommon. Also, it may be stated as a general rule that the disappearance or absence of the leucocytosis occurring in a disease (where leucocytosis is the rule) associated with severe symptoms, is to be regarded as a graver sign than when leucocytosis is well marked. The infective diseases in which a normal or subnormal leucocyte number is the rule are mentioned below.

(*b*) Toxic Leucocytosis.—The term is applied in a somewhat loose way to conditions in which there is manifestly some toxic agent in the blood, but in which there is no distinct evidence of infection. We shall afterwards have to discuss whether the leucocytosis described under heading (*a*) is not really of the same nature as toxic leucocytosis. Here we may mention as examples, gout, some cases of malignant jaundice, acute yellow atrophy of the liver, some cases of lead poisoning, chronic Bright's disease, etc. Leucocytosis may be also produced by the administration of various drugs, *e.g.* especially volatile oils, pilocarpine, etc., and has also been produced experimentally by the injection of a great many different substances, as will be described below. In such conditions the leucocytosis presents the same characters as in the first group, but is on the whole less in degree.

(*c*) Post-hæmorrhagic Leucocytosis.—When a large hæmorrhage occurs leucocytosis appears usually within two or three hours, continues for a day or two, and if there be no renewal of the hæmorrhage, gradually disappears. If, however, there are repeated hæmorrhages and a condition of marked anæmia results, the leucocytosis is found along with the anæmic conditions. Though the increase is usually quite distinct the number does not often rise above 15,000. Here the course is manifestly different from that in the two previous groups. Its exact mode of operation is not quite clear, but it is undoubtedly connected in some way with the process of dilution which the blood undergoes after hæmorrhage. It certainly is not due to retention of the leucocytes in greater proportion than the red blood corpuscles, as it is absent immediately after the hæmorrhage, and takes some time to appear.

(*d*) The number of leucocytes in cases of malignant disease varies greatly, but it may be said as a rule that where cachexia with considerable anæmia is present, leucocytosis is the rule. This occurs both with cancer and with sarcoma, but in some cases there is a considerable leucocytosis before the cachectic stage is established. In extreme anæmia the presence of leucocytosis, with increase in the blood-plates and diminution of the hæmoglobin per corpuscle, indicates a cachectic (secondary) anæmia.

*Absence of Leucocytosis, Leucopenia.* — The most infective conditions in which leucocytosis is absent are typhoid fever, malarial fever, tuberculosis uncomplicated by suppuration or cavity formation, measles, and most cases of influenza. In all these conditions the number tends rather to be below than above the normal, and this is especially the case in typhoid fever, notably in the later period of the disease. The diminution is chiefly on the part of the finely granular cells, though the others may be slightly affected. A

leucocyte count of 3000 or 4000 in the third week of the disease is not uncommon. This fact was, of course, of greater practical importance before the introduction of the serum method, but even yet there occur cases in which it is of value. In acute miliary tuberculosis the leucocytes are usually about the normal condition, but in phthisis with cavity formation a more or less marked leucocytosis is present. In connection with these facts it must, of course, be kept in view that in the diseases where leucocytosis usually occurs, it may be absent, or even a converse condition may be present, as explained above.

It follows from what we have stated that the condition of the leucocytes alone must not be accepted in any hard and fast sense in relation to diagnosis, but from the same fact it is evident that when the diagnosis is otherwise established, a variation from the condition usually present may be of great importance. Thus, for example, the fall in the leucocyte number, without corresponding fall in the temperature, is of grave significance, and in typhoid fever the appearance of leucocytosis may suggest the presence of some secondary inflammatory or suppurative complication.

Pneumonia has been specially mentioned as an example in which the disappearance or absence of leucocytosis is of grave omen; but a similar change may occur in other diseases. Thus it has been observed in some forms of very grave septicæmia and in some cases of diphtheria; in fact, it may be interpreted as evidence of a very high degree of general poisoning. It must be clearly understood, however, that in many conditions a well-marked leucocytosis may be present up to the time of death.

In addition to these more acute infective conditions, in which leucopenia may occur, the number of leucocytes is diminished as a rule in various chronic diseases. Among such may be mentioned pernicious anæmia, chlorosis, some forms of severe anæmia attended with purpura, hæmophilia, some cases of goitre, and certain cases with enlargement of spleen and anæmia, to which the term "splenic anæmia" is given. Frequently in such conditions the leucocyte number is about 3000 per c.mm.; but in some examples of severe pernicious anæmia the number may be uniformly about 1000 per c.mm. On the other hand, in some cases of pernicious anæmia the number is little diminished below normal. We do not yet know the significance, from a prognostic point of view, of these variations in different cases. Here again the diminution is chiefly on the part of the finely granular leucocytes, and therefore, according to the above definition, lymphocytosis is present. In some cases of pernicious anæmia, for example, the lymphocytes may number 80 per cent of the total number of leucocytes, but there is, nevertheless, usually no actual increase in the number of lymphocytes; in fact, their number is more often below the normal than above it.

*The Presence of Myelocytes.*—These are large cells, often measuring 14 or 16 m. in diameter, with a rounded oval or slightly indented nucleus, poor in chromatin, and with finely granular protoplasm. Their presence in large numbers is an outstanding feature of the spleno-medullary leucocythæmia or myelocythæmia, and it was for some time believed that they occurred in no other condition. More extended observations, however, have shown that they appear in the blood, though in very small numbers, in a variety of conditions. They are not infrequently met with, for example, in pneumonia, and other conditions, especially when the conditions are of grave nature, and the leucocyte number is low or falling. Even in cases running a favourable course, with a well-marked leucocytosis, one or two myelocytes may be present, and also, what is of some importance, a few cells intermediate in character between them and the ordinary polymorpho-nucleated leucocytes. In cases of grave anæmia, also, a few myelocytes may appear in the blood, and in some cases of marked cachexia due to malignant disease a considerable proportion has been observed. It must be clearly understood that the cells to which the term myelocyte is applied are those containing fine neutrophile granules. This fact has not been sufficiently attended to, and accordingly the larger hyaline leucocytes of the blood have been mistaken for myelocytes, and error has accordingly resulted in the record of cases. There can be practically no doubt that these cells are normally present in the marrow only; according to our opinion they are the progenitors of the finely granular leucocytes of the blood. It is somewhat difficult to state exactly the conditions on which their entrance into the blood-stream depends, but it is of considerable significance that a few nucleated red blood corpuscles are in a very large proportion of cases present in the blood along with the myelocytes. This is not only the case in severe anæmia, but also in severe infective conditions. We can only state that their appearance is due to some disturbance of the cellular arrangement in the bone-marrow—an arrangement by which both they and the nucleated red blood corpuscles are prevented from entering the circulation in the normal state. Such a disturbance may occur in the case of great dilution of the blood (anæmia), as a result of toxic agency, as in many infective conditions, and in some cases the result of tumour growth in the bone-marrow.

2. Lymphocytosis.—A percentage increase of lymphocytes, of course, occurs where there is leucopenia with diminution in the finely granular leucocytes (*vide supra*). An actual increase has been observed in malignant disease affecting the lymphoid tissue, and also in some other conditions of enlargement, *e.g.* tubercular, also in

some cases of gastric and intestinal catarrh, and in whooping-cough. The increase in these conditions appears to occur more readily and to be more marked in children than in adults. According to Ehrlich's view, lymphocytosis occurs where a raised lymph circulation in a more or less extensive area washes an increased number of lymphocytes out of the lymphoid tissue, *i.e.* is the result of a mechanical process, as opposed to chemiotaxis, which is the basis of ordinary and eosinophile leucocytosis. Further investigation is, however, necessary before we can definitely exclude chemiotaxis as a factor in the occurrence of lymphocytosis.

3. Eosinophile Leucocytosis. — Whatever may be the actual genetic relationship between the finely granular and coarsely granular oxyphile leucocytes of the blood, there is no doubt that in pathological conditions they behave as two distinct classes, both as concerning their emigration to the tissue and as regarding their variation in number in the blood. The increase of eosinophiles has been specially studied within late years, and now certain sets of conditions have been recognised in which it is the rule. (1) In asthma there is often a most marked increase in the number of eosinophiles, not infrequently up to 10 or 20 per cent; the increase is most marked during an attack of the disease. (2) In certain acute and chronic diseases of the skin—pemphigus, urticaria, psoriasis, etc. Here again the proportion of eosinophiles may be very markedly increased, though the total number of leucocytes does not rise much above the normal. (3) In helminthiasis, eosinophilia appears to be the rule. In trichiniasis the increase is very marked, and in one case at least the number observed was about 50 per cent of the total number of leucocytes. In affections with other round-worms, such as ankylostoma duodenale, and even ascarides and oxyurus, eosinophilia occurs; in fact, this increase appears to be the rule. An increase in the eosinophiles has also been observed in some cases of malignant disease, especially with metastases in the bone-marrow; the post-febrile leucocytosis has been referred to above.

With regard to diminution in the number of eosinophiles, the most important condition is ordinary leucocytosis, especially those of rapid occurrence, *e.g.* in acute pneumonia. As already mentioned, the leucocytes may practically disappear from the blood, and in other conditions their number may be at least very much diminished.

It is important to note that the eosinophiles are very numerous in the bronchial secretion in asthma and in the affected tissues in those skin diseases attended with eosinophilia. One other interesting point with regard to such skin affections is that when an acute inflammatory or suppurative condition is added, the leucocytes which emigrate from the blood-vessels are of the finely granular neutrophile variety. This would go to show that in the particular affection there is present some substance which acts chemiotactically upon or attracts the eosinophiles, but that agents that produce suppurative processes attract the neutrophiles. In short, all the evidence goes to show that the two classes of cells are attracted by different substances, but that their emigration into the tissue, and increase in number in the blood, are brought about by similar mechanisms.

*Nature of Leucocytosis.* — We have so far considered the chief variations in the different forms of leucocytes met with under clinical conditions, but it is important that the vital processes underlying these variations should be understood. The relations of the different forms of leucocyte are still subject of controversy, and we shall only make general statements which seem to us of importance, as well as justified by fact. If we look at the question from the experimental side, we find that leucocytosis may be produced by a great variety of methods, the chief of which are the inoculation with certain organisms and the injection of certain chemical substances. Of the latter we may mention three chief groups as examples, viz.: (*a*) Certain bacterial products or substances separated from bacteria; (*b*) extracts made from various tissues, especially those rich in cells, such as spleen, lymphatic glands, etc.; (*c*) certain definite chemical substances, *e.g.* peptone, curare, nucleic acid, etc. By the injection of these substances the number of leucocytes may be doubled in the course of an hour or two, and in such cases also the increase is on the part of the finely granular cells. What, then, is the source of these cells added to the blood? Ehrlich holds, and our own observations completely confirm his contention, that the chief, if not the exclusive, source of these cells is the bone-marrow, where they are formed from the finely granular myelocytes. Further, in the normal condition a considerable number of these polymorpho-nuclear leucocytes are present in the marrow in close relation to the blood-stream, forming a reserve store as it were. Thus a means is afforded for a rapid addition of these leucocytes to the blood. There is, we believe, practically no doubt that, just as in inflammation the emigration of leucocytes is guided in by chemiotaxis, so also their passage from the bone-marrow into the blood is brought about by a similar agency. We have also been able to show that in long-standing suppuration, where there is a great drain on the finely granular leucocytes, there also occurs a great increase of the finely granular myelocytes, and evidence of increased multiplication amongst these cells —a change which we consider can only be interpreted as a provision for the increased demand. (To a marrow thus changed we have applied the designation "*leucoblastic*," as contrasted with

the *hæmatoblastic* type which occurs after hæmorrhage.[1]) We may add that the arrangements in the marrow are such as to bring the leucocytes in it directly under the influence of any substance circulating in the blood, and at the same time are such as to admit their ready passage from the marrow into the blood. To put the matter shortly, then, local suppuration is due to agencies exerting positive chemiotaxis on the finely granular leucocytes. When these substances are absorbed in such quantities as to influence the cells in the marrow, then a blood leucocytosis occurs. As we have already stated, the mechanism of the eosinophile leucocytosis is in all probability of the same nature, the eosinophile leucocytes being derived from the eosinophile myelocytes.

We can therefore understand that in the various diseases the factor determining the leucocytosis is not the high temperature, nor even the extent of inflammatory change *per se*, but the presence in the blood of substances which exert positive chemiotaxis on the finely granular leucocytes. According to this view, also, it is at once clear how that in practically every case where there is an extensive inflammation, or, more accurately, an extensive emigration of these cells, a leucocytosis is present, and it is also equally intelligible how in various toxic diseases a similar leucocytosis occurs without any local inflammatory change. In diseases such as typhoid, malaria, etc., where there is no increase of the finely granular cells, there is, in all probability, an absence of substances which exert positive chemiotaxis. There are, therefore, two chief changes brought into play, viz.: (1) the emigration from the bone-marrow; and (2) increased formation in the bone-marrow. With regard to the condition in which the number of finely granular leucocytes is below the normal, our information is of a less definite character; but the possible factors may, however, be said to be the following:—(1) There may be structural change in the bone-marrow leading to diminished formation of these cells. This is possibly the condition in some varieties of anæmia. (2) In certain diseases, *e.g.* in typhoid, the fall in the number of leucocytes may be due to an increased breaking-down of the leucocytes without a compensatory addition of leucocytes to the blood, which, as we have stated, is brought about by chemiotaxis. (3) In conditions attended usually by leucocytes, *e.g.* pneumonia, septicæmia, etc., the occurrence of a leucopenia may depend upon various circumstances. Excessive emigration into the tissue, an extensive breaking-down of leucocytes in the blood and spleen, a failure of the bone-marrow to keep up the supply, and possibly an accumulation of leucocytes in the various organs in coagula in the heart, etc., may be involved, but it is to be noted that all these factors indicate a condition of gravity. Thus it is intelligible how the replacement of leucocytosis by leucopenia without improvement in the symptoms constitutes a grave omen. It is not, however, the diminution in the number of leucocytes in itself, but the condition bringing it about, which is the important element. On the other hand, the presence and continuance of leucocytosis in the various diseases indicate at least that there is no interference with the natural response to the demand for increased leucocyte supply.

It will be apparent from what we have said that no simple rule of universal application can be given as to the significance of leucocytosis. One must know the conditions of the leucocytes usually found in each disease running a natural course; such information is of importance in diagnosis provided there be no complications. When, however, the diagnosis is established, deviations in the conditions of the leucocytes from that usually present may be of aid in prognosis, and of these deviations the most important is the disappearance of the leucocytosis, or the appearance of leucopenia, without general improvement. We repeat again that mistakes are liable to arise if an application of various hard and fast rules is attempted. It is only by an intelligent consideration of the conditions present, in view of the facts established with regard to the various diseases, that the condition of the leucocytes aids the diagnosis and prognosis. There is no doubt, however, that, employed in this manner, examination into this condition is an important addition to our methods of clinical observation.

**Leucoderma or Leucodermia.** —Defective pigmentation of the skin, congenital (albinism) or acquired (vitiligo), causing white spots, bands, or patches; achromatosis. *See* SKIN DISEASES OF THE TROPICS *(Caused by Climatic Conditions, Leucodermia)*; SKIN, PIGMENTARY AFFECTIONS OF *(Disappearance of Melanin, Leucodermia)*.

**Leucokeratosis Mucosæ Oris.** —The disease of the mouth known also as ichthyosis lingualis, as leukoplakia buccalis, and as buccal psoriasis. *See* TONGUE *(Inflammatory Affections, Leukoplakia)*.

**Leucoma.** — A corneal opacity, milky-white in colour, due to preceding ulceration or inflammation or injury. *See* CORNEA *(Results of Ulcerative Keratitis)*.

**Leucomaines.** — Animal alkaloids formed by metabolic processes during life by the tissues, such as creatin, adenine, spermine, and xanthin; they are contrasted with ptomaines which are developed in dead bodies. *See* ALKALOIDS.

[1] For a further statement of these views see *Brit. Med. Journ.*, 1898, ii. p. 604.

**Leucomelanoderma.** *See* Skin, Parasites (*Tinea Versicolor, Diagnosis*).

**Leucomoria.** — Restless madness or melancholia.

**Leucomyelitis.**—Inflammation of the white matter of the spinal cord.

**Leuconecrosis.**—A form of dry gangrene with a whitish slough.

**Leuconychia.**—White spots or bands on the nails, or the complete whitening of the nail. *See* Nails, Affections of (*White Spots*).

**Leucopathia.**—*See* Skin, Pigmentary Affections of (*Albinism*); Choroid, Iris and Ciliary Body; Leucoderma; Vitiligo; etc.

**Leucopenia.**—A decrease in the leucocytes till their number is below the normal for the peripheral circulation. *See* Blood (*Physiological and Pathological Variations*); Leucocytosis (*Leucopenia*).

**Leucophlegmasia.**—Anasarca, more especially that form of solid œdema which is developed in phlegmasia alba dolens ("white leg").

**Leucorrhœa.**—A white discharge from the female genital organs (uterus, cervix, vagina, or vulva). *See* Chlorosis (*Symptoms*); Curettage, Uterine (*Indications*); Gynæcology, Diagnosis in; Hydropathy (*Chronic Diseases, Douche*); Pregnancy, Physiology (*Changes in Vagina and External Genitals*); Pruritus (*Varieties*); Uterus, Displacements of; Uterus, Inflammations of; Uterus, Non-malignant Tumours of; Vagina, Disorders of (*Inflammatory Affections*); Vulva, Diseases of.

**Leucotrichia.**—Whiteness of the hair; canities. *See* Skin, Pigmentary Affections of (*Pigmentary Anomalies of Hair*).

**Leukæmia.** *See* Leucocythæmia (*Definition*); Hæmatemesis (*Diagnosis*); Liver, Diseases of (*Liver in Leukæmia*); Pregnancy, Intra-uterine Diseases (*Fœtal Dropsy*); Thymus Gland (*Enlargement*); X-Rays.

**Leukanæmia.** — A morbid condition showing the characters both of leukæmia and of severe anæmia (*Leube*).

**Leukokeratosis.** *See* Tongue (*Superficial Glossitis*).

**Leukoplakia.** *See* Syphilis (*Tertiary, Mouth, Tongue*); Tongue (*Superficial Glossitis*).

**Levatores.**—Muscles which raise certain structures or parts (*e.g.* ribs, eyelids, etc.). *See* Eyelids, Affections of (*Affections of Muscles, Levator Palpebræ*); Palate (*Anatomy, Levator Palati*); Physiology, Respiration (*Levatores Costarum*); Pelvis, Perineum and Pelvic Floor (*Lesions of Levatores Ani*).

**Levico.** *See* Arsenic; Balneology (*Austria*); Mineral Waters (*Chalybeate*).

**Levigation.**—The reduction of a drug to a very fine powder by triturating it with a little water and drying the paste thus produced. *See* Prescribing.

**Levinstein's Method.**—The abrupt method of treating morphinomania; immediate and complete withholding of the drug; not often available. *See* Abstinenz-Symptome; Morphinomania and Allied Drug Habits (*Treatment*).

**Levulose.**—Fruit sugar ($C_6H_{12}O_6$); it is lævo-rotatory, *i.e.* rotates a ray of polarised light to the left. *See* Lævulose.

**Levurine.**—A dried extract of beer-yeast, which has been used in cases of sepsis, *e.g.* carbuncle (A. R. Simpson, *Trans. Edin. Obstet. Soc.*, xxv. 91, 1900).

**Leyden Jar.** *See* Electricity (*Static*).

**Leyden's Ataxia.** — A spurious form of locomotor ataxia.

**Leyden's Crystals.** *See* Charcot-Leyden Crystals.

**Leysin.** *See* Therapeutics, Health Resorts (*Switzerland, Canton de Vaud*).

**L.G.B.**—The initials of the Local Government Board, in whose care is the Public Health of England and Wales and of Scotland; in the former country the L.G.B. consists of a President (appointed by the Crown), the Lord President of the Privy Council, all the principal Secretaries of State, the Lord Privy Seal, and the Chancellor of the Exchequer; in Scotland it consists of the Secretary for Scotland (President), the Under-Secretary for Scotland, the Solicitor-General for Scotland, the Vice-President of the L.G.B., and two members (one legal and one medical).

**Lice.** *See* Pediculosis.

## Lichen.

Derivation and Definition of the Term Lichen . . . . . 407
Varieties of . . . . . 407
*Lichen urticatus, strophulus, tropicus, hæmorrhagicus, lividus, pilaris, circumscriptus* . . . . . 407
*Lichen scrofulosorum* . . . . 407
*Lichen planus* . . . . . 408
*Lichen verrucosus moniliformis* . . 408
*Lichen ruber acuminatus* . . . 408
*Pityriasis rubra pilaris* . . . 409
*Lichen ruber neuroticus* . . . 409
*Parakeratosis variegata* . . . 409

PATHOLOGY . . . . . 409
ETIOLOGY . . . . . 410
DIAGNOSIS . . . . . 410
TREATMENT . . . . . 411

*See also* DERMATITIS TRAUMATICA ET VENENATA (*Chronic Dermatitis*); GOUT (*Irregular Gout, Cutaneous System*); HYDROPATHY (*Indications*); PSORIASIS (*Diagnosis*); RHEUMATISM, CHRONIC (*Clinical Features*); SKIN, TUBERCULOSIS OF (*Lichen Scrofulosorum*); SKIN DISEASES OF THE TROPICS (*Prickly Heat or Lichen Tropicus*); SYPHILIS (*Secondary, Skin Affections*); URTICARIA (*Varieties, Urticaria Papulosa or Lichen Urticatus*).

THE derivation of the term lichen is not clear. One can hardly believe that it was selected as an appellation because one of the forms of lichen ruber planus—the sole true lichen among many which have been rejected on various grounds—somewhat resembles the botanical lichens which flourish on the boles of old trees, for this variety in a pronounced guise is rare, and it is not generally from rare varieties that generic designations become popularised. But what is now meant by the name is obvious enough. It is applied to diseases of the skin which are throughout papular, any change in feature being due to alteration in arrangement, or to intensification of existing characters, not to transformation into another type of lesion, or to the fact that the lichenous is but a stage in the course of the ailment. The essential truth of this definition may be averred, even though in very exceptional instances some slight modification has been noted. These are mere chance freaks.

The history of lichen proper does not actually date further back than the time of Hebra, and the conception he formed is adhered to now even more closely than by its originator himself. But there are many cutaneous complaints to which the prefix lichen was connected at no distant period, now relegated to other categories, yet which require brief notice here in order to clear the ground. Thus we have *lichen urticatus*, a form of chronic urticaria seen particularly in young children, in which papules evolve out of and succeed the wheals. *Lichen strophulus*, a punctiform eruption of small, acuminate red papules in infants, associated with profuse sweating, favoured by or perhaps even due to unsuitable or to coarse woollen underclothing. *Lichen tropicus*, also a sweat rash, seen chiefly in warm climates, and there most frequently in new-comers, and in one of its types papular. It is caused by sudden blocking of the mouth of the sudoriferous ducts, with cystic degeneration as a sequence. Pollitzer explains its production as due to the soaking, by perspiration, of a skin insufficiently supplied with fat. It may be that depriving the integument of its normal unctuousness by too frequent baths with soap, predisposes, since it does not appear to occur in the negro. *Lichen hæmorrhagicus* and *lividus* are mere accidents in some papular efflorescences, when in consequence of intense congestion blood is effused into the tissues, or owing to a scorbutic or purpuric element it oozes into the lesion. *Lichen palaris*, though papular, dry, and permanent, is properly a keratosis of the upper half of the hair follicles. It gives rise to the rough scaly points so frequently seen and felt on the outer aspects of the upper arms and thighs; or to the spiky prominences —called also lichen spinulosus—inflammatory in nature, met with on the neck, arms, and elsewhere, set with almost mathematical regularity, and exhibiting, occasionally at least, contagious features, where the extruded root-sheaths form short projections. *Lichen circumscriptus* must now be looked on as an extension of seborrhœa, and as constituting one of the varieties of Unna's seborrhœic dermatitis. It occurs on the back between the scapulæ, or on the front of the chest, and is particularly prone to arise in the case of those who habitually wear thick though possibly soft flannel under-vests. Individually the elements are minute red or rose-red pinhead-sized spots or elevations, which are follicular in situation. These have a tendency while extending to arrange themselves as incomplete circles or crescents, the included area being fawn-coloured, perhaps slightly scaly. The periphery is a rose-red line, which in many instances can be resolved into a chain of perifollicular papules. But occasionally the whole area is rough, the projections being closely set over the entire field. Considerable, even wide tracts may be involved, the skin in general is greasy, and there is usually seborrhœa capitis, the starting-point, as it is the maintaining source of the disease. The arrangement of the dorsal manifestation is triangular with the apex downwards.

*Lichen scrofulosorum* occupies as yet an uncertain position. Though in the large majority of instances it is encountered in children and young adults who are evidently of the scrofulous type, it has not yet been satisfactorily proved to be tubercular by the unequivocal discovery of the bacillus. It is an inflammatory process which has its seat in the immediate neighbourhood of the pilo-sebaceous follicles. It runs a chronic course, and its pinhead-sized papules are flattened and soft, of a pale red, brownish red, or whitish tint. These are found in groups, or arranged in circular lines; they bear a small scale on their summit, less often a tiny pustule; after a rather prolonged duration they disappear. They are seen both on the front and back of the trunk, but may be complicated by an impetiginous eczema of the pubic regions and their neighbourhood, and give rise to a slight sensation of itchiness.

The sole true lichen which remains is *lichen*

*planus*. Its lesions occur in two forms—as isolated papules, or when these become clustered into the patch, in which circumstances they undergo some modifications. The papules may be polygonal or round, pale, almost skin colour, or more typically crimson or bluish red. Their surface is hard and smooth, so as to impart a burnished appearance when viewed by oblique illumination. They may remain discrete, dispose themselves in lines, or from progressive increase in number may be so aggregated as to form patches. In both cases on subsidence they leave behind a degree of brown pigmentation, sometimes an atrophic depression, or the surface of the patch grows rugose and warty. A peculiarity of lichen planus is polymorphism within certain limits, displayed by the papules themselves and in their mode of coalescence. Thus the colour varies from a pale to a crimson-red in the angular, in the round more usually a bluish red, on the legs a dull purplish. The shape is determined partly by the surface lines of the skin, partly also by the situation; thus they are flatter when they form round a sweat gland, more acuminate at a hair follicle. At times the papule assumes an obtuse shape, having a convex rather than a plane summit; on the lower limbs especially their contour may be oval. Some exhibit a punctate depression in their centre, more on stretching show fine whitish lines within their structure. Though scaliness is not a feature of the isolated papule, it becomes pronounced on the patch, and this hyperkeratosis takes on even extreme proportions on the legs, where not infrequently a hard, grey, warty, veritably lichenous investment may be observed (lichen verrucosus). The patch, however, may on the contrary be fairly smooth; indeed, in some instances it much resembles a dry erythematous blotch, only it does not wholly fade on pressure. At other times it may show a species of cross-hatching, due to the approximation of angular papules. It is customary to find isolated papules in the neighbourhood of a patch. Though the linear arrangement is the ordinary, a circular is not unknown. The elevation varies; on the forearms it is not great, is more evident on the back of the hand, and often considerable on the lower extremities, particularly about the shins. The pigmentation, too, differs. In some it is residual, not noticeable till the papule has been absorbed; in others there is decided dark staining round a patch, or even a papule, and this where no arsenic has been administered.

The situations affected are to some extent characteristic. Thus the face and scalp are avoided, while the wrists and flexor aspects of the arm, the inner side of the thighs, and the front of the leg are favoured. It may be limited to the scrotum, or to the penis or its immediate vicinity; but it occurs on any part of the trunk, pre-eminently where articles of dress press on the skin, as at the waist or where garters are worn. On the palms the papules are horny and may be smooth, but often by tearing through the dense epidermis cause it to look ragged. Parts of the palm may thus present a dry, cracked appearance. The nails seem never to be involved.

On the tongue and buccal mucous membrane the tenderness of the investing layer and the moisture alters the aspect. Hence in place of definite papules there are milky-white spots or patches. Occasionally unexplained diarrhœa arises in course of lichen planus, and it has been surmised that this may be due to the eruption of lesions in some part of the intestinal tract. Nevertheless the general health seldom suffers.

So few cases of *lichen moniliformis* described by Kaposi and von During have yet been encountered that its exact nature and its relationship to ordinary lichen planus are undecided. Still, as papules such as those described and pigmentary macules, the result of their involution, were also perceptible, it would appear that this is but an extreme variant. The papules are fused into lines or elevated ridges, longitudinal in direction, and found chiefly on the throat or neck and flexor aspects of the limbs. The prominences so produced resembled strings of coral beads or elongated nodosities of keloid. The surface was glossy, brownish red in colour, and rather tender to pressure.

Lichen planus may persist in a localised form, the chronic or commonest type, or advance slowly and erratically; but in some cases it pursues an acute course, invading the greater part of the covered portion of the body in a short time, and appearing as a symmetrical eruption. The papules are a more decided red, but are otherwise like those in the chronic variety. Itchiness may precede the formation of the papules. Its degree varies; it may be trivial, or so intense as to preclude sleep. Both papules and patches are numerous, diffused over a wide area, and there is more distinct scaling, while pigmentation always supervenes. Though acute in its onset it may only leisurely fade. Recurrences may occur more than once. Whether treated or not the tendency is to disappear after a time, and this in the chronic and acute variety alike. One may with truth say that recovery is constant, but its date indefinite. The verrucous kind, whose seat *par excellence* is on the lower limbs, is peculiarly obstinate and offers marked resistance to treatment.

While interspersed among the flat papules we may find some more or less acuminate; yet the disease described by Hebra as *lichen ruber acuminatus*, in which all the papules were pointed and the termination mostly a fatal one, unless arsenic were administered, has not so far been satisfactorily identified. Two views are

held with regard to this. One, that Hebra confused with lichen planus a disease particularised by Devergie and known as *pityriasis rubra pilaris*. It is true that Hebra did not formulate an account of this complaint in separate form, but it is probably an error to hold that this ailment comprises all cases of Hebra's lichen ruber acuminatus. There are many circumstances which render this idea unlikely. The name conveys a good conception of the general features. Thus the fine, dry, white scales, so abundant in some cases, make good the pityriasis; beneath these is found a substratum of diffuse and unusual redness; while pilaris indicates that the hair system is chiefly implicated. It may commence by the formation of dry patches on the palms or soles, or with flaky seborrhœa of the scalp or face. But in other examples the peculiar papules may appear primarily on the trunk or limbs. These are conical, from a pin-head to a hemp-seed in size, red, hard, and arid, showing a broken hair in the centre, surrounded by a species of horny collar dipping down into the follicle. They are, indeed, like lichen spinulosis, more widely distributed, but set with great regularity at intervals very nearly exact. With an increase in number they become crowded together so as to lose their obvious individuality. The skin then feels thickened and immobile, looks reddish or yellowish, and is covered with an investment of scales, either fine and branny, or massed into the semblance of a coating of plaster or lime, but without trace of moisture or oozing. As a rule, isolated papules may be discovered at the edges of the thickened areas, but the eruption may be so generalised that the papular element is wholly or all but wholly concealed. Should the epidermic accumulation be removed by oil-packing, then dull brownish-red papules, not altogether unlike those in lichen planus, are disclosed. A special characteristic is the occurrence of papules corresponding to the hairs on the dorsum of the first and second phalanges of the fingers. The nails are usually attacked, are greyish and striated, while beneath them a soft concretion forms like rush pith. The face is often covered with minute scales and seborrhœic accretions; the integument is dry and stretched, giving rise to ectropion. The course of the disease is subacute or chronic, with no evident constitutional symptoms, the general health being well preserved throughout. It may last for years, with temporary aggravations and remissions.

But Unna and von During have met with cases, especially in an epidemic which occurred in Hamburg, which they think approximate more closely to Hebra's conception of lichen ruber. They state that it may attack persons apparently in good health; more commonly it is ushered in by feverishness, headache, and depression. Locally there is an erythroderma at first limited and transitory, later spreading widely. Then small, red, hard, conical, glancing papules, which are chiefly seated at hairs, but may occur apart from these, develop. Many bear a scale. They may mass themselves into patches, with infiltration of the skin and pigmentation. The nails are seldom affected, but the hair falls off. The itching is intense; there is sleeplessness, loss of appetite, emaciation, and weakness. The disease is a severe one, and has ended fatally. Reviewing the question, Brooke thinks that there may be from time to time outbursts of this *lichen neuroticus*, as Unna terms it, and Hebra drew his picture either from one of these, or he confused the three ailments together, working as he did in the early days of dermatology.

There is still, however, another rare disease which has so far not been exactly allocated. To it Unna has provisionally attached the name of *Parakeratosis variegata*, and several instances have come under my notice. The disease is an eminently chronic one, and may last very many years. There are at first minute papules, very little elevated above the surface, which arrange themselves in lines. In colour they are a dull crimson red, but have little if any of the burnish of those of lichen planus. Gradually they arrange themselves so as to form a kind of meshwork, so that the skin shows a mottled appearance, white spaces enclosed by crimson-red boundaries. There may be slight desquamation. All the body, face included, becomes affected, and the mottling gets less pronounced as the white areas grow redder, till the surface is of a patchy plum colour. The skin becomes thinned, and the hairs wax scantier and scantier everywhere. Itchiness is present in some, not complained of in others; but there is great chilliness. In one case after a duration of very many years soft excrescences or tumours formed here and there over the body, in appearance not unlike those of mycosis fungoides. Some of these broke down into spongy ulcers, secreting a serous fluid, which only slowly healed; others, after persisting for a time, were absorbed. Though the patient was not capable of much exertion, his health was pretty good and his mind unclouded. It occurs both in males and females, and commences in adult life. One cannot yet speak definitely of its termination. Like lichen planus it seems not to attack the nails, but unlike it it involves the face and scalp.

Pathology.—In relation to the pathology of *lichen planus* no micro-organism has so far been held responsible for its causation. The special changes are superficial, involving the epithelial layers, and mainly the papillary portion of the corium. As already stated, the shape assumed depends very much on whether the sweat glands, hair follicles, or the general tissue of the integument are principally or wholly implicated. The morbid changes have been shown by Török to begin in the neighbourhood of the blood-vessels

in the papillæ, of which œdema and cellular infiltration are an evidence. The œdema is a solid one which flattens the papillæ and forces them out of shape. The burnish on the surface of the papules has been explained by Unna as due to tension arising from the packing of the upper part of the corium with cells and the coexistent swelling; but, as Brooke points out, there is also a modification of keratinisation, since the polish is an early symptom, and persists even when the papules have somewhat levelled down and distension from that cause is reduced. The cells which crowd the tissues seem not to be in the main leucocytes, but are chiefly derived from proliferation of the connective tissue cells, and Walker regards them as similar in nature to those found in the granulomata. The atrophy which in some cases succeeds the subsidence of lichen may be quoted in support of this view. Pigment cells are met with in the walls of the vessels before the disease has lasted long. The white plugs visible in the centre of some of the papules are due to alterations and thickening of the sweat pore, with loosening and separation, which gives rise to the depression. The white lines and network seen in their structure arise from excessive development of portions of the granular layer, which betrays itself by an opacity like ground glass. There is always increased thickness of the corneous layer, and this acquires extreme proportions in the verrucous form. As retrogression proceeds there are degenerative changes, shown by a colloid transformation and the increasing deposit of pigment.

Quite different is the morbid anatomy of *pityriasis rubra pilaris*. Here the horny follicular papules are but part of a general hyperkeratosis. Indeed, Unna holds that in its main features it approximates most closely to ichthyosis, and when one remembers that the slighter evidences of ichthyosis are expressed in keratosis pilaris, there is good ground for the comparison. The augmented keratinisation increases the surface area of the skin, and thus it is thrown into folds of a coarse type; but there is but little infiltration into the papillæ, which themselves are not swollen. The characteristic papules are produced by the advance of the hyperkeratosis into the follicular neck, but it extends into the deeper parts as well. Reactionary changes in the neighbourhood are indicated by a degree of local leucocytosis and interepithelial œdema.

*Parakeratosis variegata* is, as one would expect, closer to lichen pathologically. My own observations agree with those of Santi and Pollitzer that the affection is limited to the epidermis and upper layers of the corium. There is moderate dilatation of the vessels of the papillæ and some œdema, but the inflammatory phenomena proper are slight. There is interstitial œdema with dilatation of the lymph spaces in the prickle layer. The horny layer is redundant and stretched, hence the degree of burnish. The appearances in sections from one of my cases led a skilled observer, who was ignorant of the source, to say that they were from a case of lichen planus. There is therefore nothing surprising in the later development of granulomatous tumours.

Etiology.—Little definite can be said as to the etiology. It is true that some of those affected are of the class termed neurotic, or have been the subjects of worry or vexation; but on the other hand it is fairly common in well-nourished women about middle life, who are leading a placid and comfortable existence. It occurs about equally in either sex, may be met with in children, though rarely, and much the same may be said as to its appearance at the other extreme of life. It is certainly of more frequent incidence in the better ranks of society; but it is found also in those who frequent hospitals, who are, however, by no means necessarily, in Scotland at least, drawn exclusively from the lower strata. It must be admitted, and this applies to pityriasis rubra pilaris and to parakeratosis variegata, that we are absolutely in the dark as to any determinate cause; an organismal origin is yet wholly hypothetical.

Diagnosis.—The peculiar and characteristic features already described must be mainly relied on. Though Hutchinson considers it as nearly related to psoriasis, and mistakes in this direction are not very infrequent among those, at least, who are not very familiar with a somewhat uncommon ailment, still there are essential distinctions. The primary spot in psoriasis is always scaly from the outset, while on enlargement the area is uniformly so, or shows central involution, while the colour is different, and the itchiness seldom so marked as in lichen. The papular variety of eczema is somewhat like it, but the papules are more plainly red and are not glistening. They become lost in the eczematous patch, and other vesicular, oozing, crusted, or pustular forms are or have been present. Syphilis, however, does provide a fairly close imitation, yet the small papular syphilide has a more coppery tint, is more widely distributed, rarely forms patches, does not avoid the face, is often mixed up with other types of eruption, and general constitutional symptoms are discoverable as a means of discrimination. The peculiar mottling with progression from above downwards, the slow course with intractability to treatment, aid in excluding parakeratosis variegata, and it is only when the encrustations have been removed by oil-packing or inunction that pityriasis rubra pilaris recalls lichen planus, and then it is the rare lichen neuroticus that is simulated, hardly the ordinary.

The prognosis in lichen is good; at most the disease is obstinate, and from the concurrent

pruritus annoying, but ultimate recovery is the all but invariable rule.

TREATMENT.—In relation to treatment, lichen planus is undoubtedly capricious. The generalised form yields much more readily than that occupying restricted areas. The more rapidly it extends the more speedily does it usually disappear, though months may be passed ere it finally vanishes. The warty forms on the lower limbs are very obstinate. To get rid of it we must avail ourselves both of constitutional and of local remedies. Arsenic internally probably takes the first rank; it may be given either in pill or in solution, and moderate doses should be persevered in for perhaps several months ere it is abandoned as unsatisfactory. Should the itching be severe, strychnia may be combined with it; and if anæmia coexist, a not very frequent circumstance, iron may be added. If arsenic fail or seem but tardily effectual, antimony, as recommended by Hutchinson, is often an efficient substitute. From eight to thirty minims of the vinum antimoniale well diluted are to be taken three, four, or even for a period six times a day. It commonly agrees perfectly, and only if it occasions sickness plainly ascribable to it, or diarrhœa, should it be discontinued or the dose lessened. If both are unsuccessful we may employ mercury, which in the form of the perchloride has caused the disappearance of the disease. From the thirty-second to the sixteenth of a grain twice a day with a grain and a half of iodide of potassium gives the best results, any trace of salivation being watched for, and obviated by temporary disuse and subsequent reduction of the dose. Washing with menthol soap, or baths of Condy's fluid, an ounce in a large bath at 90° to 95°, are useful for relieving the pruritus. Or the following lotion of C. Boeck's may be freely used: ℞ Talci, pulv. amyli, liq. plumbi subacetat. dil., sol. acidi borici in aqua 1 per cent āā 100·0, glycerini 40·0. When used, this must be diluted with twice as much cold water and painted on. In chronic, and especially in localised cases, coal tar dissolved in acetone, as recommended by Sack, is advantageous. ℞ Picis carbonis 10·0, benzol 20·0, acetone 77·0. M. In mild cases or in acute forms a cleanly and valuable application is: ℞ Acidi borici grs. 15, glycerini amyli (1 in 16) unciam. In pityriasis rubra pilaris, baths of carbonate of soda, two ounces in thirty gallons of water at 95°, followed by inunction with vaseline, and combined with the subcutaneous injection of pilocarpine, are indicated. For parakeratosis variegata no treatment has so far influenced the disease.

**Lichenification.**—The thickening and hardening of the skin which follows upon repeated scratching of a patch of pruritus; it may be a primary change, but more often it supervenes upon a skin already altered by eczema or lichen planus (*Brocq*). *See* PRURITUS (*Causation of the Pruritic State, Traumatides*).

**Lichens.** *See* DIET (*Vegetable Foods, Lichens*).

**Lidja.** *See* BALNEOLOGY (*Turkey, Anatolia*).

**Lie.** — The presentation of the fœtus in labour, or the relation of its long axis to the long axis of the uterus of the mother (*e.g.* the two long axes may coincide, as in the cephalic and pelvic lies; or they may not, as in the transverse or shoulder cases). *See* LABOUR, DIAGNOSIS AND MECHANISM (*Lie and Presentation*).

**Lieberkühn's Follicles.** *See* PHYSIOLOGY, FOOD AND DIGESTION (*Structure of the Small Intestine*); INTESTINES, DISEASES OF (*Anatomical Considerations*).

**Liebermann's Test.**—A test for proteids in which the substance is washed with alcohol and ether and treated with fuming hydrochloric acid, when a violet-blue colour is produced if proteid be present.

**Liebig's Extract.** — A beef extract. *See* INVALID FEEDING (*Meat Broths* and *Extracts or Teas*).

**Lienculus.**—An accessory spleen or lien accessorius. *See* SPLEEN.

**Lienitis.**—Inflammation of the spleen; splenitis. *See* SPLEEN.

**Lienocele.**—Hernia of the spleen. *See* SPLEEN.

**Lienomalacia.** — A morbid state of softness of the spleen; *lienomyelomalacia* is a softening of both the spleen and the bone-marrow.

**Lientery.**—Diarrhœa in which soon after its ingestion the food passes undigested through the alimentary canal; hence *lienteric diarrhœa* (Gr. λεῖος, smooth, and ἔντερον, intestine). *See* CONSTIPATION (*Physiological Considerations, Exaggeration of Peristalsis*).

**Liernur System.**—A non-gravitation method of removal of sewage in which suction is employed; it is in use in St. Petersburg, Amsterdam, Riga, and other places; the action of a vacuum draws the excreta and slop waters to a central pumping station.

**Life, Change of.**—The menopause. *See* MENSTRUATION; MENOPAUSE; CLIMACTERIC INSANITY; etc.

**Life Insurance.**

HISTORICAL . . . . . 412
DUTIES OF MEDICAL MAN . . . 413
*Family History of Proposer* . . 414
*Past History of Proposer* . . . 415

*Present Health*. . . . . 415
*Female Lives* . . . . . 419
*Habits and Occupation* . . . 420
*Place of Residence* . . . . 420
*Age*. . . . . 421
TABLES OF EXPECTATION OF LIFE . . 421

*See also* NEPHRITIS (*General Diagnosis*); SYPHILIS (*Relation to Life Insurance*); VITAL STATISTICS (*Life Tables*).

INSURANCE has been defined as "a contract whereby one party, in consideration of a stipulated sum, undertakes to indemnify the other against certain perils or risks to which he is exposed, or against the happening of some event." (From Marshall on *Marine Insurance*.)

It is difficult saying when insurance had its origin, but it is known that it was in use in commerce in the fifteenth century, because an ordinance of Barcelona refers to a contract of insurance. In all probability, insurance was a common practice in commerce before any laws on the subject were recognised. Marine insurance was one of the earliest branches, and was probably invented by the Jews and adopted by the Lombards. Life insurance was only a branch of marine insurance.

Life insurance was well known in the sixteenth century. At Genoa, in 1588, wager policies and insurances on the lives of public men were absolutely prohibited without the leave of the Senate.

All life assurance was prohibited by Philip II. in 1570, and his example was followed by other cities and states. The practice of insuring the lives of other people was soon recognised to be a public danger. Grivel remarks: "These kind of wagers are of sad augury and may occasion crimes." In 1753 Magens says: "Men insured freely. In London people take the liberty to make insurances on any one's life without exception, and the insurers seldom inquire much if there are good or bad reasons for such an insurance, but only what the person's age is and whether he be of a good constitution or not. The common premium on a good life from twenty to fifty years of age is 5 per cent, and from fifty to sixty years 6 per cent."

This insuring of other men's lives became such a crying evil in England that the famous Act of 1774 was passed, prohibiting all insurances on lives in which the person insuring had no interest. (Act 14 Geo. III. c. 48.)

The first insurance company was established in England in 1706 by a charter of Queen Anne to Thomas Allan, the Bishop of Oxford, and others, and was named The Amicable. Each member paid a fixed annual sum, and the surplus at the end of the year was divided amongst the relatives of the deceased members. All were admitted at a uniform rate, and members' ages on admission ranged from twelve to forty-five. The Royal Exchange and London Assurance were empowered to carry on life insurance in 1720. The Equitable was established in 1762, the Westminster in 1792, and the Pelican in 1797.

Insurance business has grown immensely during the nineteenth century. There were 8 companies in existence in England before 1800. In 1824 there were 39; 105 companies were added between 1824 and 1844; 272 new companies were established between 1844 and 1869. This rapid growth was partly due to the new impulse given to the starting of new companies by the repeal of the Bubble Act in 1825, and the passing of the Companies Acts of 1844 and 1862.

Many of these companies have ceased to exist. In 1880, of the 39 companies established before 1824 all but one survived. Of the 105 established between 1824 and 1844, 38 had ceased to exist; and of the 272 established between 1844 and 1869, only 29 survived.

It will be seen from the short history of life insurance just sketched that insurance in the eighteenth century could hardly be said to have been based on a scientific footing. From the quotation from Magens it will be seen that the rate of 5 per cent was put on lives from twenty to fifty, and 6 per cent from fifty to sixty. This haphazard way of imposing rates of premium resulted from a want of knowledge of the rates of mortality amongst individuals of different ages.

At that time little was known of what is called expectation of life, although it is true that the term "expectation of life" was used by De Moivre in the year 1725.

Simpson was, in 1752, the first to arrange a table of expectations, but any tables previous to the Northampton seem to have been based on hypothesis rather than on statistics. The Northampton table of mortality was first given in a work on annuity by Dr. Price in 1771, and for many years that table was used by many of the insurance companies. What is known as the Carlisle table was the result of observations in two of the parishes of Carlisle, and published by Dr. Haysham in 1797, which observations were further elaborated by Mr. Milne. This table was for many years recognised as the most accurate. In 1843 tables giving the results of the experiences of seventeen of the insurance companies were compiled, and these were found to more nearly resemble the Carlisle than the Northampton tables. The total number of policies made use of in the compilation of these tables was 83,905, of which 44,877 were in existence, 25,247 withdrawn, and 13,781 had become claims by death. The most striking results obtained were—1st, the great mortality amongst Irish lives; 2nd, the marked difference in rate of mortality between males and females; 3rd, the near resemblance between town and country experience. The

mortality amongst insured females was greater than amongst males.

In 1869 a still further advance was made in the statistics of mortality and "expectation of life." A table was compiled by the Institute of Actuaries, which is sometimes called The New Expectation table,[1] sometimes the Institute of Actuaries' table. Twenty offices aided in the compiling of this table. The data were ultimately published in four great divisions—1st, healthy lives male; 2nd, healthy lives female; 3rd, diseased lives, male and female; 4th, lives exposed to extra risk from climate, occupation, etc.

The assurance companies at first made very little attempt to select healthy lives; at least no medical examination of the applicant was made, nothing more than an inquiry as to whether he was in good health. A medical man at first was not recognised as necessary. As recently as 1815, according to the form of the Scottish Widows' Fund Society, all that was necessary was that the applicant should appear before a medical man, who certified that he was apparently in good health, and that he had never suffered from gout, asthma, or any other disease which shortened life.

About 1830 this society required a certificate from the medical attendant of the applicant, and a series of questions about his health and habits had to be answered. About the same time the office appointed their own medical adviser, who helped the manager and directors to select the lives. The society also required certificates as to health and character from private friends of the applicant. In 1835 all applicants had to appear before the medical adviser of the company as well as to have a series of questions answered by their own medical attendant. The agent and two friends had also to give a report on the life (Muirhead).

This is practically the method of selection adopted by most of the best insurance companies at the present day, with the exception that it is only in cases where a report from the medical attendant of the applicant may throw more light on his family and previous history, that a special report from the medical attendant is called for.

It seems to be pretty generally recognised that the insurance companies have in The New Experience tables a fairly accurate estimate of the expectation of life of healthy persons at different ages, and that the premiums that the insured have to pay are fair both to the insurer and the insured, when the insured is what is called an "average life."

[1] The tables for convenience are designated in a particular way, H for healthy lives, D for diseased lives, with a smaller letter above to denote the sex: thus, $H^M$, healthy lives male; $H^F$, healthy lives female; $H^{MF}$, healthy lives male and female. The $H^{MF}$, the committee say, may be fairly considered a standard table for life assurance.

But when under-average lives come to be considered, the problem as to whether such lives ought to be "loaded" with an extra premium, and how much "load" is to be put on, is one of great difficulty, and one which has given rise to much discussion. Some have maintained that the benefits of a medical examination of the applicants are lost in a very few years. It is unnecessary to enter into all the arguments which have been used in favour of this contention; but it has been as strongly maintained, on the other hand, that although much of the benefit is lost after the first few years, still the influence of selection is felt even in the older policies.

The task before a medical examiner for an insurance company is, therefore, to determine whether the life of the applicant for insurance is an average healthy life having average expectation of life, or an under-average life having an expectation of life below the average—and if the latter, whether the life can be "loaded" to such an extent that such loading would be fair both to the insured and the insurer.

Statistics of under-average lives have, of course, been prepared, but much yet requires to be done in order to accurately determine what amount of "loading" is necessary in any particular case. If a case of valvular disease of the heart be taken as an example, the question might be asked—Is the expectation of life of such an individual lower than that of one with no valvular disease; and if so, how much? Most medical men will agree with Sir Wm. Gairdner in the opinion that a large number of cases of valvular disease live to a good old age, and far beyond what one would expect. Actuaries say that this does not affect the general fact that cases of valvular disease, when taken altogether, have on the average a lower expectation of life, and they point out that expectation of life does not mean the probable duration of life of each individual, but the average duration of life of a large number of individuals. This, of course, is quite true; but the problem before medical men is to determine what are the factors in each case which make a life an average one or an under-average one. The advance of medical science added to the experience of insurance companies will probably help us to solve these difficulties, and enable us to arrive at a more accurate estimate of the amount of loading required in particular cases.

In determining whether an applicant is eligible for assurance, and at what rate of premium, there are certain factors which the medical advisers of the company have to consider and give due importance to. These factors may be classed under different heads—1st, the family history of the proposer; 2nd, the past history of the proposer; 3rd, his present state of health; 4th, his habits and

occupation; 5th, his place of residence; 6th, his age.

The lists of questions which have been drawn out by the various insurance companies to be put to the applicant, to their agent, to the applicant's personal friends, and to the medical examiner, are intended to elicit all necessary information on these points, and the medical adviser of the company ought to have all such information before him at the time of his giving his opinion as to how a life is to be classed. Insurance companies vary as to the classification of lives, but all lives can be conveniently classed under three groups:—1st: (*a*) Lives probably above the average insurable at ordinary rates; (*b*) average lives insurable at ordinary rates. 2nd: Under-average lives insurable with a certain amount of "loading." 3rd: Under-average lives not insurable.

To determine in which group a life is to be classed all the factors before mentioned have to be considered and weighed.

1. *Family History.*—Although at the present day the belief in the hereditary transmission of disease is not by any means so general among the medical profession as it used to be, still few will dispute the fact that some families are liable to certain diseases, and if the diseases themselves are not hereditary the liability to these diseases runs in families. Consumption, which was considered one of the most hereditary of all diseases, is now believed not to be transmitted at all from parent to child—yet it can hardly be denied that consumption runs in families. Although, therefore, opinion has changed as to why certain diseases are more prevalent in some families than others, there can be no denying the fact that they are, and it is therefore necessary for insurance companies to inquire into the family history of the applicant for insurance. It is necessary, in the first place, to ascertain whether the applicant's father and mother are alive or dead. If alive, in what state of health and what their ages are; and if dead, at what ages they died and what was the cause of death. The number, state of health, and ages of the proposer's brothers and sisters should also be ascertained; and how many, if any, are dead, their ages at death, and causes of death. In many cases, of course, the family history is quite satisfactory, but in some there are facts which at once arrest attention. The family may be all short or long lived, because there can be little doubt that some families seem to have a greater tenacity of life than others. If all or several members of the family have been short-lived, the causes of death will probably indicate what disease or diseases the family is liable to. If, for instance, two or more members of a family of six or seven had died or suffered from tuberculosis, one would be suspicious that the family had a tendency to contract that disease, and it would be advisable to make further inquiries as to tubercular disease amongst the more distant relations of the proposer, such as the uncles, aunts, cousins, grandfather, and grandmother. This is more especially necessary where the proposer's immediate relatives are few in number. The prevalence of any particular disease in the family should also be followed by a searching examination of the individual, especially as regards the particular organ or organs liable to be affected by that malady, to ascertain whether the proposer is free from disease, and is constituted in such a way as not to be likely to develop it. In the case of consumption, for instance, particular attention should be paid to the form, movements, and development of the chest, as well as to whether the lungs are healthy. The habits of the individual as well as his occupation would here also be of considerable importance in determining whether he was likely to develop the disease. The age of the proposer in a case of this sort is of great importance. If young and under the age at which his relations died, his life is not so good a one as if he had passed middle life, or had well passed the age at which the relatives died. This, of course, applies to consumption; but each disease has to be specially considered, as it is well known that the age at which different diseases manifest themselves varies greatly. Whilst, for instance, pulmonary phthisis is most prevalent from eighteen to thirty, gout, cancer, insanity, paralysis, etc., are more apt to prove fatal in later life. The bearing of these facts on the expectation of life of an applicant for insurance who has a family history of those diseases, is self-evident.

But the question for an insurance examiner is whether an individual case before him with its own particular family history should be admitted, and at what rate, or rejected. Can any rule be laid down as to what constitutes a family history showing a tendency to a particular disease? In answering this question, much, I think, must depend on the disease which is under consideration. The mere presence of a case of consumption, a case of gout, a case of paralysis in some near relative of the proposer, can hardly be taken as showing a tendency to any of those diseases, and yet if any of the near relatives have suffered from any of the so-called hereditary diseases the medical examiner must necessarily be on the look-out for evidences of the same or allied diseases in the proposer. Dr. James Begbie, in his reports to the Scottish Widows' Fund Society, was the first, I believe, to lay down the rule that the presence of two undoubted cases of consumption in near relatives of an applicant for insurance should be an absolute bar to his being admitted. This view was later very strongly advocated by the late Sir Robert Christison in his report to the Standard Insur-

ance Company, and later still by the late Dr. Warburton Begbie, and probably most medical examiners at the present day would adhere more or less to this view, especially where the age of the proposer is under thirty years. It might, however, become a question for discussion whether such a life might not be accepted at least with an extra premium, if he himself was in good health and over thirty years of age. The farther he had passed thirty the less risk there would be to the insurance company.

Ought the same rule to apply to gout, rheumatism, cancer, insanity, paralysis, etc.? Most medical examiners would probably not go so far as this. They would probably seek for some evidences of these or associated diseased conditions in the proposer himself before rejecting him altogether, or even recommending his acceptance at an increased rate. Each disease has, therefore, to be considered separately, and the points to be considered in regard to it are:—1st: Its liability to manifest itself in successive generations. 2nd: The age at which it is most likely to appear. 3rd: The effect the disease or constitution has on expectation of life. 4th: The kind of insurance proposed—whether endowment or whole life. These will be best considered later in connection with the particular diseases.

2. *Previous History of the Proposer.*—The previous history of the proposer often gives much information bearing on the question of his expectation of life to the medical examiner and to the insurance company. His past history may indicate what is his constitutional diathesis, and also, whether he has had any disease that is likely to have left him weak or more liable to the onset of other diseases. For instance, a history of acute rheumatism in the early life of the proposer would indicate, not merely the existence of the rheumatic diathesis, but would also clearly call for a special examination of the condition of the heart. Similarly with scarlet fever, an examiner should naturally specially look for sequelæ of that disease, such as enlarged glands, otorrhœa, cardiac disease, and kidney mischief. The previous history of the proposer is therefore more of importance as a guide to the examiner where to specially examine for any weakness which may have developed as a result of past illness, than as an indication as to whether the proposer is to be admitted or rejected, because I take it that there are very few cases where the past history alone would cause the rejection of the life if no trace or result of the past illness was found at the time of the medical examination.

3. *Present State of Health of the Proposer.*—The state of health of the proposer at the date of his examination must necessarily be the most important of the factors in determining whether the proposer is a healthy life or not. A careful examination must therefore be made of all his organs with the object of finding out not only how they are at present performing their functions, but whether there is any trace of abnormality or defects produced by previous illness or habits. Mere questioning of the individual is not sufficient, as he may not be aware of some very important weakness in his organisation, such as the presence of organic heart or kidney disease. The general appearance often gives very important information as to his state of health. Such general appearance when taken along with the family history may indicate whether the life is a good one or not; even such details as the complexion, and whether the proposer resembles more closely his father or his mother, being of importance. The weight of the proposer should always be noted and compared with the height. When a person is much over weight his expectation of life is not so good as that of a person of about normal weight. In the same way a person under weight, and especially markedly under weight, is either already affected with, or is in a condition in which he is more liable to the onset of disease. The family history, the previous health, and the constitutional diathesis of the individual, should all be considered along with the weight. Under-weight, for instance, may show a tendency to tubercular disease; whilst over-weight may point to a tendency to gout, to stomach and liver troubles, and also may give some indication of the habits of the individual. The following table of Hutchinson's gives the average relation between the height and weight of an adult man:—

| Height | | | | | Weight | | |
|---|---|---|---|---|---|---|---|
| 5 | feet | 1 | inch. | | 120 | lbs. | |
| 5 | ,, | 2 | inches. | | 126 | ,, | |
| 5 | ,, | 3 | ,, | | 133 | ,, | |
| 5 | ,, | 4 | ,, | | 139 | ,, | |
| 5 | ,, | 5 | ,, | | 142 | ,, | |
| 5 | ,, | 6 | ,, | | 145 | ,, | |
| 5 | ,, | 7 | ,, | | 148 | ,, | |
| 5 | ,, | 8 | ,, | | 155 | ,, | |
| 5 | ,, | 9 | ,, | | 162 | ,, | |
| 5 | ,, | 10 | ,, | | 168 | ,, | |
| 5 | ,, | 11 | ,, | | 174 | ,, | |
| 6 | ,, | | | | 178 | ,, | |

An approximate method of arriving at what the weight of a person ought to be, is to take the cube of his height in inches and divide by 2000; the result is what the weight ought to be in pounds. For instance, a person of six feet by this method ought to be 186 lbs. in weight. One-seventh either above or below this may be quite consistent with health (Maclagan).

Rapid changes in weight should always be looked upon with suspicion.

Deformities, such as spinal curvature or other changes indicating diseases of bone, should be specially noted, and particular attention paid to whether the disease which caused such

deformities was still active or quiescent. Whether the proposer has been vaccinated and revaccinated, or had smallpox, are important points.

*Respiratory System.*—Probably diseases of the respiratory system are responsible for more of the deaths of insured persons than those of any other system in the body. Of the diseases of this system the most important are phthisis, bronchitis, asthma, pneumonia, and pleurisy. A person actually suffering from any of these diseases should, of course, be rejected by the medical examiner, although it might be quite possible for the same proposer to be admitted after he recovered from the last four diseases, provided his family history was favourable and his recovery complete; but it must always be kept in mind that a person who had one of these diseases is probably more liable to another attack, and especially is more liable to the onset of phthisis. This is more especially the case with regard to pleurisy, and therefore a candidate for assurance who has once been affected with pleurisy should be subjected to a very searching examination for the remains of the disease or the early traces of phthisis. So with bronchitis and asthma, and also with pneumonia, although probably in a lesser degree. But phthisis is by far the most important of the diseases of the respiratory system, and in spite of the fact that insured lives are selected and submitted to an examination of the chest when admitted to insurance, in the words of Sir Robert Christison: "Consumption is of all single diseases the most important in relation to life insurance."

The statistics of insurance companies show how important it is for the insurers to reject all lives likely to be affected with consumption. They are in most cases a loss to the companies. The figures also show that the companies derive the benefit from selection for at least some years after the lives are admitted. This is the explanation of the fact that a large number of insured consumptives survive to a comparatively late life for consumptives. The figures of the companies bring out another fact, that very few of the cases insured above forty years of age die from consumption, showing that if persons remain healthy till middle life they are not nearly so likely to be affected with consumption. This is a fact of very great importance when the proposer has a family history showing a predisposition to consumption. Experience shows that if proposers have reached thirty, and still more so thirty-five or forty years of age, or if they have well passed the age at which their relatives died or suffer from the disease, there is much less risk in accepting their lives for assurance.

When there is a suspicion of a tendency to consumption in a candidate, the general development and expansion of the chest during respiration ought to be carefully noted, as well as the weight and the pulse-rate. An abnormally high pulse-rate is often an early indication of the onset of the disease. Weak digestion and disturbances of the digestive organs generally also often precede the onset of more evident symptoms.

The late Sir Robert Christison, in discussing this question, made the following statement, and it is doubtful whether our knowledge at the present day could enable us to alter it much in any way. He stated: "General delicacy—a state of health described as 'tolerably good,' or 'pretty good,' or 'not robust,' a great liability to 'slight common colds,' or 'rheumatic pains,' or 'bilious complaints,' a pulse habitually frequent, are all suspicious circumstances in one whose family has suffered at all from consumption. Among these particulars I would call attention especially to a liability to indigestion as a serious ground of doubt when only one member of a family has been cut off by consumption. Either frequent indigestion favours the development of consumption in the predisposed by further impairing a previously doubtful constitution, or simply the two liabilities may be each the direct result of the same constitutional defect."

Proposers with the actual symptoms of chest disease cannot be accepted as healthy lives. Whether such can be admitted when the attack has been well passed will depend on whether the recovery has been so complete as to leave no trace behind it, on the family history, and on the habits and occupation as well as the other circumstances of the individual.

*Heart and Circulatory System.*—The question of heart disease in relation to life assurance has given rise to much discussion, but I doubt whether we have yet much evidence to guide us as to which lives affected with valvular disease of the heart ought to be accepted, and which ought to be rejected. There can be little doubt, as pointed out by Sir William Gairdner and others, that some diseases of the heart live to a good old age—and many medical men will also admit that a heart murmur which is distinctly present may in the course of time entirely disappear.

In spite of these facts, we have not yet got sufficient data to guide us in arriving at the most important decision for the insurance companies, viz., which are most likely to live to a good old age, and which heart murmurs are most likely to disappear, leaving the heart in a healthy condition. The time may come when we will be able to classify lives with cardiac lesions, so that those most likely to live to an old age can be picked out from the others, but I am afraid at the present time our medical knowledge does not enable us to go so far. The presence of a cardiac murmur indicating organic disease of the heart must, therefore, be taken

as a very important factor in deciding whether a life is insurable as an average life or not. Whether such a case can be admitted at an increased rate of premium must depend to a great extent on the circumstances of the proposer. In justice to the companies, probably it would be better to reject all such cases; but there may be circumstances where the risk to the company in accepting those lives would be limited. The experience of the companies has shown that cases of heart disease die not in the earlier, but in the later part of the insured period. These are, of course, selected lives, *i.e.* lives presumably free from heart disease on admission to insurance, and conclusions drawn from these statistics alone are liable to many fallacies. If a proposer with a cardiac murmur is to be admitted to insurance at all, the "loading" should be a heavy one, and it would be safer for the company to have the insurance in the form of an endowment insurance, payable at a certain age, as far below sixty as possible.

In the examination of the circulatory organs the past history of the proposer and his family history ought to be carefully inquired into, with the object of finding out any traces of the rheumatic or gouty diathesis, both of which are well recognised to be associated with diseases of the heart and blood-vessels. The pulse should be carefully noted, not only as to its rate, but more carefully as to the state of the vessel wall for any trace of degeneration of the blood-vessels.

Thickening of the arterial walls is an important symptom, and ought to debar a life from being accepted. The presence, of course, of even more serious vascular wall mischief, such as aneurysm, makes the risk too serious a one for the company to accept the proposal. As is well known, vascular degenerations are often the result of an attack of syphilis, and therefore a previous history of this disease is of considerable importance.

A too rapid or too slow pulse are suspicious symptoms, the first because it may indicate the presence of other diseases such as consumption, as well as a disturbance of the nervous mechanism of the heart's action, the latter because it is often associated with serious degeneration of the heart muscle. It must not, however, be forgotten that both are to a certain extent consistent with health, and that the former may be produced by nervousness ("the insurance heart"). A past history of rheumatism, of scarlet fever, and of chorea should make the examination more searching, because they are so apt to be associated with endocarditis and disease of the heart. The presence of pericarditis or a pericardial murmur should at least cause the postponement of the insurance. Many such murmurs entirely disappear, and although, therefore, it would be too great a risk to accept a candidate with a pericardial murmur, the same life might be accepted later at the usual or an increased rate. So also with a case having a murmur which is believed to be anæmic in origin. Such a proposal should be postponed, and the candidate should be submitted to an examination later on in order to ascertain if the murmur has actually disappeared with the disappearance of the anæmia.

*Examination of the Organs of Digestion.*—Inquiries ought to be made as to the presence of indigestion, bilious attacks, constipation, or diarrhœa, as indicating whether the proposer is in robust health or not. I take it that the mere presence of occasional indigestion alone is not sufficient to reject a proposal; but a history of indigestion may indicate the presence of a serious disease such as gastric ulcer, cancer of the stomach or liver, or cirrhosis of the liver, any one of which would render the life uninsurable. Physical examination of the abdomen should never be omitted, the size of the liver and spleen being noted.

Habits as to drinking and eating should be specially inquired into in this connection. It is well known that intemperance and the habitual use of alcohol to excess leads to disorders of the stomach and liver, especially to cirrhosis of the latter organ. A history of repeated attacks of appendicitis makes the life a more risky one unless the appendix has been removed by surgical operation.

With regard to hernia, all the best companies require a declaration from the proposers that if ever they have rupture they will constantly wear a well-fitting rupture truss. The wearing of such a truss reduces the extra risk in such cases to a minimum.

The presence of an abnormal amount of adiposity is an important factor, but this has already been referred to under height and weight. The presence of dropsy indicates some serious cardiac liver or kidney disease.

*State of the Urinary Organs.*—Formerly the insurance companies only insisted on examination of the urine in cases where the examiners were suspicious of kidney disease, but all the best companies now require a report of the result of the usual tests for abnormalities. The specific gravity, the reaction, and whether there is present albumin, sugar, or other abnormal products, should be noted.

A very high or very low specific gravity may raise the suspicion either of diabetes or Bright's disease, and further tests may or may not confirm the suspicion.

Undoubted cases of diabetes or Bright's disease are uninsurable. The presence of sugar in any considerable quantity in the urine of young lives must always be taken as an indication of true diabetes mellitus, and therefore a bar to insurance; but it is well known that in persons past middle life the presence of a small quantity of sugar in the urine is not such a serious matter,

although it almost invariably occurs in persons of a gouty diathesis. Such cases, however, are more to be reckoned as cases of gout than of true diabetes, and are to be considered more from the gouty point of view than from that of diabetes.

The mere presence of albumin in the urine is not now considered to be such an invariable indication of Bright's disease of the kidneys as it used to be. It is well known that persons apparently in good health may have albumin present in their urine, at least temporarily. The presence of tube-casts in the urine along with the albumin points very clearly to Bright's disease; but in cases where no indication of Bright's disease other than the presence of albumin in the urine is present, it is rather difficult deciding whether the life ought to be accepted or not. The urine of such cases, passed at different times in the day, should be examined on various occasions to ascertain whether the albumin is constantly present or not. Sometimes the albumin may be—(1) paroxysmal, *i.e.* it occurs at intervals separated by considerable periods during which there is no albumin present; (2) it may appear only after certain articles of diet; (3) it may appear after muscular exertion, the urine being normal when the body is at rest; (4) the urine may always be albuminous, but the albumin is in small quantities and not influenced by food or exercise. It is difficult being definite as to how such cases ought to be treated by insurance companies. True paroxysmal albuminuric cases ought not to be loaded to any great extent, if at all; but if albumin is constantly present even in persons otherwise apparently healthy a certain amount of loading should be imposed, and in some cases, where the life otherwise has something unfavourable, such as a family or personal history of gout, the life should be rejected.

Albuminuria is often associated with the gouty diathesis, and the presence of albumin in the urine of persons of sedentary habits, such as those who lead a confined life in the city with little exercise and good living, is often associated with other crystalline deposits in the urine and troubles of digestion. Dr. Hingston Fox states that such cases may be accepted unless the albumin is very abundant or the crystals very large, in which case treatment becomes necessary before acceptance. Dr. Bewley's opinion is, I think, a safer one, viz., "that the inactive habit of life, over-eating, and gouty tendency noted in these cases prevent us looking on them as first-class lives." In addition to these different causes of albuminuria there may be albumin present in the urine from heart disease, and after fevers, as, for instance, scarlet fever, diphtheria, and accidentally from discharges into the urinary passages. In the first, the heart disease would render the life uninsurable; in the second, the proposal should be postponed for six months or a year to ascertain whether the albumin is permanent; and in the last cases the nature and source of the discharge would decide whether the life was insurable or not.

*Diseases of the Nervous System.*—Most of the organic nervous diseases render lives uninsurable. The most common diseases of the brain and spinal cord are those which come on in later life, such as paralysis due to hæmorrhage, embolism, and thrombosis, and are usually associated with degenerative changes in the heart and blood-vessels. They are one of the most fruitful causes of death in later life, and hence candidates for assurance after middle life should be specially examined as to the state of their vascular system, and in this connection a history of gout and rheumatism is of great importance. A distinct history of insanity in a family should also be looked upon as an unfavourable factor in a life, and in this connection a history of intemperance in the family and in the individual should be specially inquired into, and considered along with the habits and occupation of the proposer.

How candidates for assurance with suppurative disease of the middle ear ought to be dealt with has been much discussed. The consensus of opinion of the many specialists who took part in the discussion of this subject at the British Medical Association meeting in Edinburgh, in 1898, seemed to be that there are some cases which should be admitted at ordinary rates, or with a slightly increased premium, and some should be rejected.

Cases of old-standing suppuration, where the discharge has completely or almost completely ceased, with no attacks of pain, may be admitted at the usual rates. Cases where there is a fair-sized perforation, with little discharge and no attacks of pain, may also be admitted at the ordinary rates.

In judging of the amount of risk, attention should be paid to the size and situation of the perforation in the membrane. If small and high up, the risks are greater. If the discharge is copious and fœtid, the risks are also greater (M'Bride).

Cases where there are granulations or polypi, or where there is a small perforation and offensive discharge, should not be admitted without special treatment. If the result of the treatment is satisfactory the proposal may be admitted at an increased or even the ordinary rate, according to the degree of improvement in the condition. In cases having a recurrence of attacks of pain the proposal should be rejected.

Where there is evidence of the existence of suppuration in the mastoid cells, or of caries or necrosis of the bones in any part of the ear, or where there are exostoses or cholesteatomata of the middle ear interfering with free discharge, proposals should be rejected.

In cases of suppuration with facial paralysis the proposal should not be entertained.

Where there is acute suppuration the proposal should be delayed until the result of treatment is seen. In cases where there is a family history of tuberculosis, the presence of middle ear suppuration should be looked on as an unfavourable factor.

*General Constitutional Diseases.*—The most important are rheumatism, gout, and syphilis. The first two I have referred to in connection with the various organs. A distinct family history of rheumatism and of gout should be looked on as unfavourable factors, and if these diseases in addition have manifested themselves in any way in the proposer, the life should either be loaded or rejected altogether according to the form in which he has been affected.

His rejection will depend on whether his organs have or have not been affected by the disease. Whilst, therefore, a rheumatic individual would be rejected if he had a heart murmur, if that organ had escaped his life might be accepted with or without a load in special cases. The question of the loading of cases showing a gouty history has been much discussed. It is the custom of the companies to impose an extra premium for gout, but according to Meikle ("Gout as a Factor in Life Assurance," *Brit. Med. Journ.*, 1898, vol. iii. 764), the extra they have been in the habit of imposing is too little to cover the risk. His observation was based upon 525 lives charged an extra premium because of their lives being affected by gout. He ascertained the number that entered upon each age of life, and computed the number that were expected to die at these ages according to the experience of healthy lives. The number calculated to die according to this standard was 120. The actual number who died was 160, or an increase of 33 per cent. This increased mortality with one exception pervaded the whole of life.

These figures are very striking as well as the other tables in Meikle's paper, but exception might be taken to them because of the indefinite way in which cases are sometimes classed as gout. As an extra premium had been imposed, and had been paid by these cases on account of gout, in all probability most of the cases were very decidedly gouty. What difference the inclusion of cases where family history and personal condition showed only a trace or mild degree of gout not taken note of would have made on these figures, it is impossible to say; but the mortality in all probability would have been somewhat reduced. The indefinite nature of slight symptoms of gout makes it very difficult getting reliable statistics on the subject. Meikle traced the cause of death in the 160 gouty persons, and classified them as follows:—He first deducted 63 who died from various miscellaneous diseases, and found that 42 per cent died from affections of the brain, 26 from affections of the heart, 11 per cent from gout, 11 per cent from affection of the kidneys, 10 per cent from natural decay.

*Syphilis.*—A candidate for assurance with primary, secondary, or tertiary symptoms of syphilis should not be admitted as a healthy life, and his proposal should, if not absolutely refused, be postponed till all symptoms and results of the disease have disappeared, when, if the case has been properly treated, it may be admitted at an increased rate. There is, however, little doubt that many syphilitic cases show symptoms of the disease many years after all symptoms have temporarily disappeared, whilst others never have the slightest return of the disease. Careful enquiry as to the history, the method of treatment, and the progress of the symptoms, should aid the examiner in deciding as to the admission or rejection of individual cases.

*Cancer.*—Cases of cancer are uninsurable. Whilst one case of cancer in a family may not affect the life, the fact that a proposer's father and mother both died of cancer should be looked on as an unfavourable factor. Endowment insurances are preferable in such cases.

*Female Lives.*—If the candidate for assurance is a female the examiner has to pay special attention to the functions of the female generative organs, and special enquiries must be made to discover whether menstruation is and has been regular and physiological.

The presence of disease of the uterus, Fallopian tubes, or ovaries makes the life not so good a one, and may require a special report. In married women information may be elicited from inquiries as to the length of time married, the number of pregnancies, the number of children alive and their state of health. In this connection the difficulty of the labour and the rapidity of recovery from her confinements are of great importance, especially in cases where the proposer is actually pregnant at the time of examination.

What amount of extra ought to be imposed for pregnancy has been much discussed, chiefly because of the great difficulty in getting reliable statistics on the subject. In a paper by Playfair and Wallace, read at the British Medical Association meeting in Edinburgh in 1898, giving the results of their investigation into the statistics of the Royal Maternity, Edinburgh, they arrived at the conclusion that the uniform rate that the companies are in the habit of charging for pregnancy was in many cases too low. Their conclusions are the following:—

1. For the uniform extra premium at present charged, an extra premium varying in amount according to age should be substituted.

2. The extra premium for a first pregnancy should be at least three times as great as that for a subsequent pregnancy.

3. A proposal from a woman aged thirty or upwards, pregnant for the first time, should be delayed.

4. A proposal for insurance from a pregnant woman aged forty or upwards, whatever the number of pregnancy, should be delayed.

The figures of a maternity hospital can hardly be taken as the average for a community, and especially as the average amongst female lives which are likely to be insured. The worst cases of the lower class community are apt to be attended by the maternity medical officers for many reasons. The outdoor cases are in the most insanitary houses; that class of the community only engage and send for doctors when the labour is expected to be a severe one, most of the ordinary labours being attended by midwives, or other women with more or less experience, and many of the cases are unmarried females. Although the maternity figures are interesting, they cannot be accepted as conclusive. In cases where the candidate is actually pregnant it is safer for the companies to postpone the insurance, if possible, till after the confinement; but where this cannot be done, and it is necessary to have the insurance completed at once, a special loading-rate on the lines of the above conclusions may be imposed.

4. *Habits, Occupation, etc.*—The habits and occupation of the proposer have already been alluded to in various connections. The question of temperance in eating and drinking is one of special importance, especially when considered in connection with a family or personal history of gout, rheumatism, or intemperance. It will at once be seen how important also the occupation of the individual is in this connection. A person whose occupation exposes him constantly to the temptation of "nipping," or taking alchohol frequently, although in small quantities, is very apt to become more and more intemperate, and to develop other diseases as the result of his alcoholic habits. It was long ago shown (Registrar-General's Report for 1851) that persons whose occupation exposed them to such temptations died at an earlier age than the average of the community. As long ago as 1876, Stott, from the experience of a well-known company for fifty years, arrived at the conclusion that the mortality amongst publicans and innkeepers, and those connected with the retail liquor trade, was 63 per cent in excess of the Carlisle table, and 68 per cent in excess of the Actuaries' table.

He also concluded that the practice of imposing an extra rate of £1 per cent in this class was necessary, but sufficient to cover the risk. A joint inquiry which was made by the Scottish Life Offices into the mortality of the same class of persons brought out much the same results. The following table shows the annual mortality per cent at the different ages as compared with the ordinary assured lives:—

| | Annual mortality per cent. | |
|---|---|---|
| Age. | Publicans. | Other persons. |
| 30 | 1·48 | 0·77 |
| 40 | 2·59 | 1·03 |
| 50 | 3·08 | 1·60 |
| 60 | 4·59 | 2·97 |

The actual deaths exceeded the expected by 83 per cent; the actual deaths being 420, and the expected only 235.

With reference also to habits, it is well known that people who lead an outdoor life in the country are healthier than those of more sedentary habit, and especially if the latter are shut up indoors in town. The family and personal history have to be considered along with the habits and occupation. A person having a tendency or predisposition to gout is more liable to suffer from the disease if he has sedentary habits, little exercise, and little outdoor life; and in the same way a person with a family history of phthisis is more likely to escape the disease if he lives an outdoor life, with enough exercise and no unhealthy surroundings. On the other hand, if such an individual has an occupation where the air he breathes is contaminated with dust or other impurities, or if he is engaged in an office sitting over a desk where his lungs do not get properly expanded, and he is unable to get sufficient exercise, he is much more likely to become affected with the disease.

It is well known that some occupations are more healthy than others, and many statistics have been collected showing the rate of mortality among different classes of the community. The figures of Dr. Bertillon in France and of Dr. William Ogle in England bring out practically the same result. They show the enormous mortality amongst certain workers. According to Ogle, if clergymen be taken as the standard and represented by 100, then the mortality may be represented as 169 among commercial clerks, 108 amongst gardeners, 114 amongst farmers, 158 amongst shopkeepers, 189 amongst tailors, 143 amongst fishermen, 267 amongst cabmen, 160 amongst coal-miners, 222 amongst quarrymen, 211 amongst butchers, 300 amongst file makers, 229 amongst scissor makers, 314 amongst earthenware makers, 397 amongst inn and hotel servants, etc.

5. *The place of residence* of the individual insuring has to be considered by the insurance company, as it is well known that some countries are healthier than others, and the death-rate of different countries varies. In tropical countries the inhabitants do not live so long as they do in temperate climates, and this applies more especially to Europeans who reside in tropical countries. Michael Levi made the calculation that there is 1 death annually amongst every 25 of the population from the equator to the 20th degree of latitude, 1 in 35 from the 20th

to the 40th latitude, 1 in 43 from the 40th to the 60th, and 1 in 50 from the 60th to the 80th. The death-rate among Europeans in the tropical and subtropical regions is probably greater than these figures indicate, but much depends on the elevation of the country as well as its sanitary conditions and freedom from special diseases such as malaria. High tableland is healthier for Europeans than low-lying districts, and much therefore depends on the configuration of the country. Companies are in the habit of imposing a certain increase of rate for those insured who reside in tropical or unhealthy climates, such as tropical Africa and East and West Indies.

6. *Age of Proposer.*—This, of course, is one of the most important for the insurance company to ascertain, because the expectation of life of healthy persons is calculated from the age.

Many expectation of life tables have been framed to show what the average expectation of life of persons is at different ages. These tables have already been referred to; but as they are of great importance, I herewith give the Carlisle table alongside of the Institute of

EXPECTATION OF LIFE ACCORDING TO THE CARLISLE AND INSTITUTE OF ACTUARIES' TABLES.

| Age. | Carlisle | Actuaries. | | | Age | Carlisle. | Actuaries. | | |
|---|---|---|---|---|---|---|---|---|---|
| | | $H^M$ | $H^F$ | $H^{MF}$ | | | $H^M$ | $H^F$ | $H^{MF}$ |
| 0 | 38·7 | 68·4 | 55 5 | 57·6 | 41 | 27·0 | 26·7 | 27·6 | 26·8 |
| 1 | 44·7 | 57·4 | 54 5 | 56·6 | 42 | 26·3 | 26·8 | 27·0 | 26·1 |
| 2 | 47·5 | 56·4 | 53·5 | 55·6 | 43 | 25·7 | 25·2 | 26 3 | 25 4 |
| 3 | 49·8 | 56·3 | 52·5 | 55·1 | 44 | 25·1 | 24·5 | 25 6 | 24·7 |
| 4 | 50·8 | 55·3 | 53·0 | 54 8 | 45 | 24·5 | 23·8 | 25·0 | 24·0 |
| 5 | 51·2 | 54·3 | 52·0 | 53·8 | 46 | 23·8 | 23·1 | 24·3 | 23·3 |
| 6 | 51·2 | 53·8 | 51·0 | 53·1 | 47 | 23·2 | 22·4 | 23·7 | 22·6 |
| 7 | 50·8 | 53·1 | 50·9 | 52·7 | 48 | 22·5 | 21·7 | 23·0 | 21·9 |
| 8 | 50·2 | 52·1 | 49 9 | 51·7 | 49 | 21·8 | 21·0 | 22·3 | 21·2 |
| 9 | 49·6 | 51 1 | 49·2 | 50·8 | 50 | 21·1 | 20 3 | 21·6 | 20·5 |
| 10 | 48·8 | 50·3 | 18·2 | 49·9 | 51 | 20·4 | 19·6 | 20·9 | 19 8 |
| 11 | 48 0 | 49 5 | 47·3 | 49·4 | 52 | 19·7 | 19·0 | 20·2 | 19·2 |
| 12 | 47·3 | 48·7 | 46·5 | 48·4 | 53 | 19·0 | 18·3 | 19·5 | 18·5 |
| 13 | 46·5 | 47·9 | 45·8 | 47·5 | 54 | 18·3 | 17·6 | 18·9 | 17·8 |
| 14 | 45·7 | 47·0 | 45·1 | 46·6 | 55 | 17·6 | 17·0 | 18·2 | 17·1 |
| 15 | 45·0 | 46·2 | 44·3 | 45·9 | 56 | 16·9 | 16·3 | 17·5 | 16 5 |
| 16 | 44·3 | 45·3 | 43·6 | 45·1 | 57 | 16·2 | 15·7 | 16·9 | 15·9 |
| 17 | 43·6 | 44·4 | 42·9 | 44·2 | 58 | 15 5 | 15·1 | 16·2 | 15·3 |
| 18 | 42 9 | 43·6 | 42·2 | 43·4 | 59 | 14·9 | 14·4 | 15·5 | 14·6 |
| 19 | 42·2 | 42·8 | 41·5 | 42·6 | 60 | 14·3 | 13·8 | 14·9 | 14·0 |
| 20 | 41·5 | 42·1 | 40·8 | 42·0 | 61 | 13·8 | 13·2 | 14·2 | 13·4 |
| 21 | 40·7 | 41·3 | 40·1 | 41·2 | 62 | 13·3 | 12·7 | 13·6 | 12·8 |
| 22 | 40·0 | 40·6 | 39·4 | 40·5 | 63 | 12·8 | 12·1 | 12·9 | 12·3 |
| 23 | 39·3 | 39·9 | 38·7 | 39·8 | 64 | 12·3 | 11·5 | 12·3 | 11·7 |
| 24 | 38·6 | 39 1 | 38·0 | 39·1 | 65 | 11·8 | 11·0 | 11·8 | 11·2 |
| 25 | 37·9 | 38·4 | 37·4 | 38 4 | 66 | 11·3 | 10·5 | 11·2 | 10·6 |
| 26 | 37·1 | 37·7 | 36·8 | 37·6 | 67 | 10·7 | 10·0 | 10·7 | 10·1 |
| 27 | 36·4 | 36·9 | 36·2 | 36·9 | 68 | 10·2 | 9·5 | 10·1 | 9·6 |
| 28 | 35·7 | 36·2 | 35·7 | 36·2 | 69 | 9·7 | 9·0 | 9·6 | 9·1 |
| 29 | 35·0 | 35·4 | 35·1 | 35·5 | 70 | 9·2 | 8·5 | 9·1 | 8·7 |
| 30 | 34·3 | 34·7 | 34·5 | 34·7 | 71 | 8·6 | 8·0 | 8·6 | 8·2 |
| 31 | 33·7 | 33·9 | 33·9 | 34·0 | 72 | 8·2 | 7·6 | 8·1 | 7·6 |
| 32 | 33·0 | 33·2 | 33·3 | 33·3 | 73 | 7·7 | 7·1 | 7·7 | 7·2 |
| 33 | 32·4 | 32·5 | 32·7 | 32·6 | 74 | 7·3 | 6·7 | 7·3 | 6·8 |
| 34 | 31·7 | 31·7 | 32·1 | 31·9 | 75 | 7·0 | 6·4 | 6·9 | 6·6 |
| 35 | 31·0 | 31·0 | 31·4 | 31·1 | 76 | 6·7 | 6·0 | 6·6 | 6·2 |
| 36 | 30·3 | 30·3 | 30·8 | 30·4 | 77 | 6·4 | 5·7 | 6·3 | 5·8 |
| 37 | 29·6 | 29·6 | 30·2 | 29·7 | 78 | 6·1 | 5·3 | 6·0 | 5·5 |
| 38 | 29·0 | 28·8 | 29·5 | 29·0 | 79 | 5·8 | 5·0 | 5·7 | 5·2 |
| 39 | 28·3 | 28·1 | 28·9 | 28·8 | 80 | 5·5 | 4·7 | 5·5 | 4·9 |
| 40 | 27 6 | 27·4 | 28·3 | 27·6 | | | | | |

Actuaries' new experience tables. The first was based on calculations of the deaths in two parishes in Carlisle during several years at the end of last century, the latter was the experience table of twenty insurance companies compiled in 1869. These tables are used by the insurance company as the basis for calculating the rate of premium to be paid by each policy-holder, the amount of premium per cent varying according to age.

**Ligaments.**—Band-like structures, usually of compact connective tissue, which unite the articular ends of bones or adjacent organs or parts—*e.g.* the broad ligament (*see* BROAD LIGAMENTS; GENERATION, FEMALE ORGANS OF; PUERPERIUM, *Involution of Broad Ligaments*); the infundibulo-pelvic ligament (*see* GENERATION, FEMALE ORGANS OF, *Fallopian Tubes*); the ligamentum latum pulmonis (*see* PLEURA, DISEASES OF, *Anatomy*); the ovarian ligament (*see* GENERATION, FEMALE ORGANS OF, *Ovaries*; PUERPERIUM, PHYSIOLOGY, *Involution*); the round ligament (*see* GENERATION, FEMALE ORGANS OF, *Ligaments of the Uterus*; PREGNANCY, PHYSIOLOGY, *Change in Ligaments*; PUERPERIUM, PHYSIOLOGY, *Involution*); suspensory ligament of axilla (*see* AXILLA, *Anatomy*); and the uterine, utero-sacral, and utero-vesical ligaments (*see* GENERATION, FEMALE ORGANS OF, *Ligaments of Uterus, etc.*). *See also* under JOINTS, ANKLE, HIP-JOINT, KNEE-JOINT, etc.

**Ligar's Line.**—In order to determine the point of emergence of the sciatic artery, a line is drawn from the posterior superior iliac spine to the inner point of trisection of a line between the tuberosity of the ischium and the great trochanter, and the middle point in this line will indicate the artery.

**Ligation.**—The applying of a ligature, *e.g.* to a blood-vessel, pedicle, or stump (cord).

**Ligature.**—The tying of an artery, pedicle, etc. with a thread or cord of silk, catgut, etc., or the thread or cord itself. *See* ANEURYSM (*Treatment, Ligature*); ARTERIES, LIGATURE OF; LABOUR, MANAGEMENT OF (*Care of Child, Tying the Cord*).

**Light.** *See* BALNEOLOGY (*Solar and Radiation Baths, Light Baths*); PHYSIOLOGY, SENSES (*Vision*); PUPIL (*Nervous Mechanism*); SPECTROSCOPE IN MEDICINE.

**Lightening.**—The sensation of greater ease in respiration and digestion which precedes the onset of labour, and is due to the sinking down of the uterus into the pelvis. *See* LABOUR, PHYSIOLOGY OF (*Factors of Labour, the Powers*).

**Lighting.**—The illumination of dwelling houses, public buildings, towns, roads, etc. with gas, lamps, electric light, etc. *See* VENTILATION AND WARMING.

**Lightning.** *See* MEDICINE, FORENSIC (*Injuries caused by Lightning*); EYEBALL, INJURIES OF (*Effects of Lightning*).

**Lightning-Pains.** *See* TABES DORSALIS (*Symptomatology, Sensory Symptoms, Subjective*).

**Lignin.**—A body closely allied to cellulose ($C_6H_{10}O_5$), and perhaps having the formula $C_{19}H_{24}O_{10}$, and forming the greater part of wood.

**Lignosulphin or Lignosulphite.**—A disinfectant product obtained during the manufacture of sulpho-cellulose or cellulose.

**Lignum.**—Wood, especially in such expressions as *lignum vitæ* (Guaiac Wood), *lignum febrium* (Cinchona Wood), etc.

**Ligula.**—A tongue-shaped or strap-shaped structure.

**Lillenfeld's Theory.**—The theory that, in coagulation of the blood, the splitting of fibrinogen into globulin and thrombosin is due to the nucleo-proteid.

**Lily of the Valley.** *See* CONVALLARIA MAJALIS.

**Liman Cure.**—The treatment of disease (*e.g.* rheumatism) by bathing in salt lakes, called *limans*, near Odessa; limanol is an extract obtained from the mud of these limans.

**Limatura Ferri.**—Iron filings.

**Limb.**—One of the extremities in contradistinction to the head and trunk of the body. *See* DEFORMITIES; EMBRYOLOGY, HUMAN; FŒTUS AND OVUM, DEVELOPMENT OF (*Limbs*); JOINTS; LABOUR, ACCIDENTAL COMPLICATIONS (*Displacement of Arms, Prolapse of Foot*); LABOUR, DIAGNOSIS AND MECHANISM (*Podalic Lies*); PREGNANCY, INTRA-UTERINE DISEASES OF FŒTUS (*Congenital Amputations*); SYPHILIS (*in Children, Bones of Limbs*). *See also* AMPUTATIONS; FRACTURES; etc., etc.

**Limbus.**—A border, margin, edge, or rim, *e.g.* the limbus acetabuli of the hip-joint, the limbus corneæ or margin of the cornea where it joins with the sclerotic.

**Lime.** *See* CALCIUM AND ITS SALTS; DIET (*Mineral Constituents of Food*); GOUT (*Etiology, Lime in Drinking Water*); MINERAL WATERS (*Earthy and Calcareous*); STOOLS (*Intestinal Sand*); TEETH (*Dental Caries, Causes, Deficiency of Lime Salts*); URINE, PATHOLOGICAL CHANGES IN (*Urinary Calculi*).

**Lime Fruit.**—The fruit of various species of *Citrus*, from which citric acid is prepared; the lime-juice of commerce, which contains between 7 and 10 per cent of citric acid and keeps without the addition of alcohol, is used in scurvy. *See* CITRIC ACID; SCURVY IN ADULTS (*Prophylaxis*).

**Lime Process.**—A method of disposing of sewage by subsidence and chemical treatment, slaked lime being added to the sewage in the proportion of 12 or 15 grains to the gallon; modifications of this method are those in which sulphate of alumina, or clay, or protosulphate of iron, or herring brine are added as well as the lime.

**Lime Test.**—The detection of lime in drinking water by the addition of a solution of oxalate of ammonium, which gives a white precipitate.

**Limiting Membrane.** *See* PHYSIOLOGY, SENSES (*Vision, Anatomy*); RETINA AND OPTIC NERVE (*Anatomy*).

**Limnœa Truncatulata.** *See* PARASITES (*Helminths*).

**Limonis Cortex.**—Lemon peel. *See* LEMON.

**Limosis.**—Insatiable appetite or unnatural craving for food. *See* APPETITE (*Perversions*); BULIMIA; PICA.

**Limotherapy.**—Treatment of diseases by deprivation of food (Gr. λιμός, hunger, and θεραπεία, treatment).

**Linadin.**—A preparation obtained from the spleen (containing iron and iodine) and used in cases of malarial cachexia.

**Linalool.**—A constituent (an alcohol) of oil of lavender, $C_{10}H_{17}OH$, isomeric with borneol, geraniol, and menthol. *See* LAVENDER.

**Linamarin.**—A glucoside obtained from *linseed* (*q.v.*), and splitting up into glucose and hydrocyanic acid under the action of acids.

**Linctus.**—A thick, sweet, medicinal preparation which can be taken by licking (Lat. *lingere*, to lick); it usually contains honey or treacle.

**Linctus Opiatus.**—A linctus containing treacle and tincture of opium (2 m.) which is used in troublesome night cough as a local sedative to the pharynx; it is allowed slowly to melt in the mouth.

**Linea.**—A term commonly used in anatomy to signify an elongated mark, or strip of tissue, or boundary, or ridge, *e.g.* the iliopectineal line, the linea aspera, the linea alba, etc.; sometimes the line is imaginary, *e.g.* the nipple line or the axillary line. The lineæ albicantes of pregnancy are cracks due to the rapid distension of the anterior abdominal wall by the growing uterus. The blue line on the gums is characteristic of lead-poisoning.

**Linen.** *See* TRADES, DANGEROUS (*Flax and Linen*).

**Lingering Labour.** *See* LABOUR, PRECIPITATE AND PROLONGED (*Prolonged Labour*).

**Ling's System.**—Swedish gymnastics or physical culture by systematically arranged exercises.

**Lingua.**—The tongue or any tongue-like structure.

**Lingua Frænata.**—Tongue-tie. *See* TONGUE (*Malformations, Ankyloglossia*).

**Lingual.**—Relating to the tongue, *e.g.* the lingual artery (*see* ARTERIES, LIGATURE OF), the lingual nerve (*see* NERVES, NEURALGIA, *Division of Lingual Nerve*), and the lingual tonsil (*see* LARYNX, HYPERTROPHY OF THE LINGUAL TONSIL; TONGUE, *Anatomy*).

**Linguatula.**—The linguatulidæ are parasitic arthropods found in the nasal cavities and lungs; there are various species, *e.g.* l. caprina, l. rhinaria, l. serrata, etc. *See* PARASITES (*Arthropods*).

**Lingula.**—A small tongue or tongue-like structure, *e.g.* the lobule between the central lobe of the cerebellum and the valve of Vieussens.

**Linimentum.**—A pharmaceutical preparation for rubbing into or applying to the skin, usually made with oil; an embrocation. The principal official linimenta are l. aconiti (not to be rubbed in), l. camphoræ ammoniatum, l. belladonnæ, l. calcis, l. camphoræ, l. chloroformi, l. crotonis, l. hydrargyri, l. potassii iodidi cum sapone, l. opii, and l. saponis.

**Linoleum.**—The oil-cloth and linoleum manufacture is an offensive trade. Linoleum is made from cork mixed with linseed oil, rosin, and Kauri gum, and the heating of the two latter substances causes the nuisance; the vapours should be passed through a water bath and into a furnace.

**Linseed.**—Linum; the dried seeds of *Linum usitatissimum*, grown in Britain. They are small and flat in shape, with pointed ends and sharp edges, and of a shiny brown colour. The outside of the seeds contains mucilage, and the interior a fixed oil. *Preparations*—1. Linum Contusum (linseed meal). 2. Oleum Lini (a yellow viscid fixed oil).

Linseed poultices are used to relieve pain, and to act as counter-irritants over any inflamed area. Linseed meal is beat with a spatula into a thick paste by the gradual addition of boiling water. The paste is spread about ½-inch thick on linen, and applied immediately as hot as the patient can bear it. Its action may be increased by adding 1 part of mustard to 16 of linseed. Linseed oil has been applied to burns. "Carron oil," used for this purpose, contains equal parts of lime water and linseed oil. "Linseed tea" is made by infusing 150 grs. of linseed and 50 grs. of liquorice in 10$\frac{3}{}$ of boiling water for two hours, and is used as a demulcent in sore throat. It is also said to be diuretic.

**Liouville's Icterus.**—Icterus neonatorum or jaundice of the new-born infant. *See* NEW-BORN INFANT (*Diseases, Icterus neonatorum*).

**Lip.**—A fleshy fold bordering upon an opening such as the mouth, or the vulva (labium majus and minus), or the os uteri (lip of the cervix). *See* CHILDREN, CLINICAL EXAMINATION OF (*Mouth and Throat, Lips*); ECZEMA (*Types, Lips*); HERPES (*Herpes facialis*); MENTAL DEFICIENCY (*Cretinoid Cases, Lips*); MOUTH, DISEASES OF (*Diseases and Deformities of the Lips*); MYASTHENIA GRAVIS (*Symptomatology, Face*); PALATE, CONGENITAL MALFORMATIONS OF THE MOUTH (*Hare-Lip*); PARALYSIS (*Bulbar Paralysis, Lips*).

**Lipacidæmia.**—The presence of fatty acids in the blood (Gr. λίπος, fat).

**Lipaciduria.**—The presence of volatile fatty acids in the urine, such as acetic, butyric, formic, and propionic.

**Lipæmia.**—The presence of fatty particles in the blood, as occurs sometimes in diabetes mellitus, and may lead to lipæmic clots in the vessels (capillaries of the lungs). *See* LUNGS, VASCULAR DISORDERS (*Pulmonary Embolism, Fat Emboli*).

**Lipanin.**—A proposed substitute for cod-liver oil, consisting of a solution of oleic acid in olive oil (dose, 1 to 3 oz. daily).

**Liparocele.**—A fatty tumour or fat hernia; lipocele.

**Liparomphalus.**—An umbilical fatty tumour or epiplocele.

**Lipase.**—A fat-splitting ferment or enzyme, found in the blood and pancreatic juice; it can be detected in the urine of dogs after injury to the pancreas.

**Lipliawsky's Test.**—A test for diacetic acid in the urine: with 6 cc. of a 1 per cent solution of paramidoacetophenon (containing 2 cc. of concentrated hydrochloric acid), 3 cc. of a 1 per cent aqueous solution of potassium nitrite is mixed, and an equal quantity of urine is added along with a drop of ammonia; to 10 drops of this mixture 15 cc. of concentrated hydrochloric acid, 3 cc. of chloroform, and 2 drops of chloride of iron solution are added. If diacetic acid be present, the chloroform will show a violet colour.

**Lipo-.**—In compound words *lipo-* (Gr. λίπος, fat) means relating to fat—*e.g. lipocardiac*, relating to a fatty heart; *lipogenesis*, the formation of fat; *lipolysis*, the decomposition of fat; etc.

**Lipochromes.**—Fat-pigments or luteins, *e.g.* carotin, tetronerythrin, etc. *See* Physiology, Circulation (*Blood, Serum*); Pigments of the Body and Excreta (*Lipochromes*); Spectroscope in Medicine.

**Lipoma.**—A fatty tumour, the adipose tissue of which is in lobules, separated by fibrous septa. *See* Bone, Diseases of (*Tumours, Lipoma*); Broad Ligaments, Diseases of (*Tumours*); Conjunctiva, Diseases of (*Congenital Anomalies, Fibro-fatty Tumour*); Fallopian Tubes (*Tumours, Lipoma*); Mammary Gland, Diseases of (*Neoplasms, Fatty Tumours*); Mouth, Diseases of (*Diseases of Floor of Mouth, Lipomata*); Neck, Region of (*Solid Tumours, Lipomata*); Orbit, Diseases of (*Solid Tumours, Lipoma*); Pelvis, Hæmatocele and Hæmatoma (*Lipoma of Mesosalpinx*); Peritoneum, Tumours of (*Lipomata and Lipo-fibromata*); Stomach and Duodenum, Diseases of (*New Growths, Lipomata*); Tumours (*Connective Tissue, Type, Lipomata*).

**Lipomatosis.**—An abnormal deposition of fat (obesity), or fatty degeneration. *See* Obesity (*Adiposis Dolorosa and Lipomatosis, Diagnosis*).

**Lipomphalocele.**—An umbilical hernia one of the contents of which is the fat-laden omentum.

**Lipomyoma.**—A myomatous (or fibroid) tumour, containing fat.

**Lipomyxoma.**—A tumour composed of mucoid tissue and fat.

**Lipomthymia.**—Faintness or actual syncope (Gr. λείπω, I leave or desert, and θυμός, the soul or breath).

**Lipotrichia.**—Falling out of the hair, such as occurs in syphilis.

**Lippitude.**—The state of being bleareyed; the results of marginal blepharitis. *See* Eyelids, Affections of (*Blepharitis Marginalis*).

**Lippspringe.** *See* Balneology (*Germany, Calcareous Waters*); Mineral Waters (*Earthy and Calcareous*).

**Lip-Tie.**—The fixation of the lip to the gum by means of a congenital band or fold.

**Lipuria.**—The presence of fat in the urine, either in association with renal disease (chronic Bright's disease, pyonephrosis), or in chyluria, or in the absence of any disease of the kidneys (after an excessive fat diet, in phosphorus-poisoning, in diabetes mellitus, pyæmia, etc.). *See* Pancreas, Diseases of (*Pancreatitis*); Urine, Pathological Changes in (*Lipuria and Chyluria*).

**Liquefaction-Necrosis.** *See* Colliquative Necrosis.

**Liqueurs.**—Aromatic alcoholic preparations, containing from 50 to 59 per cent. of alcohol. *See* Alcohol (*Spirits, Liqueurs*).

**Liquid.**—The common name given to several pharmaceutical and chemical liquors, such as *blistering liquid* (liquor epispasticus), *Labarraque's disinfecting liquid* (liquor sodæ chlorinatæ), and *Dutch liquid* (ethylene dichloride).

**Liquor.**—A solution of a non-volatile chemical substance in water (distilled); occasionally the dissolving of the substance (which may *not* be a definite chemical substance) is aided by adding something to the water. Such solutions are termed (in Pharmacy) *liquores*, and there are about forty-three in the B.P., of which twenty-seven are given internally. If the solution be made by repeated percolation with the addition of alcohol, the result is a *concentrated solution* (liquor concentratus). The term liquor is also applied to any liquid or fluid (*e.g.* the liquor pericardii or the liquor cerebro-spinalis). *See* Prescribing.

**Liquor Amnii.**—The fluid contained in the amniotic sac in antenatal life; it shields the fœtus from injuries, allows free movement of its limbs, maintains a constant temperature round it; it acts as a hydrostatic dilator of the cervix in labour, and serves probably as a source of water-supply and possibly (to a slight extent) as a food-supply to the new organism. *See* Embryology, Human; Fœtus and Ovum, Development of (*The Amnion*); Labour, Precipitate and Prolonged (*Faults in the Passenger, Liquor Amnii*); Pregnancy, Pathology of, Ovum and Decidua (*Affections of the Amnion*).

**Liquor Ammoniæ Fortis.** *See* Ammonium.

**Liquor Anodynus.**—Spiritus Ætheris Compositus or Hoffmann's anodyne.

**Liquor Antisepticus.** *See* Listerine.

**Liquor Arsenicalis.**—Fowler's solution. *See* Arsenic.

**Liquor Arsenii et Hydrargyri Iodidi.** Donovan's solution. *See* Arsenic.

**Liquor Calcis.**—Lime-water or Aqua calcis; with sugar added, the *l. calcis saccharatus* is produced.

**Liquor Carbonis Detergens.**—An alcoholic solution of coal-tar.

**Liquor Concentratus.**—A solution prepared by repeated percolation of a drug (powdered) with the addition of some alcohol to the distilled water (20 per cent); the official concentrated solutions are those of calumba, chiretta, cusparia, rhatany (krameria), quassia, rhubarb, sarsaparilla, senega, senna, and serpentary; and the dose of nearly all the concentrated liquores is ½ to 1 fl. dr. (in the two exceptions the dose is larger).

**Liquor Epispasticus.** — Blistering fluid. *See* CANTHARIDES.

**Liquor Folliculi.**—The fluid contained in a Graafian follicle, which escapes when the follicle ruptures and the ovum is expelled; liquor graafianus. *See* FŒTUS AND OVUM, DEVELOPMENT OF.

**Liquor Graafianus.** *See* LIQUOR FOLLICULI.

**Liquor Hydrargyri Perchloridi.** *See* MERCURY (*Hydrargyri Perchloridum*).

**Liquor Hydrogenii Peroxidi.** *See* HYDROGEN PEROXIDE.

**Liquor Morgagni.**—The fluid which exists, in small quantity, between the crystalline lens and the inner surface of the posterior capsule and between the lens and the capsular epithelium.

**Liquor Opii Sedativus.**—Battley's solution.

**Liquor Pancreatis.** — Pancreatic solution which has the power of converting starch into sugar, and albumen into peptone.

**Liquor Picis Carbonis.**—Solution of prepared coal-tar.

**Liquor Sanguinis.** — The blood plasma. *See* BLOOD.

**Liquorice.**—Glycyrrhizæ Radix. The roots and underground stems of *Glycyrrhiza glabra*, grown in Britain. Occurs in long cylindrical pieces, with a yellow fibrous appearance when peeled, and a sweet taste. It contains *Glycyrrhizin*, a yellow glucoside, *Asparagin*, and *grape sugar*. *Preparations*—1. Extractum Glycyrrhizæ. *Dose*—5-20 grs. 2. Extractum Glycyrrhizæ Liquidum. *Dose*—½-1ʒ. 3. Pulvis Glycyrrhizæ Compositus, containing senna, sulphur, and fennel. *Dose*—60-120 grs.

Liquorice is employed chiefly as a flavouring agent to hide the taste of disagreeable medicines, such as cascara, aloes, and quinine. It forms a good basis for pills. It is sometimes used as a demulcent for sore throats. The compound powder is an excellent mild laxative, but it owes its properties to the senna and sulphur.

**Liriodendron Procerum.** — The tulip tree, the bark of which has been employed as a substitute for cinchona in malaria and as a general tonic.

**Lisdoonvarna.** *See* BALNEOLOGY (*Great Britain and Ireland, Co. Clare*); MINERAL WATERS (*Sulphur*).

**Lisfranc's Amputation.**—A tarso-metatarsal amputation, the metatarsal bones being disarticulated from the tarsal. *See* AMPUTATIONS (*Lower Extremity*).

**Lisfranc's Tubercle.**—A roughened eminence on the anterior surface of the first rib near its upper border.

**Lissauer's Tract.**—A tract of nerve fibres in the spinal cord between the apex of the posterior horn of grey matter and the surface of the cord. *See* PHYSIOLOGY, NERVOUS SYSTEM (*Spinal Cord, Conducting Paths*).

**Lissencephaly.**—The condition of brain in which the convolutions are poorly developed (Gr. λισσός, smooth).

**Listerine.** — A proprietary preparation said to consist of benzoic and boric acids, thymol, eucalyptus, alcohol, etc.; a similar preparation is the American *Liquor antisepticus*.

**Listerism.**—The antiseptic and aseptic treatment of wounds and technique of operations; so named after Lord Lister, who introduced it. *See* ASEPTIC TREATMENT OF WOUNDS.

**Lister's Ointment.** — A boric acid ointment containing white wax, paraffin, and almond oil, as well as boracic acid.

**Lithæmia.**—The presence of uric acid and urates in excessive amount in the blood. *See* LIVER, DISEASES OF (*Functional, Lithæmia*); GOUT; HYDROPATHY (*Subacute and Chronic Diseases*); URIC ACID.

**Lithagogue.**—Having the power of expelling calculi.

**Litharge.**—Lead oxide. *See* LEAD.

**Lithates.**—Salts of lithic or uric acid (Gr. λίθος, a stone). *See* LITHIUM; URIC ACID.

**Lithia.**—Lithium oxide. *See also* LITHIUM.

**Lithiasis.**—The formation of calculi in the urinary bladder, in the kidney, in the bile duct or gall bladder, in the pancreatic duct, in the lacrimal duct, under the prepuce, etc. *See* under the various organs and parts.

**Lithic Acid.**—Uric acid, *e.g.* the lithic acid diathesis or lithæmia is the uric acid diathesis. *See* URIC ACID.

**Lithio-Piperazine.**—A combination of

lithium and piperazine, used in doses of 15 to 45 grains (daily) in gout.

**Lithium.**—Symbol, Li.; atomic weight, 6·97. It is official in the form of the following salts — 1. *Lithii Carbonas*, a white powder, soluble 1 in 70 of water. *Dose*—2-5 grs. 2. *Lithii Citras*, a white crystalline powder, soluble 1 in 2 of water. *Dose*—5-10 grs. *Preparation*—Lithii Citras Effervescens. *Dose* — 60-120 grs. Lithii Benzoas, Lithii Bromidum, and Lithii Salicylas are unofficial.

Lithium salts are similar in action to the corresponding potassium salts, but on account of the affinity of lithium for uric acid and the ready solubility of lithium urate they have been preferred for the treatment of gout. It is probable, however, that in the blood the salts combine more readily with the acid sodium phosphate, and form no compound whatever with uric acid. They are diuretic, and render the urine alkaline, and for these reasons may be one of value in some cases. Large quantities of water are usually taken with lithium, and this in itself may account for some of the beneficial effect in cases of gout. Excessive doses or too prolonged administration of lithium salts may cause considerable depression, general feebleness, and stomach disturbance. A lotion of lithium carbonate (4 grs. to ℥j of water) has been used as a moist application to gouty joints and gouty ulcerations.

**Litho-.**—In compound words *litho-* (Gr. λίθος, stone) means relating to calculi, urinary, biliary, or other.

**Lithoclast.**—*See* LITHOTRITE.

**Lithodialysis.**—The solution of calculi in the bladder by drugs given by the mouth (of very doubtful efficacy) or by intravesical injections, or the division of vesical calculi into pieces small enough to be removed by irrigation or by forceps.

**Lithogenesis.**—The formation of calculi in the body.

**Lithokelyphopædion.** — An extra-uterine fœtus or lithopædion which has undergone calcification only in its envelopes and superficial parts (*Küchenmeister*).

**Litholapaxy.**—The crushing of a vesical calculus and removal of the fragments thus produced at one sitting.

**Lithonephrotomy.**—Incision of the kidney for the removal of a renal calculus. *See* KIDNEY, SURGICAL AFFECTIONS (*Stone, Treatment*).

**Lithontriptic.**—Medicinal means of dissolving urinary calculi, such as water, lithium, benzoic acid, piperazine, etc.

**Lithopædion.**—A fœtus, almost always extra-uterine, which has died and undergone calcification in whole or in part (lithokelyphopædion).

**Lithophone.**—An instrument for detecting the presence of a stone in the bladder by the production of sound when the two come in contact.

**Lithotomy.**—The operation of removal of a stone from the bladder by incision, lateral (perineal), median (perineal), suprapubic, or vaginal. *See* BLADDER, INJURIES AND DISEASES (*Calculus Vesicæ, Treatment, Lithotomy*).

**Lithotomy Position.**—The exaggerated dorsal posture, the thighs being markedly flexed on the abdomen. *See* GYNÆCOLOGY, DIAGNOSIS IN; CURETTAGE; UTERUS, MALIGNANT TUMOURS OF (*Vaginal Hysterectomy*); etc.

**Lithotripsy.**—The operation of crushing a calculus in the bladder. *See* LITHOTRITY.

**Lithotrite.** — The instrument used to crush a vesicle calculus, preparatory to its removal.

**Lithotrity.** — The operation of crushing a vesicle calculus into fragments small enough to pass away or be removed through the urethral canal; the crushing instrument used is called a lithotrite or lithotriptor. *See* BLADDER, INJURIES AND DISEASES OF (*Calculus Vesicæ, Treatment by Lithotrity*).

**Lithuria.**—The presence of uric acid, in excessive amount, in the urine. *See* URIC ACID; URINE, PATHOLOGICAL CHANGES IN (*Urinary Sediments*); GOUT; etc.

**Litmus Paper.**—Unsized paper which has been steeped in a solution of litmus (a blue pigment obtained from a lichen, *Roccella tinctoria*); the blue paper turns red in the presence of an acid; the paper which has been reddened with acid turns blue if put into an alkaline solution. These papers are used in the examination of urine, etc. *See* URINE, PATHOLOGICAL CHANGES IN (*Reaction*).

**Litten's Sign.**—The retraction of the lateral part of the chest (corresponding to the insertion of the diaphragm); there is lowering of the retracted part during inspiration, and rising during expiration; it is absent in pleural effusions, when there are pleuritic adhesions, etc.

**Littlehampton.** *See* THERAPEUTICS, HEALTH RESORTS (*English*).

**Little's Disease.** — The cerebral diplegia of infancy or congenital spastic paraplegia. *See* PARALYSIS (*Spastic, Infantile, Cerebral*).

**Littré's Colotomy.** — The opening into the colon in the left iliac region. *See* COLOTOMY.

**Littré's Glands.**—Racemose glands in the urethral mucosa.

**Littré's Hernia.**—Hernia of Meckel's diverticulum. *See* HERNIA (*Contents*).

**Litzmann's Obliquity.**—In cases of flat pelvis the head may enter the pelvic brim with the parietal bone which is posterior leading; this is Litzmann's obliquity; if the parietal bone which is anterior leads, the obliquity is called Naegelé's.

**Livedo Annulare.**—Lividity or mottling of the skin. *See* ERYTHEMA (*Of limited extent*).

**Liver.**—This is described in the following sections:—

1. **Physiology of.**
2. **Diseases other than those of "Tropical" origin.**
3. **Tropical Disorders, including Surgical Treatment.**

*See also* ABDOMEN, INJURIES OF (*Lesions of the Liver*); ABDOMINAL TUMOURS, DIAGNOSIS OF (*Tumours of Liver*); ACTINOMYCOSIS (*Distribution of Lesions*); ALCOHOL (*Chronic Alcoholism, Morbid Changes in Liver*); ALCOHOLISM (*Intoxication*); ALCOHOLISM (*Pathology*); APPENDIX VERMIFORMIS (*Appendicitis, Diagnosis from Abscess of Liver*); ASCITES (*Causation*); BED-SORES (*Causes, Liver Affections*); BRAIN, AFFECTIONS OF BLOOD-VESSELS (*Cerebral Thrombosis, Diagnosis, Pain in the Liver*); CHILDREN, CLINICAL EXAMINATION OF (*Abdomen, Liver*); EMBRYOLOGY, HUMAN; ENZYMES (*Diastatic Function of the Liver*); FŒTUS AND OVUM, DEVELOPMENT OF (*Nutrition of the Fœtus*); GALL-BLADDER AND BILE DUCTS, DISEASES OF; GLANDULAR FEVER (*Clinical Features, Enlargement of Liver*); GLYCOSURIA (*Alimentary, Cirrhosis of the Liver*); HÆMATEMESIS (*Causes and Source, Diagnosis*); HEART, AFFECTIONS OF MYOCARDIUM AND ENDOCARDIUM (*Effects of Cardiac Disease, Nutmeg Liver*); HEART, AFFECTIONS OF MYOCARDIUM AND ENDOCARDIUM (*Symptomatology, Enlargement of the Liver*); HYDATID DISEASE; INTESTINES, DISEASES OF (*Anatomical Considerations*); KIDNEY, SURGICAL AFFECTIONS OF (*Movable Kidney and Movable Liver*); KIDNEY, SURGICAL AFFECTIONS OF (*Hydronephrosis, Diagnosis*); LABOUR, OPERATIONS (*Induction of Labour, Indications*); LARDACEOUS DEGENERATION; LEPROSY (*Clinical Features, Lepra Tuberosa*); LUNG, TUBERCULOSIS OF (*Complications, Alimentary System, Liver*); MALARIA (*Diagnosis, Liver Abscess*); MEDIASTINUM (*Chronic Mediastinitis, Diagnosis, Cirrhosis of Liver*); MELÆNA (*Diagnosis*); OVARIES, DISEASES OF THE (*Ovarian Cysts, Symptoms and Diagnosis*); PANCREAS, DISEASES OF (*Chronic Pancreatitis, Diagnosis*); PHYSIOLOGY, FOOD AND DIGESTION (*Liver*); POST-MORTEM METHODS (*Examination of the Body Cavities, Liver*); PULSE (*Venous and Liver Pulsations*); SYPHILIS (*Visceral Syphilis, Syphilis in Children*); TOXICOLOGY (*Phosphorus Poisoning*); TRADES, DANGEROUS (*Lead Poisoning, Pathology*); TYPHOID FEVER (*Complications and Sequelæ*); UNCONSCIOUSNESS (*Auto-intoxications*).

## Physiology of Liver


OUTLINE OF STRUCTURE . . . . . 427
FUNCTIONS—
*General* . . . . . 428
*Carbohydrates* . . . . . 428
*Fats* . . . . . 429
*Proteids* . . . . . 429
*Bile-formation* . . . . . 430
INFLUENCE OF NERVES ON THE LIVER . 431


OUTLINE OF STRUCTURE.—The liver originates as a branching tubular outgrowth from the gut, and it must thus be regarded as primarily a digestive gland.

At first the tubules run in an irregular manner, but with the growth of the fibrous tissue they become massed into separate groups or lobules, with their closed extremities pointing inwards and their orifices opening into a network of ducts at the periphery of the lobule.

The original tubular character becomes lost, and the lumen of the tubules is represented by narrow spaces between the cells, the so-called bile capillaries. The liver-cells thus seem to lie in rows radiating outwards from the centre to the periphery of the lobule. Each cell is polygonal in shape, with one or sometimes two large, round, centrally-placed nuclei, and a protoplasm containing certain materials, varying in amount according to the condition of the animal. When engorged with these matters the cells are much enlarged and squeezed together; when free of these substances, as in starvation, they become smaller and more sharply defined.

One of the most obvious of the substances is *Fat*, in its characteristic globules. In many animals on a fatty diet this is very obvious.

*Glycogen* occurs dissolved in the cytoplasm, and it may be demonstrated by staining with iodine. It is very frequently confined to one side of the cells. When the organ is treated with alcohol the glycogen is precipitated in granules.

*Pigment* of a brownish colour, usually in granules, is also to be seen in the liver-cells, especially when destruction of red blood corpuscles is going on, and the presence of iron may be demonstrated by treating sections with hydrochloric acid and then with ferrocyanide of potassium.

According to Langley, what he calls "proteid granules" are to be seen in the liver-cells of the frog, especially in summer.

Minute channels passing into the protoplasm and communicating with the bile capillaries have been described, but the true *bile passages*, which commence as chinks between the liver-cells, form an anastomosing plexus of ducts between the lobules. These are lined by a cubical epithelium. They join together to form the larger bile ducts, and these present a columnar epithelial lining and a fibrous coat with non-striped muscular fibres in its substance. In many animals there is a diverticulum on the common bile duct, the *gall-bladder*, which has the same structure as the bile passages, but which, in some animals, has a few mucous glands, opening into it.

The *blood-supply* of the liver is twofold. The *hepatic artery* supplies the connective tissue of the organ, and the *portal vein* supplies the parenchyma, but between them is a very free anastomosis. Both vessels are carried in the fibrous tissue of the organ; and when such a piece of fibrous tissue is cut across, the large branch of the portal vein and the smaller branches of the hepatic artery, with one or two branches of the bile-ducts, are to be seen forming a *portal tract*. These two sets of vessels terminate in plexuses of capillaries between the lobules, and from these capillaries pass inwards between the rows of liver-cells, and end in a *central vein* which carries the blood from the lobules, and these central veins joining together form the *sublobular veins*, which by their junction make the *hepatic vein*, by which the blood is carried off to the inferior vena cava.

The *nerves of the liver* are non-medullated, and are derived from the cœliac plexus, partly from the vagi, partly from the splanchnic nerves.

Physiology.—1. *General.*—While the liver originates as an outgrowth from the gut, it soon acquires other relations, and although it never becomes so completely separated from the alimentary canal as do the thyroid and thymus glands, its main functions are connected with the general metabolism rather than with the digestion.

Early in intra-uterine life the ductus venosus bringing blood from the developing placenta opens up into a capillary net-work among the liver tubules, and the organ becomes permeated with sinuses through which the blood slowly streams, and in which the nucleated red corpuscles divide and multiply. About this time glycogen and fat, which have already appeared in the placenta, begin to be found in the liver-cells.

As the alimentary canal develops, blood is sent from it to the liver, and when at birth the placental circulation is stopped, and the animal is nourished from the intestine, the liver remains upon the main channel of absorption.

Both in intra- and in extra-uterine life the liver is the great regulator of the supply to the tissues of the proteids, fats, and carbohydrates from which the body gets its energy, and its action in this direction may be briefly summarised as follows:—

(1) It regulates the supply of sugar—
  (*a*) By manufacturing it from proteids when the supply of carbohydrates is cut off.
  (*b*) By storing it as glycogen when the carbohydrates are supplied in greater quantities than are required by the body, and afterwards giving it out as it is required.

(2) It regulates the supply of fat in many animals by storing any excess.

(3) It regulates the supply of proteid, acting along with the intestinal wall, by decomposing any excess and giving off the nitrogenous part as urea.

(4) It regulates the number of red corpuscles by breaking down the older corpuscles and decomposing and eliminating the hæmoglobin.

2. *Regulation of Supply of Sugar to the Body.*—That sugar is used in the tissues, and chiefly in muscle, as a source of energy is demonstrated by the large proportion of carbohydrates in the ordinary diet of man, and by the excretion of its great product of combustion—carbon dioxide—when sugar is taken. But although it is thus used in the tissues, its amount in the blood is not diminished when the supply from without is cut off. It must therefore be continually produced in the body; and the fact that, when proteids are given in the food, glycogen is formed from them in the liver, seems to indicate that in starvation this production of sugar from proteids is carried on in that structure. This production of sugar in the liver Bernard named its glycogenic functions. On account of the small amount of sugar present in the blood, and of the imperfections in the analytic methods at our disposal, the direct experimental evidence upon this point is by no means satisfactory. But the indirect evidence is sufficiently convincing, and in certain pathological conditions, such as diabetes, the production of sugar from proteids becomes very manifest.

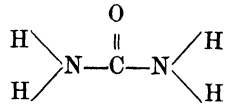
When the supply of carbohydrates in the food is excessive, the liver takes up the surplus sugar, and by synthesis and dehydration, probably effected only after the sugar has become part of the liver protoplasm, converts it into the polysaccharid glycogen, and in this form stores it for future use. There is evidence that this glycogen is in close chemical union with the living matter, and that the separation is only brought about at the death of the cell, or when it is being discharged. The various monosaccharids, *e.g.* glucose, levulose, galactose, can all be stored as glycogen; but the disaccharid lac-

tose, which is largely absorbed unchanged, is apparently not available for glycogen production. Not only is glycogen thus stored from the excess of carbohydrates taken in the food, but when large amounts of proteid are given, these are to a great extent split up and the non-nitrogenous part used in the formation of glycogen or of sugar. There is no evidence that fats are a source of sugar or glycogen in the liver, though recently attempts have been made to prove that they undergo such a change in phloridzin poisoning.

The manner in which glycogen is reconverted to sugar must be considered as unsettled. Bernard and others have described the process as due to a zymin in the liver, but other physiologists have been unable to accept this view, and believe that it is a process similar to the conversion of zymogen to zymin, and probably presided over by the nerves to the liver. In support of the former view, the fact that an amylolytic zymin can be extracted from the liver after death has been dwelt upon, while the latter view is supported by the fact that many agents which do not influence the action of zymins, *e.g.* quinine and methyl-violet, inhibit the conversion of glycogen, and that this conversion is accelerated by stimulation of the cœliac plexus.

3. *Regulation of Supply of Fat to the Body.*— Although the liver is not upon the channel of the absorption of fat from the intestine, in many animals it has a very special power of storing any excess of fats in the food. This function is perhaps best seen in the cod, and it is well marked in the cat and in the human subject. On the other hand, in the salmon, and in the pig and ox, the power of storing fat in the liver is very limited. The fat thus stored is given out when the supply from without is withdrawn. While the ordinary fats vary with the supply, lecithin is a constant constituent of the liver-cells, even in prolonged starvation. It has been suggested that this lecithin is the first step in the synthesis of inorganic phosphorus to the complex nucleins of the cells, and that the fats of the liver may act by combining with this phosphorus to fix it and prevent its excretion.

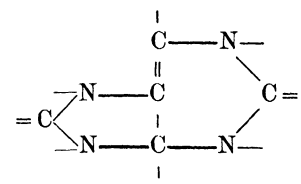
4. *Regulation of Supply of Proteids.* — The researches of Nencki, Sieber, and others have shown that any excess of proteid in the food is, in part at least, broken down in the wall of the intestine, and that the nitrogenous part is sent to the liver as ammonia compounds. Von Schroeder has shown that such ammonia compounds, by a process of synthesis, are built into urea—



During starvation the proteids of the body are broken down, the non-nitrogenous part is converted to carbohydrates, and the nitrogenous moiety is excreted as urea. Where this breaking down occurs is not known, whether in the muscles or in the liver, but the ultimate stage of the manufacture of urea takes place chiefly in the liver. This is shown by the fact that even partial destruction of the liver leads to an increase of the ammonia in the urine and a decrease of the urea. By what stages the formation of urea is brought about is not clearly known, but we do know that proteids readily break down into amido-acids and ammonia compounds, and that such compounds are changed into urea in the body.

But urea is not the only form in which the effete nitrogen of proteids is eliminated. In fœtal animals *allantoin* takes its place, while in birds and reptiles *uric acid*, with traces of hypoxanthin and xanthin, are the chief waste products. These substances are diureides—that is, they consist of two more or less modified urea molecules linked by an organic acid. Allantoin has as its linking bond glycoxylic acid, while the others have acrylic acid. Uric acid is one of a series of bodies formed by modifying the nucleus which Fischer has termed the Purin Nucleus—

Urea. Acrylic Urea.
Acid.



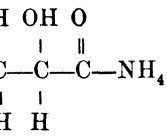
Oxy-purin is Hypoxanthin.
Dioxy-purin is Xanthin.
Trioxy-purin is Uric Acid.

By the introduction of amidogen, Aminopurin or Adenin is formed, and from this, Amino-oxypurin or Guanin is produced.

This series of diureides is formed when nucleins break down; xanthin, hypoxanthin, adenin, and guanin have thus been prepared, and although outside the body uric acid has not been so produced, the administration of nucleins leads to its increased formation within the body. In dogs allantoin is formed when nucleins or uric acid are given. There is no indication that the formation of uric acid in this way goes on in the liver, and, in fact, the observation that it may be produced by digesting leucocytes with blood in a stream of oxygen would tend to show that it goes on in other tissue. The uric acid and other purin bases so formed are all, in part at least, further changed to urea in the liver.

But there is another mode of formation of uric acid, which occurs in birds and reptiles, and, at least under certain conditions, in man. In

the former type of animals the ammonium lactate—



which in mammals is changed to urea, is converted to uric acid. Now when it is remembered that sarcolactic acid is hydroxy-propionic acid, and that acrylic acid, the linking bond in uric acid, is an unsaturated propionic acid, it is obvious that uric acid may be formed either directly on the road to the formation of urea or only very slightly off that road.

That uric acid in birds is formed in the liver is shown by Minkowski's observation that when the liver is removed lactate of ammonia takes its place in the urine. This production of uric acid must be carried out by a process of synthesis, since the amount of nitrogen is greater in uric acid than in lactate of ammonia, and that this synthesis is carried out as part of the metabolism of the liver protoplasm is indicated by the fact that in birds urea is changed to uric acid, and that in dogs uric acid is changed to urea and to allantoin.

Now in certain conditions of the liver in mammals the process of the elaboration of urea either stops to a greater or less extent at the stage of uric acid or by a slight modification is in part diverted to it, and thus the proportion of uric acid to urea rises. What these conditions are has not been fully investigated, but apparently maloxygenation of the blood and the action of various toxic substances in the liver are apt to induce this alteration in the metabolism.

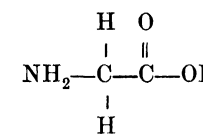
One proteid, with the breaking down of which the liver has a very direct relation, is hæmoglobin. This may be considered as a compound of about 96 per cent of a globulin, with 4 per cent of an iron-containing pigment, hæmatin. The liver-cells take up hæmoglobin, break it into its two components, and split the hæmatin into an iron-containing part which is retained for further use, and an iron-free portion which is excreted as the pigment of the bile. It also breaks down the globulin and forms urea, and thus when experimentally a large amount of hæmoglobin is set free in the circulation, the excretion of urea is proportionately raised.

The relationship of the liver to proteids and hæmoglobin leads to the study of its

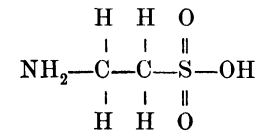
5. *Bile-forming Function.*—Formerly the formation of bile was considered *the* function of the liver, and it was only after Claud Bernard had discovered its glycogenic function that the secondary nature of bile production was recognised.

Bile is an alkaline, viscous, olive-yellow fluid. When secreted from the liver-cells it has a specific gravity of about 1010, and contains about 2 per cent of solids. When it has been some time in the gall-bladder, water is absorbed, and it becomes more viscous and the percentage of solids rises.

In freshly secreted bile the *inorganic salts* amount to less than 1 per cent. The principal salt is chloride of sodium. The chief constituent of the bile is the soda salt of *glycocholic acid.* A small amount of *taurocholic acid* is also present in human bile. These salts are alkaline in reaction. Glycocholic acid readily splits into cholalic acid, $C_{24}H_{40}O_5$, the constitution of which is not definitely known, and amido-acetic acid (glycocoll)—



Taurocholic acid yields cholalic acid and amido-ethane-sulphuric acid (Taurin)—



Since these both contain amidogen they must be derived from proteids. That these acids are formed in the liver and not merely excreted by it is shown by the fact that whereas when the bile-ducts are ligatured they appear in the blood, when the liver is excluded from the circulation they do not appear. They may be considered as the essential constituents of the bile, and it is their presence which gives to bile any action it may have in digestion. About 7 or 8 grams are excreted per diem.

The pigments *bilirubin*, *biliverdin*, and *biliprasin* are derived from the decomposition of the hæmatin of hæmoglobin, and they are increased when hæmoglobin is set free. The fact that they do not appear in the blood when the liver is excluded from the circulation shows that they are formed in the organ and not merely excreted by it. They amount to about half a gram per diem.

*Cholesterin*, $C_{26}H_{43}OH$, is a monatomic alcohol insoluble in water, but dissolved in the salts of the bile acids. The amount secreted in normal conditions is small. That it is not merely excreted by the liver is shown by the fact that when injected or given in the food it does not appear in the bile. It is probably formed from the cells of the bile passages, since researches carried on in Naunyn's laboratory show that when these passages are inflamed the cholesterin is increased. It is the chief constituent of gall-stone.

*Fats and Lecithin* are present in small amounts, and are derived from the liver-cells.

The viscosity of bile is due to the presence

of a nucleo-proteid, with small traces of mucin. These are formed in the bile-passages.

*The action of bile in digestion* is unimportant, since its exclusion from the intestine does not prejudicially affect either men or animals. Its only action is as an adjuvant to the pancreatic juice by dissolving the fatty acids set free, and thus favouring their absorption. When the bile does not enter the intestine, about 30 per cent of the fats escape absorption, and it is the presence of these which gives the peculiar white appearance to the stools in jaundice.

The very small importance of bile in digestion raises the question of how far it is to be regarded as a digestive secretion and how far as a waste product. The facts that its secretion begins before birth and is continuous during starvation, that its formation has no immediate relationship with the taking of food, that its pigment is derived from the decomposition of hæmatin and its chief constituents—its acids—from proteid disintegration, all strongly support the view that it is a waste product which has come to play a minor part in digestion. There is no evidence that the proteids of the food are excreted as the acids of the bile, and it must be concluded that they are derived from the proteids of the body generally, or more probably from the waste of the liver tissue itself.

*Excretion of Toxic Substances by the Liver.*—The study of bile secretion leads to the consideration of another function which the liver seems to perform. It has been found that it can take up pigments of various kinds and secrete them in the bile. It has also been shown that the salts of the bile acids are reabsorbed from the intestine, carried to the liver, and again secreted. Now, certain other substances are treated in the same way. Curare, when administered hypodermically, is a powerful toxic agent, but when taken by the mouth its action is not manifested. It appears to be absorbed, carried to the liver, and excreted in the bile. It seems to circulate in the portal circulation until gradually eliminated from the bowel, and thus is prevented from entering the general circulation. At one time it was supposed that snake venom, which is not toxic when swallowed, undergoes the same treatment, but Fraser has shown that bile has a destructive action upon it. Possibly ptomaines formed in the intestine are prevented from producing their prejudicial effect by being re-excreted by the liver and in this manner got rid of.

Influence of Nerves on the Liver.—The influence of the nervous system has been investigated on only two of the functions of the liver, the secretion of bile and the production of sugar.

The subject is much complicated by the fact that the nerves act upon the blood-vessels, and that both bile-production and sugar-production are influenced by the vascularity of the organ. Thus it has been shown that section of the splanchnic nerves, which causes dilatation of the blood-vessels, causes an increased flow of bile, while stimulation of these nerves causes a diminution in the secretion. Similarly, factors which dilate the vessels tend to cause a more rapid conversion of glycogen to glucose.

Apart from this indirect influence, there is so far no evidence that the secretion of bile is governed by the nervous system, while the facts that injection of pilocarpine, which increases the flow of saliva and of pancreatic juice, has no effect on bile secretion, and that atropine does not arrest it, seem opposed to the idea that there is any nervous mechanism directly involved. On the other hand, the flow of bile already secreted into the bile-passages is directly under the control of the nerves, and a discharge of bile into the duodenum is reflexly brought about when food is taken.

Recently a good deal of evidence has been forthcoming of a direct influence of the nervous system on the glycogenic function of the liver. Cavazzani has shown that stimulation of the cœliac plexus causes glycogen to be converted into sugar, and Butler states that stimulation of the peripheral end of the cut vagus increases the amount of sugar in the blood leaving the liver. This may, of course, have been due to vascular dilatation.

**Diseases of Liver**


**Anatomical Abnormalities** . . . 432
Liver . . . . . 432
Biliary Apparatus . . . . 432
Post-mortem Changes . . . 432
Acquired Deformities . . . 433
Displaced Liver . . . . 434
**Hepatoptosis** . . . . . 435
**Functional Disease** . . . . 437
**Diseases of—**
Hepatic Artery . . . . . 440
Hepatic Veins . . . . . 441
Lymphatic Vessels . . . . 441
**Chronic Venous Engorgement** . . 442
Hepatic Pseudo-Cirrhosis . . . 444
Hepatic Infarcts . . . . 445
**Biliary Cirrhosis** . . . . . 445
(*a*) Hypertrophic Biliary Cirrhosis . 445
(*b*) Obstructive Biliary Cirrhosis . 450
**Portal Cirrhosis** . . . . . 451
**Varieties of Hepatic Cirrhosis in Early Life** . . . . . 462
**Degenerations and Infiltrations** . . 462
Fatty Liver . . . . . 462
Lardaceous Liver . . . . 464
Pigmentary Change . . . . 464
Calcareous Infiltration . . . 465
Leukæmic Infiltration . . . 465
**Tumours of Liver** . . . . 466
Innocent Tumours . . . . 466
*Adenoma* . . . . . 466
*Angioma* . . . . . 468
*Lipoma* . . . . . 468

**Cysts of Liver** . . . . . 468
**Infective Granulomata** . . . . 469
TUBERCLE . . . . . 469
SYPHILIS . . . . . 472
LYMPHADENOMA . . . . . 477
ACTINOMYCOSIS . . . . . 477
**Malignant Disease** . . . . . 478
PRIMARY . . . . . 478
*Carcinoma* . . . . . 479
*Sarcoma* . . . . . 479
SECONDARY . . . . . 481
**Special Conditions characterised by Jaundice—**
ICTERUS GRAVIS . . . . . 483
ACUTE YELLOW ATROPHY . . . 483
WEIL'S DISEASE . . . . . 487
**Jaundice of Phosphorus Poisoning** . 60

ANATOMICAL ABNORMALITIES

ANATOMICAL ABNORMALITIES . . . 432
*Liver* . . . . . 432
*Biliary Apparatus* . . . . 432
SOME POST-MORTEM CHANGES . . . 432
ACQUIRED DEFORMITIES . . . . 433
*Tight-laced Liver, or Corset Liver* . 433
*Tongue-shaped Lobe* . . . . 433
DISPLACED LIVER—*Congenital* . . . 434
*Acquired* . . . 434

In general transposition of the viscera the liver is on the left side of the body, and the left lobe is the larger. Cases sometimes occur in which the liver is in its normal situation, but the left lobe is large and the right lobe small—transposition of the lobes without *situs transversus*. Sometimes one lobe—often the left—is found to be dwarfed out of all recognition, and to be represented only by a small appendage attached to the other; this is probably a result of some interference with the blood-supply early in life. When the left lobe is practically absent the stomach is more widely uncovered, and the gall-bladder appears to arise from the left border instead of from the under surface of the liver.

Small accessory lobes, of about the size of the last joint of the forefinger, are fairly common; they are more frequently seen on the under surface of the right lobe of the liver. If they become markedly pedunculated they might form small "accessory livers." The Spigelian lobe has been observed to be curiously pedunculated.

Very rarely small detached pieces of hepatic substance have been found in the falciform or other peritoneal ligaments of the liver, "accessory livers," or hepatic "rests." It is noteworthy how rare hepatic "rests" are, as compared with accessory suprarenal bodies and splenunculi.

Extensive lobulation of the liver is a condition sometimes met with; there may be as many as 16 lobules; this lobulation is, I believe, not homologous with the fœtal lobulation of the kidney, but pathological, and due to some pre-existing morbid process, such as syphilis, or possibly tuberculosis, perihepatitis, or coarse cirrhosis.

*Abnormalities in the Gall-Bladder and Bile Ducts.*—Complete absence of the gall-bladder—the normal condition in the horse and other animals—is sometimes seen in men; this must be distinguished from early pathological obliteration of an existing gall-bladder, such as occurs in congenital obliteration of the ducts.

On the other hand, two gall-bladders, each with a cystic duct, have been seen in the same liver (Purser); a bifid gall-bladder has also been described.

An hour-glass contraction of the gall-bladder is generally associated with gall-stones, and is probably a secondary change. In cases where the fundus of the gall-bladder projects beyond the anterior margin of the liver, the terminal portion, being somewhat dilated, may appear to be separated from the rest by an hour-glass constriction. Personally, I regard the hour-glass gall-bladder as an acquired and not a congenital change.

Direct communications between the gall-bladder and the liver (hepaticocystic ducts) are sometimes found to occur in men. The gall-bladder has been found attached to the left lobe instead of to the right. Considerable variation may exist in the arrangement of the larger bile ducts. Sometimes the two hepatic ducts do not unite until comparatively close to the duodenum. The common bile duct may open quite separately from Wirsung's duct into the duodenum.

*Post-mortem Appearances of the Liver.*—A few words may be said about certain common though striking post-mortem appearances of the liver.

The surface of the liver, where it has been in contact with the stomach or colon, very commonly shows dark purple stains. These stains are quite superficial and are due to the action of gases, among them sulphuretted hydrogen, which diffuse through from the colon and stomach, and meet with iron in the liver; as a result, some compound like sulphide of iron is manufactured.

Irregular white areas on the surface of the liver are seen in cases of fevers and other infections, and show congestion and degeneration changes (Hanot). This appearance was formerly thought to be merely due to mechanical pressure exerted after death in laying out the body.

*Cloudy Swelling.*—After death, one, if not the most frequent, change in the liver is that of cloudy swelling. The organ is enlarged, heavier than natural, and looks as if it had been boiled, being dull, somewhat more opaque, and paler in colour than normal. These changes are due to cloudy swelling or parenchymatous

inflammation of the liver-cells set up by the toxines of numerous diseases. The changes which are shared by other organs, such as the kidneys and myocardium, are especially well seen in pneumonia. In this disease the enlargement of the liver is very considerable. Long ago Bright thought that the pneumonic lung materially depressed the liver, but it is clear that any increased hepatic dulness below the costal arch is due to cloudy swelling and congestion.

*Foaming Liver.*—Under certain conditions the liver becomes infected, shortly before or at the time of death, with the bacillus capsulatus aerogenes, with the result that it becomes a spongy mass of gaseous cysts.

### Acquired Deformities of the Liver

*Effect of Tight-Lacing on the Liver.*—Modifications in the shape of the liver due to tight-lacing and corsets are, of course, commoner in women, but considerable deformity of the liver may be produced in men by the pressure of a tight belt or strap.

The effect of tight-lacing on the liver varies to a certain extent with fashion, or, in other words, with the position of the waist. Hirtz, who has studied the changes produced in the liver by tight-lacing in great detail, finds that the deformities of the liver may be divided into two main types, though mixed or transitional forms may occur.

(1) The liver is flattened from above downwards, the right lobe is elongated, and at the point where it passes over the right kidney is thinned, so that below this point there is a constriction or movable lobe attached by a fibrous hinge, so to speak, to the rest of the liver. To this condition the term partial hepatoptosis has been applied. Sometimes the left lobe is similarly prolonged downwards, and may have a constriction lobe attached to it. Frerichs figures a capital example. It has indeed been thought that such a constriction lobe is more likely to give rise to symptoms from the ease with which it would press on the stomach, pylorus, pancreas, and large nerve plexuses. But it is doubtful whether this is so. Clinically there is a close resemblance between these constriction lobes of the right lobe and the tongue-shaped or Riedel's lobe usually described as occurring in special association with gall-stones. From impaired nutrition and diminished resistance morbid changes are more likely to occur in the constriction lobes than in the remainder of the liver; thus fibrosis or gummata may be confined to them. On the other hand, the whole of the liver, constriction lobe included, may be uniformly affected by cirrhosis.

(2) The liver lies high in the abdominal cavity, and is much thicker above than below. It is curved across the spinal column so that the left lobe may touch or even overlap the spleen. The fossa for the inferior vena cava is exaggerated; while the lower margin of the right lobe is compressed by the belt or corset, and shows local perihepatitis and underlying atrophy. The circumferential pressure may throw the upper surface of the liver into folds. These furrows are often seen on the convexity of the right lobe, and have been thought to be due to indentations of the ribs, or to be due to the impress of folds, or hypertrophied columns, of the diaphragm, since they are said to occur in cases where respiration has been difficult during life, such as bronchitis and emphysema. A constriction lobe may be found attached to the right lobe, but is not a constant feature as in the previous form, and is never present in connection with the left lobe.

*Clinical Significance.*—Tight-laced livers may and often do coexist with dyspepsia, which no doubt partly depends on the abdominal or gastric embarrassment produced by the pressure of a tight corset. Very frequently, however, the patient has no symptoms in any way referable to a tight-laced or corset liver. The chief interest about a tight-laced liver is that the constriction lobe may, when accidentally detected, be easily mistaken for something more important, such as a floating kidney, a tumour of the pylorus or transverse colon, a dilated gall-bladder, cysts of the pancreas or of the mesentery, or, in extreme cases, a fibromyoma of the uterus, an ovarian tumour, or appendicitis. The connection between the constriction lobe and the main part of the liver, as has already been pointed out, is sometimes very thin, and may therefore give a resonant note on percussion. Its actual continuity with the remainder of the organ is, therefore, difficult to make out.

### Tongue-like Lobes

Synonyms: *Linguiform lobe, Riedel's lobe, Partial hepatoptosis, Floating lobe.*—This condition is very much the same as the constriction lobes that have been described in the corset liver. The association of these tongue-like lobes with distension of, or with the presence of calculi in, the gall-bladder appeared extremely intimate to Riedel, after whom these lobes have sometimes been called, and was therefore regarded by him as corroborative evidence of gall-bladder disease in any case of doubt. It has indeed been thought that these tongue-like lobes are only produced by gall-bladder distension, inflammation, or calculi; but this is probably too exclusive a statement. Tight-lacing must also be taken into account, especially as it may lead to bending and twisting of the cystic duct, and so to partial obstruction of the outflow of bile from the gall-bladder, thus disposing to catarrh and so to cholelithiasis.

The elongation of the right lobe downwards carries with it the gall-bladder, and since this

is often affected with calculous cholecystitis, the abnormal lobe is frequently the site of pain.

As in the case of the constriction lobes of the corset liver, this abnormal lobe may be connected with the liver only by a thin bridge of fibrosed hepatic substance, and may easily be regarded as some form of abdominal tumour, such as a floating kidney, a tumour of the pylorus or colon, and the other tumours mentioned in the section on tight-laced liver.

From repeated attacks of congestion the floating lobe is very often structurally altered, and shows fibrous increase.

The subjects of floating lobes are usually women of mature years, and, as has been already said, gall-stones are frequently found in association. M'Phedran, however, has described a case in a baby aged eleven months, and is inclined to regard these lobes as of developmental origin.

The floating lobe is freely movable, and may be tender on palpation. The symptoms referred to it are a feeling of heaviness and dragging in the hypochondrium, pain sometimes like biliary colic, and in all probability due to gall-stones. It should, however, be pointed out that it may be associated with a floating kidney.

The tongue-shaped lobe is rarely diagnosed clinically, and is usually only discovered when an exploratory laparotomy is undertaken. The condition has been known to disappear after associated disease of the gall-bladder has been removed, and this is the rational and most satisfactory treatment.

In some recorded cases the floating lobe has been successfully fixed by sutures to the abdominal wall, or even removed with relief of the symptoms referred to its presence. But such radical measures are not really necessary, since it is doubtful whether serious symptoms are ever due to the floating lobe apart from associated disease of the gall-bladder.

### Displaced Liver

This condition must be distinguished from a movable or displaceable liver, which is considered in the next section on hepatoptosis, and from the various enlargements of the liver.

Displacement of the liver may be due to causes that are—

(1) Congenital.

(2) Acquired.

*Congenital Malposition or Displacement of the Liver is rare.*—When the diaphragm is defective or represented only by a membranous partition, the liver may project upwards into the thoracic region inside a thin pouch. In 12 cases of true congenital diaphragmatic pouches collected by Jaffe, 8 were on the left side. The left lobe of the liver has been found in these pouches, and its displacement naturally gives rise to tilting of the organ.

From congenital defect of the abdominal muscles the liver may project under the skin either at the umbilicus or between that point and the xiphoid cartilage. When it occurs at the navel it is spoken of as hepatomphalos, when elsewhere as congenital ectopia, or hepatocele. The tumour is dull on percussion, and can be reduced unless it contracts adhesions to the wall of the hernial sac.

*Acquired displacements of the liver* may be due to very various causes.

In angular curvature of the spine, the liver, like other organs, may be very considerably displaced. In rickets, the deformity of the thorax may account for some of the increased extent of the hepatic dulness below the costal arch. The liver is generally regarded as being enlarged in rickets. This is partly real, partly apparent, as explained above. In tight-lacing, narrowing of the lower part of the thorax often squeezes the liver downwards.

In traumatic diaphragmatic hernia the liver may pass through the rent into the cavity of the pleura; a constriction has in some cases been found between the intrapleural portion and the rest of the liver; under these conditions the distal portion may become very congested. Acquired diaphragmatic hernia is very much commoner on the left side; it may contain the left lobe of the liver.

Among the intrathoracic conditions that give rise to displacement of the liver the most important are pleural effusion and pneumothorax on the right side. The right lobe is depressed, and the liver then tends to be rotated from right to left on its antero-posterior axis. Similar conditions on the left side, or a large pericardial effusion, will depress the left lobe of the liver. Emphysema, and very extensive infiltration of the lung with new growth, also push the liver downwards, but only to a comparatively slight degree. In mediastinal tumour there is no displacement of the liver, unless, as not infrequently occurs, there is a large pleural effusion at the same time.

*Abdominal Conditions leading to Displacement of the Liver.*—Generally speaking, abdominal conditions that displace the liver do so in an upward direction, but occasionally the liver may be depressed or rotated from side to side. Ascites, flatulent distension of the intestines, congenital dilatation of the colon, or the presence of large abdominal tumours, such as ovarian cysts, uterine fibromyomata, etc., push the liver and diaphragm up, and thereby encroach very seriously on the capacity of the thorax. The convexity of the diaphragm may then be on a level with the third rib. When this upward displacement is very considerable the liver may largely or even entirely cease to be in contact with the anterior abdominal wall, and undergoes a very striking alteration in its relation to other organs. The anterior surface travels backwards and becomes posterior, while

the inferior surface comes to look forwards and upwards instead of downwards and backwards. This is due to the liver moving upwards on a transverse axis running through its connection with the inferior vena cava, which is relatively a fixed point.

Occasionally coils of intestine or the colon may intervene between the liver and the anterior abdominal wall, thus displacing the liver backwards. In acute yellow atrophy complete disappearance of the liver dulness is largely due to the flabby liver allowing resonant bowel to come between it and the abdominal parietes.

A subdiaphragmatic abscess, especially one between the liver and the diaphragm, such as results from rupture of a suppurating focus on the convexity of the liver (suprahepatic abscess), or a hydatid cyst lying between the liver and the diaphragm, may depress the liver.

As the result of inflammatory adhesions the liver may be pulled downwards towards the pelvis. In very rare instances a wandering liver has become fixed by peritoneal adhesions in an abnormal position, such as the right iliac fossa (Richelot).

Dilatation and distension of the stomach or of the left part of the colon will rotate the liver on its antero-posterior axis towards the right.

A displaced liver is, as a rule, not more movable than one in its normal position. It differs from a wandering liver in this respect, and also in the fact that it cannot be replaced in its normal position, while in addition a definite cause for its displacement is often forthcoming.

Symptoms that might be referred to a displaced liver, such as weight and pain in the right hypochondrium, are generally thrown into the shade by those of the condition responsible for the displacement.

The various forms of enlargement of the liver—fatty, lardaceous, leukæmic, new growth, abscess, cirrhosis, etc.—must be differentiated from a displaced liver by a careful physical examination of each individual case.

## Hepatoptosis


*Definition, etc.* . . . . . 435
*Etiology* . . . . . 435
*Causation* . . . . . 435
*Physical Signs* . . . . . 436
*Symptoms* . . . . . 436
*Diagnosis* . . . . . 437
*Treatment* . . . . . 437


Synonyms: *Wandering Liver; Movable Liver; Ptosis, Dislocation, Prolapse of the Liver.*

*Definition.*—The liver, being unduly movable in a downward direction, leaves its normal position and appears as an abdominal tumour.

*Historical.*—Heister as long ago as 1754 published an account of an autopsy, illustrated by a plate, showing this condition; but Cantani is credited with the first clinical recognition of wandering liver, in 1865. Attention has been largely directed to this curious anomaly by the numerous contributions of Glenard, who has recently collected 80 examples.

*Introductory.*—A movable liver must be distinguished from mere displacement of the organ by pleural effusion, etc., and from the constriction or floating lobes that have been termed partial hepatoptosis. It is probable that what were really only floating lobes have often been described as wandering livers or complete hepatoptosis.

A wandering liver is analogous to a wandering spleen; both the organs are normally "floating," viz., supported by the abdominal viscera and tethered by peritoneal ligaments. The term "floating" is therefore unsuitable as descriptive of an abnormally movable liver, though it is applicable to nephroptosis. When these normally "floating" organs become unduly and spontaneously movable, they may be said to be "wandering."

*Etiology.*—The female sex are chiefly affected; out of 80 cases collected by Glenard 73 were in females, Graham in 70 cases found 56 in women, while in 30 cases seen in private practice Max Einhorn records 21 in women. The majority of patients are over forty years of age; cases in early life are very rare; Freeman in 494 autopsies on children records 4 instances of hepatoptosis.

Repeated pregnancies, abdominal distension, and other causes leading to a relaxed and pendulous condition of the abdominal parietes, dispose to hepatoptosis. Tight-lacing may, by interfering with the healthy tone of the abdominal muscles, indirectly dispose to hepatoptosis; but it plays a minor part, and its chief manifestations have been already referred to (p. 433).

*Causation.*—An unduly movable liver may be part of general abdominal ptosis or Glenard's disease, already described. (See "Enteroptosis.") On the other hand, hepatoptosis may exist without universal visceroptosis, or only in association with one of its manifestations, such as floating kidneys.

A considerable amount of discussion has taken place as to the essential factors in the production of a movable liver. From a general survey it appears that the necessary conditions are:—

(1) Diminution in the Intra-abdominal Tension.—This allows the supporting pad of intestines to fall away from the liver, and permits that organ to drop down towards the pelvis. The causes which lead to lowering of the intra-abdominal pressure are numerous; among them may be enumerated repeated pregnancies, ascites, abdominal distension, sedentary occupations, tight-lacing, and an enfeebled state of health; all of these tend to impair the healthy tone of the muscles of the abdominal wall, and to pro-

duce a pendulous state of the abdomen. Though undoubtedly a very important factor, diminution of the intra-abdominal pressure is probably not of itself sufficient to induce complete hepatoptosis.

(2) Failure in the Suspensory Apparatus of the Liver.—This is probably somewhat subordinate in importance to diminished intra-abdominal pressure, but is a necessary condition to the production of hepatoptosis.

The liver is suspended in its place by the following means:—(*a*) By the Suspensory and Coronary Ligaments.—It has been thought that they may be congenitally absent or deficient; this may be so in a few isolated instances, but it is so exceptional an occurrence that it cannot be maintained that congenital abnormalities in these peritoneal folds have any real bearing on the production of movable liver. It is much more probable that relaxation of these ligaments is acquired and due to nutritional defects, such as may well be responsible for atony of the abdominal parietes. It has been suggested that fatty and generative changes are induced in the ligaments. (*b*) By the Inferior Vena Cava.—Faure has laid great stress on the importance of the inferior vena cava in keeping the liver in contact with the diaphragm. Probably defective general nutrition will lead to relaxation of its controlling influence in the same way as in the case of the suspensory ligaments.

The *immediate cause* of hepatoptosis may be found in sudden injuries or strains, the displacement then resembling a traumatic dislocation; in other cases repeated efforts, such as coughing, vomiting, sneezing, or prolonged straining, have been invoked. In many cases no definite determining cause is forthcoming.

Forms of Hepatoptosis.—The liver is not simply displaced downwards; its shape is considerably modified, and it frequently becomes rotated on one or more of its axes.

The liver settles down and becomes flattened out so as to lie like an apron over the intestines; the superior or diaphragmatic surface tends to become anterior, and the inferior surface to become posterior. The organ hangs down from its attachment to the inferior vena cava, and is so thinned and elongated that it may reach the right iliac fossa. The liver is thus anteverted or, in other words, rotated forwards on its transverse axis, and its long axis becomes vertical instead of oblique.

Frequently the liver is in addition rotated on its antero-posterior axis. From the fact that the most fixed point of the liver is its attachment to the inferior vena cava, the right lobe, which is the heavier as well as the more movable, descends more freely than the left lobe, which may be the only part left in contact with the diaphragm. The position of the liver thus becomes oblique, the right lobe being tilted downwards and depressed.

A further change may be met with, viz., rotation of the liver on its long or vertical axis, so that the anterior surface looks towards the right and the posterior surface to the left; in rare instances the liver is said to be rotated in the opposite direction, so that the anterior surfaces face towards the left and the posterior towards the right.

There are thus at least three forms of movable liver.

The flattened and elongated liver frequently shows a line of constriction running transversely across its anterior surface. This is probably due to the pressure exerted by the costal margin and tight-lacing. The line of constriction may become so thin as to contain little but blood-vessels between the two layers of somewhat thickened peritoneum. A constriction lobe is then found attached to the rest of the liver by a kind of tendinous hinge.

Generally speaking, the liver itself is healthy, but in a certain number of instances the existence of concomitant disease, such as gall-stones or cirrhosis, has been recorded.

Occasionally a movable liver contracts adhesions, and becomes fixed in an abnormal part of the abdomen; in consequence it cannot be replaced in the normal position, and presents great difficulties in the way of a correct diagnosis.

*Physical Signs.*—There is an abdominal tumour which is displaceable, and can be returned to the normal position of the liver. It resembles the liver in outline, size, and in descending on respiration, while examination of the normal situation of the liver shows that the organ is partially or entirely absent. The normal liver dulness is replaced by resonance, and the right hypochondrium is sunken.

When the organ is very freely movable, it not only moves with change of posture, descending when the patient sits up, and tumbling to the left when he is turned on that side, but it can be rotated on its vertical axis, an exaggeration of the displacement induced by a dilated stomach or colon under ordinary conditions of hepatic stability. The relaxed condition of the abdominal walls often renders the liver visible as a rounded tumour on the right side, about the level of the umbilicus, extending down into the right iliac fossa and approaching the pelvis. Other forms of visceroptosis, such as a floating kidney, displaced stomach or uterus, etc., may be present.

*Symptoms.*—The onset is generally insidious and attracts no attention; in a few instances it is suddenly manifested by pain, like that of biliary colic, or a feeling of something giving way, and suggests traumatic dislocation.

Cases of hepatoptosis sometimes present no symptoms, and the condition is only discovered accidentally. On the other hand, patients may complain of one or more of the following symptom groups:—

1. Pain and a feeling of weight in the right hypochondrium are very common: the traction exerted by the liver may be transmitted through the diaphragm, pericardium, and cervical fascia to the root of the neck, and be felt there. These symptoms are made worse by exertion and are relieved by lying down.

2. Symptoms imitating Cholelithiasis.—Sometimes attacks of colic, which may resemble biliary colic, are met with; they may be explained as due to torsion of the bile ducts, or possibly to concomitant cholelithiasis. In other cases intestinal colic occurs, and is probably due to ptosis of the colon. Jaundice rarely occurs in hepatoptosis; it may be the result of torsion, gall-stones, or be of a simple catarrhal nature.

3. Symptoms imitating Cirrhosis.—Ascites and hæmatemesis have been met with, and have been referred to twisting of the portal vein; in like manner œdema of the feet has been explained by kinking of the inferior vena cava. These symptoms are infrequent.

4. Symptoms referable to the respiratory system are somewhat uncommon. In some instances dyspnœa and asthmatic symptoms appear to depend on hepatoptosis (Max Einhorn). Persistent cough has been found to be associated with displaceable liver, and to be cured when appropriate treatment — the application of a bandage—for the latter condition was adopted (Vène).

5. In a considerable proportion of the cases the symptoms are not due to hepatoptosis alone, but to complete or partial visceroptosis. Thus the neurasthenic or hysterical manifestations, so commonly associated with visceroptosis, may be most prominent features. In other instances the symptoms are referable to the stomach and intestines, such as dyspepsia, vomiting, constipation, flatulence, mucous colitis, etc. Though congestion of a displaceable liver may play a considerable part in the production of these functional disturbances, they are intimately connected with visceroptosis (*vide* Glenard's disease, article "Enteroptosis"). In other instances the symptoms may be chiefly due to a floating kidney, while in others leucorrhœa, menorrhagia, etc., due to visceroptosis, are complained of.

*Diagnosis.* — The presence of a movable tumour resembling the liver, taken into conjunction with evidence that the liver is absent from its normal position, are the essentials in the diagnosis. But care must be taken in distinguishing it from a floating lobe with or without a distended gall-bladder, from simple downward displacement due to factors like pleural effusion (see p. 434), and from enlargement of the liver depending on malignant disease or other causes. One of the most frequent mistakes seems to be to regard as a floating kidney what is in reality a prolapsed liver; the former condition is, of course, more familiar, and may complicate hepatoptosis. In a case of doubt an attempt should be made to replace the movable tumour in the hepatic region, while careful note should be made of the extent of the liver dulness and the relation of the intestines to the tumour.

Hepatoptosis has been mistaken for various other abdominal tumours and conditions, such as omental or renal tumours, carcinoma of the stomach, hydatid cysts or malignant disease of the liver, gall-stones, and even for ovarian cysts.

*Treatment.*—A suitable bandage, belt, or apparatus should be applied to the abdomen so as to support the abdominal walls and keep the liver in its proper place. Massage and electricity have been employed in order to strengthen the abdominal muscles and improve their tone.

Diet is a matter of importance; generally speaking, the subjects of hepatoptosis are feeble, wanting in tone, and require good and liberal feeding to improve their state of nutrition. When there is decided corpulence, the amount and quality of the food will require careful consideration by the medical attendant.

Symptoms of neurasthenia and nervous debility should be treated on appropriate lines. Constipation should be met by massage and purges, so as to prevent congestion of the liver and accumulation of bile in the gall-bladder.

When all other measures fail to relieve the symptoms, and the patient is quite incapacitated, the advisability of surgical interference must be considered. Various methods of fixing the liver permanently in its normal position, or "hepatopexy," have been employed, such as suturing the prolapsed liver to the costal arch or anterior abdominal wall, or fixing the round ligament to the abdominal wall while at the same time promoting adhesions between the convexity of the liver and the diaphragm. The operation has in many cases been successful, but should only be undertaken when all other means have failed. The objections to its adoption are (i.) that it hardly affects the underlying conditions responsible for the prolapse of the liver, though it may counteract them, and (ii.) that the wound made at the time of the operation may subsequently become the site of a hernial protrusion. This is more likely to occur in old women with pendulous abdominal walls than in younger patients. In any case, an abdominal belt should be worn after the operation.

### Functional Disease of the Liver

In the section on the physiology of the liver the functional importance of the liver has been fully explained, and it is clear that any failure in the discharge of these numerous duties must be followed by definite symptoms.

Functional disturbance of the liver is undoubtedly a frequent occurrence in common with functional disturbance of the other organs

in the body. The only questions are—Whether this functional disturbance is primary, and whether the inadequacy is independent of any structural change?

So many conditions were formerly described as due to functional disease of the liver, many of which had little or nothing to do with that organ; and this idea became firmly implanted in the lay mind, and therefore so recklessly employed, that the tendency at the present time among medical writers is rather to ignore the subject or to deny the existence of primary functional disease of the liver. This swing of the pendulum to the opposite extreme is due to the knowledge that the symptoms ascribed to functional disease of the liver can in great part be explained as due to other factors, such as indigestion, constipation, auto-intoxication, or to subacute congestion of the organ; the latter condition being often secondary to intestinal disturbance, or to an excessive ingestion of proteid food combined with an imperfect excretion of waste products. In other words, the hepatic disturbances formerly regarded as primary functional insufficiency are in the vast proportion of cases dependent on morbid processes occurring elsewhere, or to morbid changes on the liver itself.

Thus, to consider the symptoms commonly referred to functional disease of the liver. The distaste for food, dyspepsia, and flatulence are the expression of gastro-intestinal catarrh set up by poisonous or unsuitable food. The icteric tint of the conjunctivæ, the muddy skin, and the constipation or diarrhœa, are explained by the spread of the gastro-duodenal catarrh to the biliary papilla, and the slight obstruction to the flow of bile thus induced; or possibly to catarrh of the minute intra-hepatic ducts set up by poisons absorbed from the alimentary canal and subsequently excreted into the ducts. The headache, giddiness, muscæ volitantes, malaise, muscular debility, mental depression, and irritability are due to the local action on the nervous system of poisons absorbed from the alimentary canal. These toxic bodies are either produced in such quantities that the liver fails to filter them off, or more probably they act on the liver-cells and impair their vitality and function; in either case the general circulation becomes flooded with toxic bodies.

The piles, the feeling of weight in the right hypochondrium, and shoulder pain, point to hepatic congestion, or even slight hepatitis, brought on by the advent to the liver of digestive products in excessive amount and probably of altered (*i.e.* toxic) quality. This state of hepatic congestion is especially apt to be set up in patients who have suffered from malarious fevers in the Tropics (*vide* Tropical Liver).

Nevertheless there can be no doubt that in some instances morbid results are traceable to the functional disturbance of the liver, without its being always possible to determine satisfactorily that this disturbance is secondary. Thus in diabetes mellitus there is an excessive activity of the glycogenic function, while in alimentary glycosuria the liver is unable to discharge efficiently its function of stopping the sugar brought to it by the portal vein. Thus diabetes mellitus and glycosuria may be regarded as diseases due to functional disturbance of the liver, but these are not the conditions ordinarily spoken of as functional liver disease.

It has recently been urged that puerperal eclampsia is in many cases due to hepatic insufficiency, and that the renal symptoms are secondary to a primary hepatic toxæmia. It is supposed that during pregnancy auto-intoxication results from retention of the menstrual discharge, and that in women who inherit a diminished hepatic activity and resistance the liver fails to rise to the occasion, and that as a result of this insufficiency the blood becomes loaded with poisons.

As has already been admitted, functional disorder of the liver no doubt is responsible for many symptoms. The difficulty in regard to the subject is to prove that the functional disturbance is primary in the liver, and not secondary to disease or morbid factors elsewhere. The discussion is not a mere academic exercise, but has a practical bearing on the treatment. Thus, if it were thought that there was a primary failure of hepatic activity, the rational course would be to stimulate the organ; whereas, if there was an underlying and primary factor elsewhere, this should be attacked.

The difficulties about the recognition of primary functional disorders of the liver may be best explained by considering some examples of the conditions of which it has been, or might be, supposed to be the cause.

Lithæmia was described by Murchison as a condition of innate defect of power, often hereditary, in the liver, in virtue of which its healthy functions are liable to be deranged by the most ordinary articles of diet. As a result of this hepatic insufficiency, uric acid instead of urea was produced in the liver and turned out into the blood. Among the results of lithæmia Murchison enumerates such different conditions as dyspepsia, constipation, gout, urinary calculi, biliary calculi, and acute and chronic renal disease.

This theory is very far reaching, and offers an explanation of gout; in fact, many of the manifestations of lithæmia are those of irregular gout.

This theory of lithæmia depends on the assumption that the production of uric acid instead of urea, occurs in the liver as the result of imperfect oxidation. But more recent in-

vestigations show that the production of uric acid is certainly not confined to the liver, but takes place elsewhere in the body, being especially dependent on changes in lymphoid tissue and on leucocytosis, the uric acid being derived from nuclein obtained from the leucocytes; while according to Latham, Kolisch, and Luff the formation of uric acid occurs in the kidney. It is, therefore, too narrow a view of the faulty metabolism of proteid material that results in an excessive production of uric acid, to say that it depends on functional disorder of the liver to the exclusion of the rest of the body.

An increase in the urates and uric acid in the urine is found in hepatic disorder such as cirrhosis, congestion, and in conditions such as fever, where the liver-cells might be affected by toxins in the blood, and so incapable of performing their proteolytic function. But even granting for the moment that the formation of urea under normal conditions, and of uric acid in excess in abnormal states, takes place in the liver, this process is due to the functional disturbance that is not primary, but due to structural modification, and secondary to morbid processes elsewhere.

It is, however, erroneous to conclude that even as a secondary effect the faulty metabolism takes place in the liver rather than elsewhere in the body. For in grave disease of the liver, such as cirrhosis or extensive malignant disease, the increase in the amount of uric acid in no way corresponds with the view that its formation depends on an imperfect metabolism of proteids by the liver; while in cases of fever and toxæmia the remainder of the body, being equally thrown out of gear, is liable to faulty metabolism, one of the results of which might well be the production of uric acid instead of urea.

Habitual high arterial tension and its accompaniments, such as migraine, might be thought to depend on a failure of the liver to stop and destroy the poisonous bodies that are carried to it from the intestines. The liver undoubtedly exerts this important function of protecting the body from auto-intoxication; but it is difficult to prove that failure in the discharge of this duty leads to high arterial tension, since in cases of extensive disorganisation of the liver, for example in cirrhosis, hepatic insufficiency must exist, but the arterial tension is low and not raised. It is much more likely that high arterial tension is, like gout, due to some general disorder of metabolism of the body.

In cases popularly described as "biliousness," or "torpid" liver, where there is indigestion, some hepatic pain, headache, slight icteric tingeing of the conjunctivæ, with a deficiency of colouring matter in the fæces, the explanation is gastro-duodenal catarrh with slight catarrhal jaundice, and not a primary diminution in the secretion of bile. In these cases it is possible either that there is catarrhal swelling of the biliary papilla in the duodenum, or that, as the result of gastro-intestinal indigestion, poisonous products are carried to the liver, and then, when excreted into the bile ducts, set up a certain amount of catarrh in the small intrahepatic ducts. This leads to reabsorption of the bile with the poisons contained in it, which pass into the general circulation and poison the body as a whole. Sir Thomas Brunton has ingeniously shown that the proverbial bitter taste of the bile is probably pathological, and due to the presence of poisons absorbed from the bowel and then excreted into the ducts, and that in health the bile is tasteless.

But because the ingenious conception of lithæmia and other time-honoured views as to primary functional disease of the liver do not commend themselves in the light of later knowledge, it does not follow that hepatic insufficiency or inadequacy is a negligible factor.

It must be borne in mind that the liver, like other organs, must vary greatly in different individuals as to its functional activity and reserve power, and an amount of food products that could be satisfactorily dealt with by the liver in one individual would in another be beyond the scope of the liver. This difference in the inherent powers of the liver in different persons is analogous to the differences in their muscular and mental power, and the less powerful should not be described as suffering from functional disease of their muscles or brain because they fail to accomplish the work that their better developed companions have no difficulty with.

If, therefore, an individual consumes an amount of food that is excessive for his powers of digestion, fermentation and auto-intoxication will result. These poisons will impair the functional activity of the liver, and as a result the poisons and the products of digestion will be allowed to pass into the general circulation and give rise to the various toxic manifestations already referred to.

From what has gone before, it is evident that the well-known symptoms ascribed to a torpid or inactive liver are chiefly due to factors which secondarily interfere with the functional activity of the liver, and not to a primary inadequacy of the organ.

### Secondary Functional Disorder of the Liver

The symptoms have already been referred to on pp. 437–439, and a few lines as to their treatment will now be given.

*The treatment* of the symptoms of secondary hepatic inadequacy must therefore be directed to the causes, and not to the liver itself.

In the first place, the alimentary canal should

be cleaned out; this is most satisfactorily effected by the use of the old-fashioned blue pill and haustus sennæ. The mercury drives out the bile out of the gall-bladder, unloads the bile ducts, and by sluicing the common duct tends to remove the causes of catarrh of its lower end. At the same time it acts as an intestinal antiseptic, and inhibits excessive fermentation, and then puts a stop to further auto-intoxication. The purgative action of these two remedies removes the poisons from the body.

Plenty of water should be taken so as to wash out the poisonous products from the circulation and stimulate the functional activity of the kidneys.

During the existence of symptoms a liquid diet, of which milk is the staple, should be adopted, while alcohol in any form should be rigidly avoided.

In the second place, the patient should be warned to avoid the forms of food likely to set up intestinal catarrh and fermentation. The articles of food that must be avoided as indigestible will, of course, vary in individual cases; but, generally speaking, the following should be avoided—concentrated and highly spiced soups or essences, pork, duck, hare, game, made dishes, sauces, melted butter, tea cakes, crumpets, cheese, and much proteid food. Alcohol should be taken in great moderation, and chiefly in a diluted form, as whisky, or claret and water; while beer, porter, champagne, sherry, port should be prohibited.

Exercise is important, and should be of an active nature; horse exercise is the best, bicycling useful, and walking the least effective. The skin should be got to act, and Turkish baths are useful for this purpose. A visit to spas such as Carlsbad, Marienbad, Vichy, or Ems, and a regulated course of treatment there, will benefit the patient.

As to the prognosis, the digestive disturbances which give rise to these symptoms are the same that lead to cirrhosis; and, indeed, the symptoms of "functional disease" of the liver may in some instances be the early manifestations of cirrhosis.

## Diseases of the Hepatic Artery

Diseases of Hepatic Artery—
- Arteriosclerosis . . . . 440
- Thrombosis . . . . . 440
- Embolism . . . . . 440
- Aneurysm . . . . . 440
- Enlargement . . . . . 441

Hepatic Veins—
- Thrombosis . . . . . 441
- Embolism . . . . . 441
- Stricture . . . . . 441
- Suppurative Inflammation . . 441

Lymphatic Vessels—
- Glands in Portal Fissure . . 441

Arteriosclerosis. — A certain amount of change occurs in the hepatic artery in arteriosclerosis, and no doubt may dispose to the rare events—thrombosis and aneurysm.

It is, however, noticeable that arteriosclerosis of the hepatic artery does not lead to any change in the liver comparable to a granular kidney.

In cases of hæmochromatosis there is endarteritis of the hepatic artery, while in the neighbourhood of gummata and in syphilitic disease of the liver endarteritis obliterans is found.

Thrombosis. — Thrombosis of the hepatic artery has very rarely been noted, and is a pathological curiosity. Lancereaux refers to a rather doubtful case in a man aged sixty-five, who died with arteriosclerosis and gangrene of the feet.

Embolism.—Like thrombosis few cases are on record, probably because the condition of the hepatic artery is rarely investigated.

As a result of embolism of the main trunk necrosis of the liver has been noted both in man (Chiari, Lancereaux) and in animals; in a case that I had an opportunity of seeing with Dr. C. Ogle, of embolism of the bifurcation of the artery, the liver showed white infarcts, but was not completely necrosed. Experimentally, ligature of the hepatic artery shows the flow of bile, and thus disposes to cholangitis. Septic emboli give rise to multiple abscesses. Emboli of the small branches occur in melanotic sarcoma.

Aneurysm of the Hepatic Artery.—There are about thirty recorded examples of hepatic aneurysm.

*Situation.* — Aneurysms may occur on the main trunk of the artery, at the bifurcation, or on its main branches, in which case the aneurysm may be either in the substance of the liver or outside it, just in the portal fissure. Symmetrical aneurysms on the two branches of the hepatic artery have been met with. An aneurysm has been found in the wall of an hepatic abscess; this lesion was evidently due to ulceration attacking the artery from without, and is comparable to the production of an aneurysm in the walls of vomicæ in pulmonary tuberculosis. Small intrahepatic aneurysms may occur in great numbers in the rare condition of periarteritis nodosa.

*Causation.*—They may be due to embolism, arteriosclerosis, and in rare instances to traumatism.

*Symptoms.*—Pain resembling that of biliary colic is generally present, while jaundice due to pressure on the bile ducts may further increase the clinical resemblance to cholelithiasis. In other cases the aneurysm ruptures either into the bile duct, peritoneum, or duodenum; on the latter event it may resemble a duodenal ulcer.

*Diagnosis* is very difficult. In the absence of pulsation the symptoms suggest gall-stones. If

pulsation is present, the commoner condition of aortic aneurysm would be more likely to suggest itself.

ENLARGEMENT of a compensatory nature of the hepatic artery is seen in some cases of new growth, cirrhosis, and in thrombosis of the portal vein of some standing.

### DISEASES OF THE HEPATIC VEINS

THROMBOSIS of the hepatic veins is somewhat rare, and is hardly likely to be diagnosed correctly during life.

*Causes.*—It may be secondary to changes in the liver, such as the extension of new growth, the softening down and discharge of adenomata (*vide* p. 467) into the veins, or the spread of inflammation from a focus in the liver to the walls of the vein. Thrombosis may be secondary to stricture of the trunks of the hepatic veins (*vide infra*). In rare instances thrombosis may spread from a parietal clot not completely obstructing the inferior vena cava, or be secondary to obliteration of that vessel. In exceptional instances it is met with as part of a widespread thrombotic process.

*Results.*—A condition of chronic venous congestion or nutmeg liver with the rapid development of ascites follows thrombosis of the hepatic veins. Occasionally the stagnation thus induced may set up thrombosis of the portal vein.

EMBOLISM.—Embolism of the hepatic veins can only occur when the embolus travels against the blood stream and enters the hepatic veins from the inferior vena cava, or, in other words, be retrograde. Retrograde embolism of veins is very rare; when it occurs it is more frequently seen in the hepatic veins, since they are not protected by valves, and are so close to the heart that fragments of growth or thrombus may drop into their orifices either from the inferior vena cava or the heart and superior vena cava. Welch, in his article on embolism, quotes examples of fragments of new growth being found in the hepatic veins in cases where the primary growths were in the abdomen and thyroid body.

It seems probable that in cases of cranial suppuration with secondary abscesses in the liver without any abscesses in the lungs, the micro-organisms may drop down the jugular vein, superior vena cava, right auricle and inferior vena cava into the orifices of the hepatic veins, and so infect the liver. The production of retrograde embolism probably depends on the temporary stagnation or reversal of the direction of a blood flow. Thus if the thrombus was passing up the inferior cava and a violent expiratory effort or cough occurred at the moment when it was opposite the openings of the hepatic veins, the embolus might be carried into the liver.

STRICTURE AND STENOSIS.—Stricture may be due to the contraction of adhesions around the hepatic veins near their entrance into the inferior vena cava. This may be due to changes starting in or outside the liver. Gummatous inflammation may spread to the walls of the vein and set up peri- and endo-phlebitis, the latter leading to narrowing of the lumen (endophlebitis obliterans). This may occur in congenital or in acquired syphilis. Syphilis seems a probable factor in the production of most of the recorded cases of this rare condition. Probably chronic inflammation due to other causes may bring about a similar narrowing of the hepatic veins. The hepatic veins may be pressed upon from without by new growths, tumours, etc., and so be narrowed.

The symptoms referable to stricture of the hepatic veins are much the same as those of thrombosis of the veins, to which it may give rise.

SUPPURATIVE INFLAMMATION is more likely to spread to the hepatic veins in hepatic abscess, etc., than to the branches of the portal vein, since the latter are more protected by Glisson's capsule. In suppurative phlebitis of the hepatic veins secondary abscesses in the lungs and general pyæmia are, of course, likely to follow

### DISEASES OF THE LYMPHATIC VESSELS OF THE LIVER

Very little is known about the morbid conditions of the lymphatic vessels of the liver. They are affected when tuberculosis and lymphadenoma attack the liver, and can hardly escape in acute cholangitis and pylephlebitis. In tuberculous and chronic peritonitis and perihepatitis the inflammatory process spreads inwards from the capsule, possibly by means of the lymphatics, for some little distance.

New growth may sometimes be seen working its way into the liver against the lymph stream along the lymphatics of the portal fissure; more commonly the glands in the portal fissure become infected secondarily to a growth in the liver, the infecting cells travelling in the normal direction along the lymphatic vessels.

Distension of the lymphatic vessels in the portal spaces occasionally occurs from pressure; it has also occurred in hepatoptosis from torsion of the lymphatics around the bile duct.

In diabetic lipæmia I have seen the lymphatics of the portal spaces graphically mapped out by the contained fat.

THE LYMPHATIC GLANDS IN THE PORTAL FISSURE.—Any enlargement of those glands is of importance, inasmuch as pressure may thus be exerted on the bile ducts and jaundice set up. Thus it has been thought, but probably without sufficient reason, that the jaundice which in rare instances occurs in the roseolous stage of syphilis may be due to swelling of the glands in the portal fissure. Enlargement of the portal glands may occur in lardaceous disease and leukæmia, but cannot be credited with producing jaundice

or ascites by compression of the bile duct or portal vein in these diseases.

Enlargement of the portal lymphatic glands may be due to various conditions, chiefly inflammatory, inside the liver, such as abscess, pylephlebitis, suppurative cholangitis, tubercle, hypertrophic biliary cirrhosis, and primary carcinoma.

As already mentioned, new growth may extend into the portal fissure along the lymphatic vessels against the flow of lymph, and occasionally infiltration of the portal lymphatic glands may be secondary to carcinoma in the peritoneal cavity; when this has occurred jaundice may result.

### Chronic Venous Congestion of the Liver

Chronic Venous Engorgement of Liver—
Etiology . . . . . 442
Morbid Anatomy and Histology . 442
Physical Signs and Symptoms . 443
Termination and Prognosis . . 444
Diagnosis . . . . . 444
Treatment . . . . . 444
Hepatic Pseudo-Cirrhosis . . . 444
Infarcts . . . . . 445

Synonyms: *Nutmeg Liver, Cardiac Liver, Cyanotic Atrophy, Red Atrophy, Hepatic Asystole.*

This condition is almost always secondary to obstructive heart disease, especially of the mitral valve, or lung lesions, such as chronic bronchitis, emphysema, and some forms of pneumoconiosis. The symptoms due to the hepatic condition are, as a rule, merely added on to those of the primary disease; in some instances, however, the former are more prominent than those of the primary lesion, and to these cases the term "hepatic asystole" has been applied.

Etiology.—Any causes that lead to backward pressure and tricuspid regurgitation will produce chronic engorgement of the inferior vena cava, the hepatic veins, and their branches—the sublobular and intralobular veins. Mitral stenosis, dilatation of the left ventricle, mitral regurgitation from whatever cause, and obstruction to the pulmonary circulation, such as emphysema, are the common causes of tricuspid regurgitation and chronic venous engorgement of the liver. Tricuspid stenosis is rare; when it does occur it is always combined with, and secondary to, mitral stenosis. When it is present the hepatic engorgement is very marked.

It is possible that tumours or new formations, such as a hydatid cyst, gumma, or cicatrices, might compress the inferior vena cava between the entrance of the hepatic veins and the right auricle. Another conceivable cause is kinking of the inferior vena cava, from displacement of the heart caused by the presence of a large pleural effusion. Obstruction at the orifices of the hepatic veins due to cicatricial contraction of gummatous tissue or to syphilitic change in the walls of the veins—both of them rare lesions (*vide* p. 441)—may induce a nutmeggy condition of the liver. Lastly, new growths or hydatid cysts in the substance of the liver may produce local areas of chronic venous engorgement.

Morbid Anatomy.—The liver is usually somewhat enlarged, though not so much as in life. Its size depends on the amount of blood in it; further, the organ is much more distensible in life than after death, when its protoplasm coagulates or enters into rigor mortis. In advanced cases the liver-cells undergo atrophy, and the liver tends to become smaller. Externally it is of a mottled purple colour, and the subcapsular veins, seen as slight depressions in its surface, show up from atrophy of the surrounding liver substance. Sometimes there is subcapsular fibrosis (replacement fibrosis), which must be distinguished from perihepatitis. When ascites has existed for some time the capsule may be opaque. In rare cases chronic peritonitis spreads from an adherent pericardium to the capsule of the liver; the condition then becomes chronic universal perihepatitis.

On section the appearance is like that of a cut nutmeg; the sublobular veins and their branches—the intralobular veins—are dilated, and being full of blood, appear as dark purple spots or streaks corresponding to their transverse or longitudinal section. Apart from these venules the liver-cells, being stained with bile and infiltrated with fat, appear of a yellowish-white colour. This contrast gives rise to the name "nutmeggy" liver. This nutmeggy appearance may be fine or coarse; the latter condition is less characteristic.

The hepatic veins are dilated and their walls opaque and somewhat thickened, the liver tissue around being somewhat atrophied and compressed. Oppinet has suggested that the incidence of hepatic asystole or marked hepatic phenomena in a case of morbus cordis is determined by an anatomical condition of the hepatic veins at their entry into the inferior vena cava, which is congenital in origin and fairly common. But it seems more natural to regard these changes as secondary to backward pressure.

Occasionally there is some sporadic fibrosis of the liver, and it has been thought that chronic venous congestion causes cirrhosis. It is true that as the result of atrophy of the liver-cells the fibrous tissue becomes more prominent, but genuine cirrhosis is not due to chronic venous engorgement of the organ pure and simple. The state of chronic portal congestion may, and often does, lead to intestinal catarrh, and by the consequent formation of poisons some cirrhosis in the liver might be expected much more often than is actually

the case. In long-standing cardiac disease a considerable amount of alcohol is often given, which again might lead to cirrhosis. On the other hand, dilatation of the heart due to alcoholic excess may induce chronic venous engorgement in a liver already cirrhotic. Still, with all these possibilities the liver is, as an actual fact, very seldom genuinely cirrhotic in chronic venous engorgement.

HISTOLOGY. — The intralobular veins are dilated, and their capillaries are two or three times larger than normal from distension with blood. This dilatation spreads outward through the lobule as the condition of passive engorgement becomes more marked.

The liver-cells in the centre of the lobule are atrophied from pressure and malnutrition, inasmuch as their supply of oxygen is curtailed by the venous stagnation. They degenerate and contain hæmatoidin, an iron-free product, derived from the red blood corpuscles. This pigmentation must be distinguished from the infiltration of the cells in the peripheral zone of the lobule with hæmosiderin, an iron containing pigment, seen in pernicious anæmia. The cells in the peripheral parts of the lobule undergo fatty change. As a result of the atrophy of the liver-cells the supporting fibrous framework of the liver becomes more prominent, and in some instances sporadic cirrhosis is seen. This fibrosis may be especially marked directly underneath the capsule of the liver: to the naked eye this gives an appearance not unlike that of universal chronic perihepatitis.

PHYSICAL SIGNS. — The liver is enlarged, uniformly smooth, and tender. The tenderness is due to the distension and stretching of the capsule. The size of the liver varies considerably from time to time, and depends on the condition of the right side of the heart; efficient cardiac treatment may, therefore, rapidly have a marked effect.

In a small percentage of cases the liver pulsates with each beat of the heart. In 235 cases of tricuspid regurgitation it was present 15 times, and in 87 cases of tricuspid stenosis on 8 occasions (Pitt). True expansile pulsation, compared to that of an accordion, is best felt with one hand in the right loin and the other over the anterior surface of the liver. It is due to the blood being driven into the hepatic veins with each beat of the heart. It should be remembered that not uncommonly the liver receives a jog from the contraction of a dilated or hypertrophied right ventricle, but there is no expansion of the organ. Similarly, in rare cases pulsation may be communicated to the liver from an abdominal aneurysm. In expansile pulsation the blood regurgitates more easily into the left lobe, which, therefore, pulsates more freely.

The enlarged liver may push up the right leaflet of the diaphragm, and lead to some collapse of the case of the right lung, with dulness and absence of breath sounds. In some instances pleural effusion may occur.

Sometimes as a result of infection there may be some acute perihepatitis, with friction audible or even palpable over the liver.

The urine is concentrated, high coloured, and lithatic. It may contain excess of urobilin, which has been regarded as a sign of hepatic insufficiency (Hayem). There is sometimes albuminuria without any gross lesion of the kidney, due in all probability to chronic venous congestion impairing the vitality of the epithelium covering over the glomerular tufts. As a result, albumin is allowed to leak into the cavity of Bowman's capsule. Alimentary glycosuria has in rare instances been noticed.

Auto-intoxication is favoured in several ways:—

(1) The liver being ill-nourished, from venous stagnation impeding the advent of arterial blood, does not destroy poisons absorbed from the alimentary canal so completely as in health. Toxic bodies, therefore, pass into the general circulation.

(2) Portal congestion favours the manufacture of toxic products in the intestines; hence poisons in increased quantity are carried to the liver.

(3) Chronic venous engorgement of the kidneys interferes with proper renal excretion.

*The symptoms* referable to chronic venous engorgement of the liver are heaviness and discomfort in the right hypochondrium. Definite pain may be met with when perihepatitis is implanted on the engorged liver. The chronic portal engorgement gives rise to slow and feeble digestion, loss of appetite, dyspepsia, flatulence, and tympanites; while gastro-intestinal catarrh is readily set up. The condition of the alimentary canal interferes with assimilation, and the patient's general nutrition is impaired; this is especially the case in growing children in whom mitral disease with backward pressure on the portal system may be considered as a wasting disease.

Extension of catarrh to the biliary papilla may occur. The slight icteric tint of the conjunctiva and skin, so characteristic of advanced mitral disease, is due either to this cause or to slight cholangitis of the small intrahepatic ducts. This jaundice is slight and not due to complete obstruction. Occasionally a terminal infection, leading to acute degenerative changes in the liver-cells and icterus gravis, may carry the patient off. Orthopnœa and dyspnœa are mainly dependent on the primary lesion, but in hepatic insufficiency there may be dyspnœa of a uræmic type due to auto-intoxication.

Œdema of the feet and ascites are common accompaniments of this hepatic condition; in 235 cases of tricuspid regurgitation œdema

occurred in 200 and ascites in 140 (Pitt). The ascitic fluid is usually straw-coloured, but has been noted to be chyliform.

*Termination.* — Death is commonly due to increasing cardiac failure, or to some terminal infection setting up pneumonia, pleurisy, etc. Infection may fall on the liver itself, and give rise to acute degenerative changes in the liver-cells and icterus gravis.

*The prognosis* depends on the character of the primary cause; when the chronic venous engorgement of the liver ensues in the course of heart or lung disease of old standing, the outlook is naturally bad. If secondary to more acute dilatation, the outlook is more hopeful.

*Diagnosis.* — When the cardiac lesion is definitely recognised no difficulty arises; but in the cases described as hepatic asystole, where attention is focussed on the liver, the condition may be thought to be one of cirrhosis, or possibly malignant disease, with secondary cardiac failure. The smoothness of the liver, the absence of dilated veins on the abdominal wall, and of any splenic enlargement, and the effect of cardiac tonics, are in favour of passive congestion of the liver and against cirrhosis. The smoothness of the surface, the slighter degree of enlargement, the diminution in size produced by appropriate cardiac treatment, together with absence of severe pain and cachexia in nutmeg liver, will usually prevent any difficulty in the diagnosis from malignant disease of the organ.

*Treatment* should be directed to the primary lesion, whether cardiac or combined pulmonary and cardiac disease. Digitalis is the most efficacious drug, and may be combined with or replaced by strophanthus in cases of mitral stenosis. A pill containing digitalis, squills, and mercury is a valuable compound, and may be given at the same time that citrate of caffeine is administered by the mouth.

The hepatic engorgement may be successfully treated by purgatives, such as the old-fashioned blue pill and haustus sennæ, or by the administration of 2 to 4 drachms of sulphate of magnesia in hot water after an abstinence from liquid for some hours (Matthew Hay's method). Paracentesis of the abdomen may be necessary.

For insomnia, hypodermic injection of morphia is the most satisfactory remedy; if there be respiratory distress from bronchitis its administration is contraindicated, and paraldehyde or chloralamide should be tried.

The diet should be simple and nutritious, and not contain too much fluid, as this would tend to aggravate the often already water-logged condition.

### Hepatic Pseudo-Cirrhosis

Synonym: *Pericardial Pseudo-Cirrhosis.*

Under this title a number of cases have been described that are practically only chronic venous engorgement of the liver. Pick describes the clinical aspect as being rather that of cirrhosis, inasmuch as ascites is a prominent feature, while œdema of the legs is slight or may be absent. The pathological change is adherent pericardium, and not valvular disease of the heart, with subsequent circulatory disturbance in the liver, which is in a state of nutmeggy atrophy, showing some increased fibrosis without any perihepatitis.

The adherent pericardium may be the result of rheumatic, or more rarely tuberculous inflammation; in the latter case the liver may be also tuberculous. The adhesions dilate the right auricle, inferior vena cava, and hepatic veins, and thus render hepatic asystole permanent.

The condition is thus one of exaggerated nutmeg liver, and the stress of the backward pressure falls on the peritoneal cavity: the veins of the legs thus suffer less from chronic engorgement, and œdema of the feet is not induced so readily as in ordinary cardiac lesions. It is possible that at the time of the primary pericarditis inflammation spreads to the mouths of the hepatic veins, and by weakening their walls leads to dilatation and to a freer entry of blood into them, and that this condition, once established, remains permanently. It is thus possible to explain the connection of adherent pericardium with nutmeg liver and marked ascites, accompanied by less prominent œdema of the legs. I have examined some of these cases, expecting to find an extension of fibrosis from the adherent pericardium along the hepatic veins into the substance of the liver, but have never found any perivenous fibrosis, though the inner walls of the hepatic vein and inferior vena cava are opaque and thickened as is commonly seen in cases of backward pressure.

The liver shows marked chronic venous congestion (ramose atrophy) with irregularly scattered areas of fibrosis. There are signs of hyperplasia of the connective tissues, and limited areas exactly like multilobular cirrhosis; but, taken as a whole, the amount is scanty, and large areas may be quite free from fibrosis. The condition may be summed up as advanced chronic venous congestion with sporadic cirrhosis. There may be a thin layer of cirrhosis immediately under the capsule, forming a kind of second or inner capsule for the organ, and looking to the naked eye like perihepatitis (*cp.* nutmeg liver, p. 442).

*Prognosis and Results.*—When the condition of hepatic pseudo-cirrhosis has become established the prognosis is very bad.

Tuberculous peritonitis may supervene as a secondary result; this was proved to be the sequence of events in a case recorded by Nachod, where laparotomy a year before death proved the absence of tubercle at that time. Secondary tuberculous infiltration of the portal spaces may then occur. The term cardio-tuberculous cir-

rhosis has been applied to cases where advanced chronic venous congestion and tuberculosis of the liver are combined. These cases, which are chiefly met with in children, are associated with more advanced tuberculous disease elsewhere, especially in the peritoneum and pleura. These conditions of hepatic pseudo-cirrhosis and cardio-tuberculous cirrhosis are closely allied both to nutmeg liver and to the cases of general peri-hepatitis secondary to adherent pericardium. Clinically, the chief difference from nutmeg liver is the absence of any signs of cardiac valvular disease. The treatment, however, is that of chronic venous engorgement of the liver, viz., cardiac tonics and diuretics. The treatment suitable for cirrhosis is of no use in these conditions.

### Infarcts in the Liver

Infarction of the liver is rare; but appearances resembling infarcts in other organs undoubtedly occur, and are probably less infrequent than is thought. Lazarus-Barlow has collected 32 examples—of these 28 were hæmorrhagic and 4 anæmic. They resemble pulmonary apoplexies, and differ from infarcts in other organs in several ways. Thus in both the lung and liver there is a double blood-supply—the bronchial and pulmonary arteries, and the hepatic artery and portal vein respectively; like pulmonary apoplexies, hepatic infarcts are usually hæmorrhagic, do not show coagulation necrosis, or project above the surface when recent, and are not succeeded by depressed cicatrices, thus contrasting with the typical infarcts in the spleen and kidneys, which are possessed of end arteries. For these reasons it might be convenient to speak of these appearances in the liver as "pseudo-infarcts," inasmuch as they are not in the strict sense of the term infarcted (stuffed or swollen).

Infarcts in the liver have been met with most often in association with portal thrombosis or embolism of the branches of the portal vein. Obstruction of the intrahepatic branches of the portal vein by new growth, and embolism or thrombosis of small intrahepatic branches of the portal vein, may also appear to be a cause; while combined portal and hepatic vein thrombosis (Pitt), embolism of the hepatic artery (Ogle, Chiari), severe traumatism, and retrograde embolism of the hepatic veins, have been found in isolated instances. Infarcts of the liver have been described in Cirrhosis and in Nutmeg Liver (Bonome). None of these conditions, however, are essential to or necessarily followed by infarction of the liver; some other factor, possibly a toxæmic state, is requisite for the formation of the infarction.

The hæmorrhagic infarcts resemble nævi to the naked eye, and are not raised above the surface of the organ. The capillaries are dilated and the liver-cells atrophied, but not necrosed or involved in the process of coagulation necrosis seen in infarcts elsewhere.

The anæmic infarcts resemble, only they are more sharply defined, the anæmic patches often seen in the liver in infectious disease. The capillaries are empty.

Infarction of the liver has no clinical aspects, and is only of pathological interest.

### Biliary Cirrhosis

1. Hypertrophic Biliary Cirrhosis . 445
2. Obstructive Biliary Cirrhosis . . 450

This condition may conveniently be considered under two distinct heads:—

(1) Hypertrophic biliary cirrhosis.
(2) Obstructive biliary cirrhosis.

### Hypertrophic Biliary Cirrhosis

Synonyms: *Hypertrophic Cirrhosis with Chronic Jaundice, Hanot's Disease, Biliary Cirrhosis proper.*

It is sometimes spoken of as hypertrophic cirrhosis. This is to be avoided, since it is likely to lead to confusion as there are several other kinds of large cirrhotic livers; in common or portal cirrhosis the organ is often much enlarged, a fatty cirrhotic liver is of very considerable size, and the pigmented cirrhotic liver in hæmochromatosis is also entitled to the adjective hypertrophic.

Definition.—The disease is characterised by chronic jaundice, fever, absence of ascites, enlargement of the liver and of the spleen; it usually occurs in young persons. There is no gross obstruction to the larger bile ducts; histologically the cirrhosis is more monolobular than in ordinary portal cirrhosis.

History.—Although the condition was recognised by Requin in 1846, by Todd eleven years later (1857), and by Hayem (1874), it did not attract any attention until Hanot (1875) sharply struck out the disease in his thesis on *Hypertrophic Cirrhosis with Chronic Jaundice.* In 1893 Kiener suggested that the disease should be called Hanot's disease. Somewhat different forms of hypertrophic biliary cirrhosis have been described of late years in France, by Hayem, and by Gilbert and Fournier, and Gilbert and Castaigne, and discussion has arisen as to the channel by which the cause of the disease reaches the liver.

Of late years the opinion has been growing that the description given by Hanot was too crystallised, and that few cases conformed to the rigid type he erected. It must, however, be admitted that there is a very distinct difference between common cirrhosis and the condition to be described as hypertrophic biliary cirrhosis. Transitional forms between them are met with just as there are between the arteriosclerotic kidney and that of chronic

parenchymatous nephritis; but it would be incorrect to assume that they are different manifestations of a process that is essentially one and the same.

ETIOLOGY.—*Age.*—It is commonest between the ages of twenty and thirty, and is rare after forty, thus contrasting with common cirrhosis, in which the average age is about forty-eight years. A number of cases are met with in young children; Gilbert and Fournier have described a special juvenile type.

*Sex.*—In children the incidence of the disease falls equally on the two sexes; but apart from the juvenile cases, it appears that males are more often attacked. In Schachmann's 26 cases only 4 were females.

HEREDITY.—The disease is sometimes met with in several members of one family when exposed to the same conditions. In Brahmin infants around Calcutta a form of cirrhosis described as biliary is very common, and is especially apt to attack members of the same family; thus, as many as 14 children of the same parents have died of it one after another.

In this country Dreschfeld has met with the disease in two brothers, and Osler has had a similar experience in America.

It is interesting to note that in other members of the same family, who have no other manifest signs of the disease, the spleen may be found to be enlarged; this is analogous to the loss of knee-jerk in apparently healthy members of a family containing some children affected with hereditary ataxia.

ALCOHOL.—The antecedents of patients with hypertrophic biliary cirrhosis sometimes include heavy drinking, but there is no reason to regard alcoholism as related to the disease in the same way as it is to common cirrhosis. It may safely be said that alcoholic excess does not protect against biliary cirrhosis, but on the contrary disposes to infection by reducing the resisting power of the body as a whole, and of the liver in particular. In the case of biliary cirrhosis in Brahmin infants, alcohol can play no part. Of two brothers whose cases were recorded by Dreschfeld, one was a hard drinker, while the other was temperate. Boix has recently put forward the view that the infection is introduced into the body in water, and it has been thought that cold and damp houses favour the occurrence of the disease.

Malaria in like manner has been an antecedent condition in some cases, but in the great majority of instances this can be put out of court.

There is no evidence that syphilis causes the disease.

The disease has been noticed to start after typhoid fever (Boinet) in a few instances.

Hanot originally regarded the initial lesion as being a catarrhal inflammation of the small bile ducts. Such a lesion might originate in the minute ducts, and be due to a poison reaching them by the blood, as in experimental poisoning by toluylenediamine; in other words, be a descending cholangitis. The condition of the liver would then be a local manifestation of a general infection. In favour of this infective origin for hypertrophic biliary cirrhosis are the following facts:—

(1) The frequency of fever.

(2) The considerable splenic enlargement which indeed may precede, or be more marked than that of the liver.

(3) The existence of leucocytosis.

(4) Granular enlargement not only in the portal fissure, but sometimes in distant parts of the body.

The enlargement of the spleen, which may precede and be more prominent than the hepatic enlargement, is best explained as due to an infective agent in the blood, which, at the same time that it leads to changes in the liver, settles down in the spleen, and there multiplies and produces poison. It is possible that the poison thus poured into the portal vein sets up a secondary portal cirrhosis on the top of the already existing biliary cirrhosis, and thus accounts for the mixed type of cirrhosis so often found histologically in the livers of long-standing cases of biliary cirrhosis.

The alternative view is that hypertrophic biliary cirrhosis is due to a local infection of the bile ducts from the duodenum—an ascending cholangitis. According to this theory it would be analogous to broncho-pneumonia following bronchitis of the larger tubes. Gilbert and Fournier regard it as an ascending infection from the intestine, and due to the prolonged action of the colon bacillus. The enlargement of the spleen is regarded as secondary to the local and primary infection of the liver, and due to micro-organisms or their poisons absorbed from the infected bile ducts.

Against the view that it is an ascending infection might be urged the comparative infrequency of dyspepsia as an antecedent symptom, and the fact that a catarrhal condition of the duodenum is not found at the autopsy. The fact that the spleen is sometimes found to be enlarged before the liver, and may even be larger than the liver, is also against this theory, and in favour of the primary factor being a general hæmic infection.

On the whole, it seems more probable that hypertrophic biliary cirrhosis is due to a hæmic infection of a chronic nature leading to inflammatory changes in the liver, than that it is an ascending infection of the bile ducts from the duodenum.

Congenital obliteration of the bile ducts (*vide* vol. iii. p. 372), which is associated with very marked monolobular cirrhosis of the liver, can be regarded as due to a poison circulating in the blood, which, when excreted into the small

bile ducts, set up a descending cholangitis. This cholangitis leads to union of the inflamed surfaces of the larger ducts, analogous to obliteration of the vermiform appendix after catarrhal appendicitis.

Possibly among the different forms of hypertrophic biliary cirrhosis there are some cases due to an ascending infection, though the majority are, like scarlatinal nephritis, due to a poison reaching them by the general circulation.

A question which cannot at present be answered is whether poisons reaching the liver by the portal vein ever set up the lesions of hypertrophic biliary cirrhosis. As shown by experiments with toluylenediamine, poisons in the general circulation reaching the liver are excreted into the small bile ducts, and set up inflammation of the smaller ducts; this is analogous to hypertrophic biliary cirrhosis. As far as we know, poisons arriving by the portal vein tend to produce common (portal) cirrhosis. An exception, however, must be made for congenital syphilis (*vide* p. 475), where the fibrosis is intercellular.

Bacterial Origin.—Although a specific origin for the disease has been anticipated, no microbic cause has been established. The colon bacillus has been found in blood withdrawn by puncture from the liver during life, and subsequently in the liver and spleen in the same case (Gilbert and Fournier). But further evidence must be brought forward before the colon bacillus can be regarded as the specific cause. Hayem, in a class of cases he describes as chronic infectious jaundice with splenic enlargement and exacerbations, but which is very closely allied to, if not the same disease as hypertrophic biliary cirrhosis, found the diplococcus pneumoniæ in blood aspirated from the spleen during life. The absence of suppuration and chronicity of the disease are against its being due to pyogenetic cocci.

Probable though the bacterial origin of the disease is, further research is urgently required before it can be considered as proved.

Morbid Anatomy.—The liver is enlarged, and weighs from 80 oz. to 8 lbs. or more; it is uniformly increased in size. Not infrequently there are perihepatitic adhesions, but apart from them the surface of the organ is almost smooth and does not show the gnarled aspect of common cirrhosis. Sometimes from secondary portal cirrhosis the surface becomes slightly irregular. It is of a dark green colour, and on section is firmer than natural.

The portal vein and hepatic artery show no signs of inflammation.

The gall-bladder contains bile, and is usually healthy, though its walls are sometimes thickened. The larger bile ducts appear normal. It is remarkable that, inasmuch as there is cholangitis, bilirubin-calcium calculi are not more often present in the ducts. Gall-stones have been found in cases of hypertrophic biliary cirrhosis, and can be quite well explained as a secondary formation; it is not necessary to assume that they are primary and the cause of the cirrhosis.

*Microscopically the liver* shows monolobular cirrhosis; connective tissue of a delicate and open structure, fibrillar rather than fibrous, separating the individual lobules from each other. This fibrosis in parts invades the lobules, and then becomes pericellular; so that as compared with common cirrhosis, the fibrosis is less dense, but is more intimately related to the liver-cells. In addition there is in most cases ordinary multilobular cirrhosis. Very probably this is a secondary lesion, and may, as Chauffard has suggested, be due to poisons manufactured in the spleen and conveyed to the liver by the portal vein.

The small bile ducts show proliferation of their epithelium, which may block up their lumen (cholangitis), so that instead of being lined by a single layer of columnar cells, the ducts may contain smaller proliferated cells. In places there is an increased amount of fibrous tissue around the ducts, due to pericholangitis. The bile capillaries may contain plugs of inspissated bile or microscopic calculi. Around the portal spaces there are numbers of the so-called new bile ducts, rows of small deeply staining cells. A good deal of discussion has taken place as to their nature and origin; they are met with in very diverse conditions, *i.e.* common cirrhosis, acute yellow atrophy, and gumma. Various interpretations have been put upon this appearance. They have been thought to be new bile ducts, old bile ducts that from recession of the surrounding parts have become unduly prominent, degenerating and compressed liver-cells, or the result of compensatory hyperplasia of the liver-cells. They may, perhaps, more conveniently be called "pseudo-bile canaliculi." Though once regarded as connected with biliary cirrhosis no such importance can now be attached to them, inasmuch as they are met with in such various conditions, and are sometimes absent in hypertrophic biliary cirrhosis.

The liver-cells are often extremely well preserved, and commonly do not show the fatty and atrophic changes met with in ordinary cirrhosis. They may show signs of karyokinesis. Hanot laid stress on the absence of degeneration in the liver-cells; but this must not be pressed too far, for acute degeneration changes leading to icterus gravis may supervene and rapidly prove fatal. It appears that Hanot's early observations were largely based on examination of a case that succumbed from pneumonia, and did not run the ordinary course of the disease.

The *spleen* is also much enlarged; it may, indeed, exceptionally in children, be bigger than

the liver. Its weight is usually between 15 and 40 oz. It shows peritoneal adhesions, and on section is firmer than normal, and microscopically presents lymphatic hyperplasia and fibrosis.

The *lymphatic glands* in the portal fissure are sometimes enlarged, but are so soft that they do not exert pressure on the bile ducts. They are dark in colour and œdematous; microscopically there is fibrosis and pigmentation. Besides those in the hilum of the liver the glands around the pancreas may be similarly affected. In some exceptional instances glandular enlargement has been detected in distant parts of the body, in the axilla, the groin, the mediastinum, and the neck (Popoff).

The *alimentary canal* is usually free from signs of past inflammation. Hanot noted that the duodenum in the region of the biliary papilla was not affected by catarrh; Debove's experience, however, is rather in the opposite direction.

The *pancreas* is not increased in size, but shows a very intimate embryonic fibrosis spreading from the ducts, and thus resembling the changes described in the liver.

All the organs are bile-stained.

Symptoms.—The *onset* may be gradual, and before jaundice sets in malaise, loss of strength, and in some cases dyspepsia may be noticed.

Jaundice is slight at first, and becomes more marked as the disease progresses; it is permanent, but varies in degree, being intensified at intervals when exacerbations in the disease occur. After these crises it recedes, and eventually it may become green.

The abdomen enlarges from the increase in size of the liver and spleen, and there is dull pain and tenderness in the hepatic region. It is noteworthy that there is no enlargement of the subcutaneous veins on the abdominal wall.

The tongue is furred; the appetite is sometimes poor, but in other cases is good; there is said not to be any distaste for fatty food as there often is in obstructive jaundice. Exceptionally the appetite is ravenous. Nausea and vomiting may occur, but are by no means constant. Diarrhœa is often present. The motions contain bile; this is a point of distinction between the disease and obstructive jaundice with hepatic enlargement.

From time to time attacks of abdominal pain with fever and increase in the degree of jaundice occur; these exacerbations are like those seen in pernicious anæmia and in Addison's disease. Occasionally acute degenerative changes in the liver-cells occur in one of those attacks, with the result that the jaundice becomes deep; delirium, nervous symptoms, and a typhoid condition develop, and death follows.

The jaundiced skin may become very dark in colour, even resembling melanodermia; it may also be very irritable, and become covered with an eczematous or lichenous eruption. The long-continued jaundice may lead to xanthelasma.

When the disease occurs in childhood, growth is naturally interfered with, and the appearance may be infantile, and bodily development greatly retarded, so that the onset of puberty is arrested. In some rare instances clubbing of the fingers and toes with deformities of the nails have been noticed. Examination with X-rays has shown that there is no bony enlargement of the terminal phalanges. In their spleno-megalic type of biliary cirrhosis occurring in children, Gilbert and Fournier have further recorded enlargements of the ends of the long bones, pain in the joints, and synovial effusion. These lesions, which resemble Marie's hypertrophic pulmonary osteo-arthropathy, are extremely rare in biliary cirrhosis, and are not dependent on pulmonary lesions. They are not limited to this form of jaundice, for they were marked in a case in St. George's Hospital under the care of Dr. Ewart, in which a gumma obstructed the bile ducts of a boy aged seventeen years. The bulbous or "Hippocratic" fingers have been found associated with perforating ulcer of the foot and neuritis in hypertrophic biliary cirrhosis.

The blood may show leucocytosis, thus differing from common cirrhosis where it is not found.

It was found in three cases by Hanot and Meunier where there was no cause for it, such as inflammation elsewhere, and they quote two other cases, making five in all. Kirikow, however, finds that leucocytosis is not constant.

The heart tends to dilate, and its contractions are somewhat feeble, but its rate is not slowed.

Epistaxis and hæmorrhages from the gums and in the skin are frequent in the later stages, but the copious hæmatemesis met with in common cirrhosis rarely occurs.

The urine is acid, diminished in quantity and high-coloured, but not prone to lithatic deposit as in common cirrhosis. Albumin and sugar are absent; owing to the liver-cells being preserved, at any rate for a considerable time, in good nutrition, glycosuria cannot be produced by giving syrup or sugary food by the mouth. (Absence of alimentary glycosuria.) Urea is diminished in amount. Bile pigment is practically always present in the urine. The toxicity of the urine is feeble, and this has been used as an argument against the view that hypertrophic biliary cirrhosis is primarily due to a general hæmic infection.

Physical Signs.—The *liver* is much enlarged, and smooth, and firm to the touch; occasionally it is slightly irregular from the presence of perihepatitic adhesions. It is uniformly increased in size. Its dulness often extends upwards to the fourth rib in the right nipple line, and downwards to the umbilicus, or even

below that point. The pressure of the enlarged organ pushes the costal arch out. On palpation there is general but not any localised tenderness. There is no enlargement of the gall-bladder. The enlargement of the liver is, generally speaking, progressive; it may vary from time to time, and late in the disease may sometimes diminish in bulk from some degree of contraction of the fibrous tissue, probably from secondary multilobular cirrhosis.

The spleen is very considerably enlarged, much more so than in common cirrhosis. The enlargement is more marked in children in accordance with the fact that its capsule is more distensible than in adults. A special form of hypertrophic biliary cirrhosis has been described by Gilbert and Fournier as the juvenile type or cirrhose biliare splenomegalique. The spleen may indeed be not only relatively but absolutely heavier than the liver. Auscultation over the spleen sometimes reveals a soft blowing murmur.

Three forms of the disease have been described: (i.) the common one, in which both organs are much enlarged; (ii.) a form in which the liver is chiefly prominent; and (iii.) the one where the splenic enlargement is especially marked.

The splenic enlargement may precede the hepatic, and may occur in some members of a family in which others have the fully developed disease. Thus in a family recorded by Boinet the father and two children had hypertrophic biliary cirrhosis, while three other children had enlarged spleens.

There is no enlargement of the subcutaneous veins of the abdomen, and ascites is not present except in the last stages, and then usually only in a slight degree.

At first and for a considerable time the general health is often well preserved. After repeated exacerbations the condition advances, and as wasting sets in the patient goes down hill.

Death may occur from intercurrent disease, from icterus gravis, or gradually in coma. Fatal hæmatemesis is very rare indeed.

Diagnosis. — In cases of common cirrhosis with big livers and intercurrent jaundice the diagnosis depends on the jaundice being transitory and not permanent, on the slight degree of splenic enlargement, and on the presence of signs of common cirrhosis such as ascites and enlargement of the subcutaneous veins of the abdomen. It cannot, however, be maintained that the two diseases (portal and biliary cirrhosis) are always distinct either anatomically or clinically. Sometimes they are combined, and not infrequently the diseases overlap in the same way as the parenchymatous and interstitial forms of nephritis.

In hæmochromatosis, a condition where there is widespread pigmentation of the body with secondary cirrhosis of the liver and pancreas, the liver is enlarged, and many of the symptoms resemble those of hypertrophic biliary cirrhosis. The skin, however, though pigmented is not jaundiced, and in five-sixths of the cases there is glycosuria (bronzed diabetes).

In cases of obstructive jaundice the liver may be enlarged and swollen from retained bile; but this condition differs from hypertrophic biliary cirrhosis in the absence of bile from the fæces, in the fact that there is no splenic enlargement, and often in the presence of an enlarged gall-bladder.

When a gall-stone lies in the common duct and gives rise to chronic jaundice, some bile often passes by the stone and enters the duodenum, so that the fæces are not necessarily pale. The spleen, however, is not enlarged; this and the history of severe attacks of biliary colic should differentiate it from hypertrophic biliary cirrhosis.

In prolonged catarrhal jaundice the spleen is but slightly enlarged, and bile is absent from the fæces.

In prolonged cases of what appear to be infectious jaundice the condition is indistinguishable from that of hypertrophic biliary cirrhosis, except in the fact that recovery occurs; in other words, the diseases are practically identical in nature though not in their results.

In Weil's disease the clinical course is rapid and acute, whereas in hypertrophic biliary cirrhosis it is a matter of years, not of days.

Malaria, which has been regarded by Lancereaux as the cause of hypertrophic biliary cirrhosis, can be eliminated by examination of the blood and by the failure of quinine to affect the course of the disease.

Some rather exceptional cases of syphilitic disease of the liver, with chronic jaundice and very considerable enlargement of the liver and spleen, may imitate hypertrophic biliary cirrhosis. Syphilitic lesions elsewhere, albuminuria as pointing to lardaceous disease as the cause of splenic enlargement, irregularity of the surface of the liver from gummata, the presence of enlarged veins on the abdomen, and the beneficial effects of antisyphilitic treatment, point to syphilis. Another point is the absence of leucocytosis in syphilis and its presence in hypertrophic biliary cirrhosis.

Treatment.—In the early stages an attempt may be made to put the patient in more healthy surroundings, and to remove him from the conditions, among which the water-supply may play a part, that favoured the onset of the disease.

The patient should avoid cold, especially damp cold, and should be warmly clad. A course at Carlsbad, Vichy, Marienbad, or Kissingen may be tried.

It is most important that the diet should be of a simple character and free from spices or

irritating constituents. Milk is the staple, and is specially advantageous from its diuretic effect on the kidneys; to this eggs and bread and butter, with occasionally fish, may be added. Alcohol should be avoided as far as possible.

Intestinal antiseptics, such as calomel, β-naphthol, salol, and so forth, should be given. The first named is said to give good results; it is also useful in preventing constipation and auto-intoxication from stagnation of fæcal matter.

Duration and Prognosis. — Though the disease is probably incurable it is often prolonged for many years, jaundice lasting for ten or more years. The average duration of symptoms is about five years. The symptoms may recede on careful treatment, but alcoholism, exposure, or over-work will bring them back.

In some few recorded cases the disease has run a very acute course.

Clubbing of the fingers, though a rare condition, is only met with in long-standing cases, and is an indication that the course of the disease is slow.

### Obstructive Biliary Cirrhosis

By obstructive biliary cirrhosis is meant a fibrosis spreading from the bile ducts around the lobules of the liver, and due to obstruction of the large bile ducts. Charcot and Gombault, who described this condition, supported their contention by the results of ligaturing the common duct in animals, an experiment that Wickham Legg had previously performed in this country. In these experiments the ducts were found to be dilated, and to be the starting-point of fibrosis which surrounded the individual lobules and sometimes penetrated into their substance; in the fibrous tissue surrounding the hepatic lobules there were numerous new bile ducts (pseudo-bile canaliculi) which joined on to the liver-cells. These experiments have been frequently repeated, and the conclusion to be drawn from them is that the cirrhosis obtained by the earlier workers was due to infection, and that aseptic ligature of the ducts leads to little or no fibrosis, but merely to focal necrosis of the liver-cells. The facts observed in the human subject are in general agreement with the foregoing. When the common bile duct is compressed by malignant disease, for example in carcinoma of the head of the pancreas, the bile ducts become dilated, and there are degenerative and necrotic changes in the liver-cells, but practically no fibrosis. On the other hand, when a gall-stone is lodged in the common bile duct the results are not so constant; sometimes the changes are the same as in aseptic closure of the common ducts; but in other instances there is cholangitis and pericholangitis, which if the process is chronic results in fibrosis around the ducts. The determining factor is evidently in great measure the freedom from or presence of an ascending infection of the ducts. The histological changes thus produced are not the same as those of hypertrophic biliary cirrhosis (*vide* p. 447).

It must, however, be remembered that obstruction of the bile ducts necessarily carries with it the absence of bile in the intestines; the latter is a factor that will tend to increase fermentation in the alimentary canal, and so lead to the production of poisons, which when carried to the liver might be expected to set up the ordinary portal or multilobular cirrhosis. This change would indeed, in all probability, more often be met with were it not that the liver, being flooded with bile, which has acquired toxic properties (as shown by the focal necrosis of the liver-cells), is incapable of any reaction.

Cases of long-standing gall-stone obstruction associated with ordinary portal cirrhosis of the liver undoubtedly occur; but the symptoms are chiefly those of biliary obstruction, and not of portal vein obstruction.

Hypertrophic biliary cirrhosis has sometimes been found associated with gall-stones, but it is quite reasonable to think that the gall-stones are secondary to cholangitis, and not the primary morbid factor.

*Clinical Features.*—When cirrhosis of the liver, whether it be pericholangitic or portal, occurs in a patient with biliary obstruction, it does not give rise to any special signs or symptoms. The features remain those of biliary obstruction.

In some instances a gall-stone may become lodged in the common bile duct without any history of colic being obtained. In such cases the question of diagnosis may be one of considerable difficulty (*vide* p. 449). As time progresses bile may escape by the side of the stone into the duodenum, and the fæces are no longer pale; they then contain bile just as they do in hypertrophic biliary cirrhosis.

In differentiating these two conditions the size of the spleen is important; big in hypertrophic biliary cirrhosis, not enlarged in gall-stone obstruction. In hypertrophic biliary cirrhosis the liver is greatly increased in size, in biliary obstruction it is swollen from retention of bile, but in the late stages and when fibrosis develops it becomes smaller.

*To sum up*: A chronic ascending cholangitis leading to pericholangitic fibrosis may be associated with and favoured by gall-stone obstruction, but it depends on infection, and not on obstruction of the ducts *per se*. This fibrosis is clinically of little importance; the features of the case are those of obstructive jaundice.

In some instances ordinary portal cirrhosis may develop after gall-stone obstruction has been established, and is reasonably explained as the result of poisons manufactured in the intestines and carried to the liver by the portal vein.

Complete aseptic obstruction of the common bile duct leads to dilatation of the intrahepatic ducts and to focal necrosis of the liver-cells, but not to cirrhosis. The functional activity of the liver is thus very gravely interfered with, and as a result of this hepatic inadequacy cholæmia or biliary toxæmia results, a condition which is much more rapidly fatal than cirrhosis.

Finally, biliary obstruction does not give rise to any fixed type, either pathological or clinical, of cirrhosis.

## Portal Cirrhosis


Introduction . . . . . 451
Etiology . . . . . 451
*Alcohol* . . . . . 451
*Gastro-intestinal Catarrh* . . . 451
*Micro-Organisms* . . . . 451
Sex. Age . . . . . 452
Morbid Anatomy . . . . . 452
Associated Lesions—
*Tuberculosis* . . . . . 455
*Renal Disease* . . . . . 455
Course . . . . . 455
Signs of Cirrhosis . . . . . 456
*Ascites* . . . . . 456
Symptoms . . . . . 458
*Hæmatemesis* . . . . . 458
Complications . . . . . 459
Diagnosis . . . . . 460
Prognosis . . . . . 460
Treatment . . . . . 460


Synonyms: *Common, Atrophic, or Multilobular Cirrhosis; Hobnailed Liver; Gin or Whisky Drinker's Liver; Chronic Interstitial Hepatitis.*

Introduction.—The term cirrhosis was first employed by Laennec (1819), who regarded the yellow bile-stained "hobnails" as due to some new formation, and therefore termed it cirrhosis (κιρρός = yellow). The term atrophic cirrhosis, though in common use, is undesirable, inasmuch as many of the livers of multilobular cirrhosis are by no means small. The adjectives portal, multilobular, or common are more suitable.

Etiology.—The changes of cirrhosis are due to the action of poisons or possibly poison-producing bodies — micro-organisms — reaching the liver.

These bodies are usually conveyed to the liver by the portal vein, but in some instances they reach the liver by the hepatic artery. Thus in the rare condition hæmochromatosis, where there is widespread infiltration of the body with blood pigments set free by hæmolysis, possibly of microbic origin, the liver and pancreas become fibrotic. The hepatic artery in these cases shows endarteritis. Again, in scarlet fever there may be an acute interstitial hepatitis analogous to acute nephritis, and, like it, due to a poison reaching the liver from the general circulation; it is possible that traces of this lesion may persist and lay the foundation of ordinary cirrhosis. The same may be true of other specific fevers such as measles. Similarly, in the hæmic infections it is not improbable that focal necrosis of the liver-cells and connective tissue proliferation around these areas may, under certain conditions, lead to cirrhosis.

In some instances, however, cirrhosis appears to be due to poisons reaching the liver by the portal vein. Alcohol has always been considered the cause *par excellence* of hepatic cirrhosis, and figures largely in the past history of patients. It is not, however, the typical drunkard so much as the constant tippler who develops cirrhosis. Experimentally alcohol gives rise to degeneration and fatty changes in the liver-cells and not to cirrhosis, so that it would appear that alcoholism only produces cirrhosis indirectly by favouring the development of the accessory factors.

It has been suggested that though alcohol itself does not lead to cirrhosis, alcoholic liquors, in virtue of other constituents, such as sulphate of potash (Lancereaux), with which wines are "plastered," amyl alcohol, or fatty acids, have this effect.

A very probable view is that alcoholism gives rise on the one hand to gastro-intestinal catarrh, and thus to the formation of poisonous bodies, which are the active factors in the production of cirrhosis, and that, on the other hand, it acts as a protoplasmic poison, and reduces the resistance of the liver, thus allowing the aforesaid poisons to act more vigorously and at greater advantage.

Cirrhosis may undoubtedly occur without alcoholism, and recently Hanot and Boix have described dyspeptic or "Budd's cirrhosis" probably brought about by fatty acids, such as acetic, butyric, valerianic, and lactic, manufactured in the alimentary canal as the result of fermentation. In this way cirrhosis may be set up by spices and other articles of stimulating diet.

In some instances, as Chauffard has suggested, poisons may be manufactured in the spleen and be carried to the liver by the portal vein, and then set up cirrhosis. Thus in Banti's disease, a severe form of splenic anæmia with terminal cirrhosis, it is probable that a chronic intoxication or infection chiefly affecting the spleen leads to this further change in the liver.

The rôle of micro-organisms in the production of cirrhosis, though rendered highly probable by the suggestive work of Adami and his pupils, is not at present satisfactorily established. It would appear probable that, as the result of alcoholism, the walls of the intestines may be so damaged as to allow of their penetration by micro-organisms, which thus reach the liver; and if its resistance is also diminished by the toxic effects of alcohol they may multiply, and by

their toxins induce cirrhosis. A small diplococcus belonging to the colon group has been found by Adami, not only in cirrhotic, but also in other and even in normal livers. It was suggested that while in health the micro-organisms are destroyed by the liver; in pathological conditions, where the resistance of the liver is reduced, the micro-organisms may become virulent, and lead to the changes of cirrhosis.

Syphilis does not give rise to ordinary portal cirrhosis. The hepatic lesion of congenital syphilis is a diffuse pericellular infiltration, which is a curable condition. As pointed out elsewhere (*vide* p. 477), patients who have presumably had this lesion may, their liver being a place of least resistance, develop ordinary multilobular cirrhosis on slight provocation; this may be regarded as a parasyphilitic lesion.

Malaria is often mentioned as a cause of cirrhosis, but there is reason to believe that the two diseases are rather associated together than related as cause and effect. Though malaria induces changes, necrosis and hyperplasia of the liver-cells, which might cause cirrhosis, this is not very frequently proved actually to occur in practice.

Incidence.—Males are more often affected than females in the proportion of $5\frac{1}{2}$ to 2; in 508 cases of cirrhosis, obtained by adding together the statistics of Price, Kelynack, Yeld, Fenton's and my own, 374 were males and 134 females. It appears, however, that the disease is more often latent in men than in women. In children also the male sex is more often attacked than the female.

Age.—The average age at which cirrhosis is fatal in adults is about forty-eight years; if the examples of cirrhosis in young children are included the age would, of course, be lower. A large proportion of the cases fatal in children occur before six years of age.

Morbid Anatomy.—The size and weight of the liver in portal cirrhosis vary considerably. Sometimes the liver is much reduced in size, and may weigh under 30 oz.; in other cases it is as much as twice the normal weight. As a rule, it is rather heavier than natural. In 114 cases at St. George's Hospital the average weight was 65 oz., in 100 cases collected by Hawkins the average was 52 oz., and in 93 collected by Kelynack 53 oz. It is noteworthy that a cirrhotic liver, which looks considerably smaller than a normal one, often weighs as much or more, its specific gravity being increased.

Various forms of portal cirrhosis have been described, and different causes are assigned for the large cirrhotic livers. In some cases the increased size is due to fatty change in the liver-cells, and it has been assumed, but probably incorrectly, that this is especially associated with indulgence in malt liquors. When cirrhosis is associated with pulmonary tuberculosis the liver is often enlarged and fatty. In some instances the increase in size is due to compensatory hyperplasia of the liver-cells—the hypertrophic alcoholic cirrhosis of Hanot and Gilbert—and is associated with latency of the symptoms and arrest of the disease. In other instances the large size is due to the fibrosis having a smaller mesh and approaching a monolobular type; these cases may appropriately be described as mixed cirrhosis. Generally speaking, the liver is larger in young subjects with cirrhosis than in cases fatal later in life. The larger cirrhotic livers are less knobby than the small cirrhotic livers, which especially merit the term "hobnail."

The capsule is more opaque than in health, but there is very seldom much chronic perihepatitis. There may be adhesions between the surface of the liver and the diaphragm; when present they are scattered rather than extensive, and are usually markedly vascular.

The surface of the organ is irregular; the projections vary in size from a pea to that of a pigeon's egg. When they are small the surface somewhat resembles that of a granular kidney, and the term "granular liver" is applicable. When, as more rarely occurs, the hobnails are large, the organ may look as if it was occupied by numerous secondary growths, especially when the projections show marked fatty change and appear white, though it is worthy of note that the hobnails are never umbilicated, as is usual in secondary carcinoma. When the projections are exceptionally large the condition is sometimes spoken of as nodular cirrhosis, or cirrhosis with multiple adenoma (*vide* p. 466). The hobnails are of a tawny yellow or brown colour, being often stained by bile; the peritoneum over them sometimes shows dilated vessels. During life the liver looked uniformly red in the laparotomies in cases of cirrhosis I have seen. The capsule, which is not much thickened as a rule, is more opaque in the depressions between the nodules.

Usually the liver is uniformly affected, especially when it is enlarged and the nodules are small, but the change may be irregular, and the left lobe is often in a more advanced condition, and may be very small. It is possible that the resistance of the left lobe is less than that of the right, for it is not infrequently more affected in acute yellow atrophy (*q.v.*) than the right. Sometimes, on the other hand, one of the smaller lobes, such as the Spigelian or caudate lobe, may be enlarged out of proportion to the others, even when the organ as a whole is little if at all bigger than normal.

On section the liver is much tougher than normal, and is like a section of conglomerate stone being divided up into areas of irregular size by grey, slightly gelatinous-looking fibrous tissue. This fibrous tissue is continuous with the depressed, more opaque areas on the capsule, and by its contraction has squeezed into prominence the more healthy parts of the liver,

which thus form the nodules or hobnails. This fibrosis spreads out from the medium-sized portal canals, and exerts its constricting influence on the branches of the portal vein. The areas of liver substance thus enclosed vary in size, usually being from $\frac{1}{4}$ to $\frac{1}{6}$ inch in diameter, and enclose six to ten lobules, each of which normally measures about $\frac{1}{16}$-$\frac{1}{20}$ inch in diameter. The liver substance is much paler than in health, and has a yellowish-brown colour from staining with bile. In exceptional cases there may be hæmorrhage either into the hobnails or into the surrounding interstitial tissue; in the latter event, if there is fatty degeneration in the hobnails, the resemblance to new growth may be very realistic. In some cases the hobnails soften down in the centre.

*Histology.*—In the early or more progressive stages there is small-cell infiltration in and around the portal spaces; these cells are due to hyperplasia of the existing connective tissues of Glisson's capsule; some leucocytes are also present. In a well-marked case there is an irregular mesh-work of fibrous tissue extending throughout the liver, and dividing it up into variously sized islands of liver tissue. Inasmuch as a number of lobules are enclosed within the same fence of fibrous tissue the term multilobular cirrhosis is applied. The number of lobules enclosed in different compartments differs; in some parts there are many, in other areas a single lobule or half a lobule is separated off from the rest.

The French school considers that the fibrosis is not only portal, but also around the sublobular veins, or bivenous. It is true that the pressure of the surrounding fibrous trabeculæ may obliterate the intralobular veins, and in other ways so alter the appearance of the lobule that it is difficult to count the number of lobules enclosed in the alveoli of the fibrous tissue; but it does not appear, at any rate to me, that there is fibrosis around the intralobular veins. At the margin of the lobules the fibrous tissue can be seen to surround bits of the lobule, and thus to shave off groups of cells from the edge of the lobule. In some large cirrhotic livers, where the mesh-work is still multilobular as a whole, there are parts where it is more diffuse and approaches the monolobular type; this condition of mixed cirrhosis is a transitional stage to biliary cirrhosis. The fibrous tissue varies according to the age and rate at which the process is progressing. Usually there is some well-formed fibrous tissue containing younger connective tissue and small round cells. The interstitial fibro-nuclear tissue contains numerous small vessels with thin walls derived from the branches of the hepatic artery. Elastic fibres are present in the fibrous tissue.

In addition the fibrous tissue contains columns of small cells with deeply-staining nuclei, often described as new bile ducts. This appearance is seen in many conditions, such as gumma, tubercle, lymphadenoma, and acute yellow atrophy, where destruction of the liver-cells is occurring. They have been regarded in the following various lights: as degraded liver-cells reverting to the type of bile ducts, as normal bile ducts which have become exposed by atrophy and recession of the liver-cells, and as a hyperplasia of the liver-cells—an attempt to compensate for the destruction of liver-cells. The latter seems a satisfactory explanation, and this appearance may therefore be spoken of as "pseudo-bile canaliculi."

In some cases of malarial cirrhosis, in the liver of hæmochromatosis, and in the rare condition of cirrhosis anthracotica, the fibrous tissue may contain opaque masses of pigment.

The normal arrangement of the liver-cells in the lobule is lost, probably from the pressure exerted by the contracting fibrous tissue. The cells show degenerative changes, the atrophy and fatty changes are common, while pigmentary infiltration may occur.

Several views have been put forward as to the relation of the fibrous tissue formation to the degenerative changes in the liver-cells; it has been thought that the fibrous tissue is first formed by active proliferation, and that by its subsequent contraction, atrophy and degeneration of the liver-cells are induced, while conversely it has been held that the degeneration of the liver-cells is primary and that the fibrosis is only apparent, or at best a replacement fibrosis, and the process comparable to that in a granular arteriosclerotic kidney. Probably the two changes are both due to toxic causes and independent of each other at first; later fibrous contraction may increase the atrophy of the liver-cells, while in return the products of degeneration of the cells may further stimulate hyperplasia of the connective tissue elements.

*The large bile ducts and the gall-bladder* are usually healthy to the naked eye. The walls of the gall-bladder are, however, sometimes rather thickened and contracted.

Biliary calculi are not specially frequent in cirrhosis; in 100 cases of fatal cirrhosis examined at St. George's Hospital 12 showed calculi either in the gall-bladder or the small ducts, the later being bilirubin-calcium calculi.

The *hepatic* artery is usually enlarged inasmuch as it supplies the added fibrous tissue in the organ. In the multilobular cirrhosis of hæmochromatosis the artery shows endarteritis.

*Portal Vein.*—The intrahepatic branches are compressed while the trunk is accordingly somewhat dilated. Its walls show some thickening, and its intima is rather opaque. The communications between the radicles of the portal vein and the general systemic veins are greatly enlarged and increased in extent. Thrombosis of the portal vein occasionally occurs in cirrhosis. Cirrhosis of the liver, in-

deed, is the most frequent associated condition of pylethrombosis, but it is not a common occurrence.

*Communications between the Portal System and the General Systemic Veins.*—The anastomoses which normally exist between the radicles of the portal vein and the adjacent systemic veins become dilated and increased in extent in common cirrhosis. The portal circulation is thus short-circuited, and the engorgement relieved by the passage of some of the blood into the inferior or superior vena cava without traversing the liver. The development of this collateral circulation relieves portal congestion, and is thought to be compensatory.

These communications are—

1. A general anastomosis between the veins of the peritoneum and those of the abdominal walls, such as the lumbar and renal. These anastomoses are especially well marked where the duodenum and colon are bound down to the abdominal wall and are only partially covered by peritoneum. This subperitoneal anastomosis, described by Retzius, gives rise to marked injection of the peritoneum, which is especially noticeable during life, as seen in laparotomies on cases of cirrhosis.

2. Those around or in connection with the liver. The phrenic and intercostal veins on the diaphragm communicate between the layers of the coronary ligament with the veins in the liver; this is not of much utility. Dendritic venous markings on the skin along the line of attachment of the diaphragm occur in conditions like emphysema, and have no constant relation to cirrhosis. In the falciform ligament the parumbilical veins of Sappey put the portal vein into communication with the veins of the anterior abdominal wall. A large vein may thus run up in the falciform ligament which imitates the anterior epigastric vein of the frog. This anastomosis may show itself as a "caput medusæ," or number of dilated veins around the umbilicus. This anastomosis must be distinguished from the more marked "caput medusæ" which results from obstruction to the passage of blood along the inferior vena cava; in the latter the dilated epigastric and mammary veins avoid and do not centre around the umbilicus. In cases of extensive ascites, both collateral circulatory channels may be developed.

An epigastric venous hum, audible with the stethoscope, has been referred to the collateral circulation in the falciform ligament.

3. Between the œsophageal veins, discharging into the azygos veins and so into the superior vena cava on the one hand, and the gastric veins on the other hand. These veins may become varicose (œsophageal piles), and as the result of chronic inflammation the mucous membrane may become first adherent and then ulcerated. Profuse and even fatal hæmatemesis may thus be induced. In 80 per cent of the cases of fatal gastro-intestinal hæmorrhage these œsophageal varices are present. Varicose gastric veins, especially around the cardiac orifice, are present in a small proportion of cases.

4. Between the superior hæmorrhoidal veins, tributaries of the inferior mesenteric vein, and the middle and inferior hæmorrhoidal veins which open into the internal iliac veins. Dilatation and varicosity of these veins lead to piles. It is probable that cirrhosis is not so important a cause of piles as has sometimes been stated, and at any rate takes a very subordinate position in this respect to constipation.

This collateral circulation is regarded as compensatory; but it often fails in this object, as shown by its presence in fatal cases, while it is sometimes absent in cases where latent cirrhosis is found in persons dying from other causes. Its good effects have been imitated in the recent treatment of cirrhosis by the production of artificial adhesions. If carried to its logical conclusion this measure would result in short-circuiting the portal circulation as in Eck's fistula, or the union of the portal vein with the inferior vena cava, an experiment that induces a uræmic tendency in dogs. It is probable that the good effects of the operation are not due to relieving portal congestion alone, but to improve nutrition of the liver, promoting hyperplasia of its cells.

*The Spleen.*—Enlargement of the spleen is an important and very frequent feature of cirrhosis; it is present in 80 per cent of the cases. It does not, however, appear to be enlarged in cases where cirrhosis, though present, is latent. It is enlarged early in the course of the disease, and may diminish in size as the result of hæmorrhage, severe diarrhœa, or ascites. The enlargement does not bear any relation to the size of the liver in ordinary cirrhosis, though in biliary cirrhosis there is a certain relation between the large liver and the spleen.

The splenic enlargement was formerly thought to be mechanical, and due to congestion depending on portal obstruction; that this is not the exclusive factor is shown by the following facts:—

(i.) That the enlargement is an early sign before evidence of portal obstruction has become apparent.

(ii.) That in biliary cirrhosis, where portal obstruction is slight or at any rate much less marked than in common cirrhosis, the splenic enlargement is much more marked.

(iii.) That the average weight of the spleen in 56 cases of morbus cordis, uncomplicated by any febrile or toxic process, was 7·3 oz., while in 84 cases of cirrhosis the spleen averaged 12·9 oz. (Kelynack). It is true, however, as Foxwell has pointed out, the liver acts as a kind of buffer in cases of morbus cordis, and

the passive congestion of the spleen need, therefore, not be so great as in cirrhosis where the obstruction is in the portal circulation.

On the other hand, passive congestion plays some part in the splenic enlargement, for hæmorrhages may lead to considerable diminution in the size of the organ in cirrhosis, and thrombosis of the splenic vein may be followed by very great splenic enlargement.

No doubt the important factor in the splenic enlargement in cirrhosis is toxic rather than purely mechanical. The poisons reaching the organ by the splenic artery give rise to an inflammatory swelling; but when there is passive congestion superadded the enlargement will be accentuated.

A certain amount of chronic inflammation of the capsule, or perisplenitis, is common; it may be localised in the form of corneal or lamellar fibromata, or generalised as in chronic peritonitis. Adhesions to the diaphragm are not uncommon. In 131 cases of cirrhosis analysed by Yeld there was chronic perisplenitis in 43, or 33 per cent.

Histologically there is proliferation of the splenic pulp in the earlier stages of cirrhosis, which may be succeeded later on in the disease by fibrosis and atrophy like that seen in experimental chronic intoxications.

*Peritoneum.*—Besides the dilatation of the blood-vessels of the peritoneum already referred to there is no constant lesion. A certain degree of chronic peritonitis is not infrequently seen, and secondary infections may give rise to acute or tuberculous peritonitis.

The *œsophagus* shows dilated and varicose veins towards its lower end, which, as already pointed out, may rupture and give rise to severe or fatal hæmorrhage; the mucous membrane of the œsophagus may be thickened.

The *stomach* usually shows chronic gastritis; as evidence of this, pigmentation at the pylorus is not uncommon.

The *intestines* also show signs of catarrh; when there is chronic peritonitis their length may be considerably curtailed. The pancreas is larger and heavier than normal, and shows a wide-marked fibrosis with fatty and pigmentary degeneration of the gland cells.

The *heart* is commonly flabby, occasionally dilated, and sometimes shows fatty degeneration, probably from concomitant alcoholism.

The *lungs* are often œdematous; the occurrence of tubercle will be referred to below.

### Associated Lesions

Tuberculosis. — The subjects of alcoholic cirrhosis are more prone to tuberculous infection than non-alcoholic persons dying from other diseases. This is probably due to alcoholism and not to cirrhosis. Tuberculosis is most often seen in the lungs and peritoneum; it may be obsolete, and only found at the autopsy, or it may be acute, and then throws into the shade the cirrhosis. Tubercle is found in the bodies of about 30 per cent of patients with cirrhosis.

Cirrhosis certainly seems to dispose the peritoneum to tuberculous infection, for its occurrence is comparatively infrequent in adult males apart from cirrhosis. Probably chronic venous engorgement reduces the resistance of the peritoneum and its lymphatics.

Kidney Disease. — Adding together the statistics of Pitt, Kelynack, Yeld, and those of Fenton and myself, 387 cases of cirrhosis are obtained, among which 87 or 22·5 per cent showed a granular kidney. The arteriosclerotic change in the kidney does not complicate the cases of cirrhosis occurring early in adult life. Statistics show that the average age of patients with both lesions is higher than those dying with cirrhosis alone. There does not seem to be any special relation between the size of the liver and its association with a granular kidney. Price found a granular kidney more often associated with a large liver, while Pitt's statistics as well as my own were exactly opposed to this conclusion. As would naturally be expected from the greater frequency of arteriosclerosis in males, the association of cirrhosis with the granular kidney is commoner in men than in women. When the two lesions coexist the symptoms are chiefly those of renal disease.

Fatty degeneration of the renal epithelium may occur in cases of cirrhosis, while sometimes the kidneys show the effects of backward pressure from cardiac failure.

### The Course of the Disease

The disease may be divided into the early or pre-ascitic stage and the late or ascitic period.

In the early stage of cirrhosis the symptoms are chiefly those of dyspepsia, often of an alcoholic nature, with loss of appetite, sickness, and irregularity of the bowels. Symptoms, indeed, may be absent, or be largely those of alcoholism.

The pre-ascitic stage begins very vaguely and gradually, but its tenor may be roughly broken by the occurrence of hæmatemesis. This may come on after some discomfort and fever, or may occur with little or no warning. After it the patient is blanched for a time, but soon recovers, and usually months or years elapse before ascites develops. In some rare instances the disease runs a rapid and often febrile course, and almost before the patient has recovered from the effects of hæmatemesis ascites begins to show itself.

After hæmatemesis the cirrhotic process may become latent and give rise to no further symptoms, especially if the patient alter his habits of life.

The late or ascitic stage may be preceded by gaseous distension of the abdomen, so that its

onset is obscured. Œdema of the feet may precede the ascites or follow it.

By the time ascites has developed the patient is already pulled down in strength and weight. The ascites increases in amount, sometimes rapidly, until tapping is required; in pure cirrhosis, without any chronic peritonitis, a second tapping may be required, but rarely more. The ascites then ceases to accumulate, and may indeed disappear, while the patient further emaciates, rapidly loses strength, and eventually passes into a drowsy, typhoid, or comatose condition, which gradually ends in death. The mental apathy is often varied by delirium of a low type. There may be hæmorrhage from hepatic insufficiency not only in the skin, but from the stomach or bowel, which may be very considerable.

The patient may linger on in a semi-comatose condition for some weeks, and then die from an acute and terminal infection or from asthenia.

On the other hand, death may occur before the stage of ascites has been reached, from some complication or acute infective process. Exceptionally, death may occur from hæmatemesis quite early in the course of the disease.

SIGNS OF CIRRHOSIS


| | |
|---|---|
| FACIAL ASPECT | 456 |
| LIVER | 456 |
| *Venous Hum* | 456 |
| ASCITES | 456 |
| CIRCULATORY SYSTEM | 457 |
| URINE | 457 |
| ŒDEMA OF FEET | 458 |


FACIAL ASPECT.—The face may be bloated and show acne rosacea due to dyspepsia, either induced by or independent of alcoholism; the area of skin affected is that around the nose and on the cheeks, and roughly corresponds with that involved in lupus erythematosus, the so-called "flush area." The skin of the face elsewhere is muddy and dirty-looking, and often presents stigmata or small clusters of dilated vessels, which sometimes bleed readily on slight provocation, and capillary hæmorrhages. The face is drawn and thin, the eyes deeply set, and the conjunctiva muddy or slightly icteric. The wasting of the temporal muscles is often very manifest. These are the appearances usually seen in the later and more advanced stages.

At an earlier period the skin may be uniformly pale, sallow, and smooth, and quite free from the blemishes seen in advanced stages.

The lips are usually dry and apt to be fissured, the tongue flabby or dry, and the gums show a tendency to become spongy and, when hepatic insufficiency has become established, to bleed. The throat is apt to be chronically congested and the breath foul.

The skin of the body is often dry and harsh, with loss of elasticity. Local hæmorrhages may occur as the result of slight or unnoticed traumatism.

THE LIVER.—It has been widely assumed that the liver is enlarged in the early stages of the disease, and that subsequently it becomes smaller from shrinking and contraction of the fibrous tissue inside it. This sequence of events is sometimes noted; thus the organ a considerable time before death has been found to be large, while at the post-mortem it has receded behind the costal arch. At the same time it is by no means certain that alterations in size of the organ can be referred solely to the contraction of the added connective tissue; for, in the early stage, the enlargement may vary within a comparatively short space of time, thus showing that the increase in size is due to engorgement.

Before ascites has appeared the liver may usually be felt below the ribs, sometimes several finger-breadths beyond the costal margin in the right nipple line, its surface being firm, slightly irregular, and often tender.

In other instances its rough, hobnailed margin can just be felt by pushing the fingers under the margin of the ribs, while sometimes it cannot be felt, and percussion may show that it has apparently diminished in size.

Before the onset of ascites tympanitic distension of the abdomen often appears, and, like ascites, prevents accurate palpation of the liver.

*Venous Hum.*—On rare instances a venous hum, louder on inspiration, can be caught over the epigastrium, and has been thought to be due to the presence of dilated vessels in the falciform ligament. In some exceptional cases a similar venous hum, compared to the uterine souffle, has been heard over the spleen.

THE SPLEEN may sometimes be definitely felt to be enlarged and firm; but though almost constantly enlarged as shown by examination after death, tympanites or ascites often masks it during life. Considerable enlargement may precede hæmatemesis, and its detection may therefore be regarded as a danger signal and call for free purgation.

ASCITES.—The onset is gradual; when it is sudden and rapidly accumulates, it may be due to thrombosis of the portal vein. It may come on shortly after injury, exposure to cold or factors that lower the resistance of the body, or after inflammation elsewhere in the body. But, as a rule, no definite exciting cause is found. It occurs in a large proportion of the cases dying from cirrhosis; but taking all cases in which cirrhosis of the liver is found on post-mortem examination, whether fatal from cirrhosis or from some other disease, the proportion is about 50 per cent.

Ascites is a late event in the course of cirrhosis, and patients seldom live to be tapped more than twice. When paracentesis has to be performed frequently in a case regarded as

cirrhosis, the condition is either complicated by chronic peritonitis or the diagnosis is incorrect.

*The Causation of Ascites.*—The obstruction to the portal circulation exerted by the cirrhotic liver is hardly sufficient to account for the ascites, since in experimental ligature of the portal vein ascites is not a necessary result. Again, it does not occur when presumably the pressure in the portal vein is highest, namely, at the same time as hæmatemesis and melæna. If the peritoneal effusion depended solely on mechanical obstruction, it should come on *pari passu* with cicatricial contraction around the portal canals in the liver. Further, the rapidity with which the fluid is sometimes poured out—a pint or more a day—is hardly compatible with the view that it is solely due to increased venous pressure. It has been suggested that the onset of ascites may depend on thrombosis of minute branches of the portal vein, or of its compensatory communications with the general systemic veins; but of this there is no proof.

The character of the fluid shows that it is not an ordinary acute inflammatory exudation, and there is nothing to support the hypothesis that the onset of ascites is due to infection. Chronic peritonitis would account for the ascites, but this lesion is not a necessary accompaniment of ascites in cirrhosis.

An attractive theory is that the ascites is toxic and due to the presence of a poison exerting a lymphagogue action; this view would explain the onset of œdema of the feet before ascites, but some further factor is required to explain the predominance of ascites over œdema elsewhere in the body. Probably this is to be found in portal congestion; it is easy to understand that stagnation of venous blood in the portal area would diminish the resistance of the endothelial cells of the peritoneum, and thus render them more susceptible to the action of a lymphagogue, while the large amount of blood at hand would further assist.

Ascitic fluid is usually slightly yellow in colour and clear; in rare instances it may be chylous, chyliform, or hæmorrhagic. Admixture with blood seems to be traumatic, and due either to damage done by a previous tapping, or to rupture of small vessels either in the peritoneum or in vascular adhesions. When peritonitis is present, the fluid becomes turbid from the presence of pus cells.

The specific gravity is 1008-1015; the fluid is alkaline, and contains 0·2-0·4 per cent of albumin and occasionally traces of sugar, urea, and urobilin. Bacteriological examination of ascitic fluid has in three cases shown the presence of Adami's diplococcoid form of the colon bacillus (M. Abbott).

Ascites pushes the diaphragm up, often displacing the heart and leading to collapse of the bases of the lungs, especially on the right side. For the physical signs and diagnosis of ascites due to different causes the reader is referred to special article on "Ascites," vol. i.

*The skin of the abdomen* shows dilated subcutaneous veins which, when much in evidence, are spoken of as *caput medusæ*. The collateral circulation between the veins of the abdominal wall and the parumbilical veins in the falciform ligament, centring around the umbilicus, is characteristic of portal obstruction, and must be distinguished from the dilated superficial epigastric veins, and the mammary and long thoracic veins, which carry on the circulation when the flow through the inferior vena cava is interfered with, and avoid the umbilicus.

In cirrhosis, with ascites, the intra-abdominal pressure may so compress the inferior vena cava that the collateral circulation through the epigastric and mammary veins becomes evident, in addition to that due to portal obstruction.

The abdomen becomes pendulous and flaccid from degeneration and atony of the muscles in its parietes, and when the ascites is extreme the umbilicus may become everted and even burst. After tapping, the skin shows lineæ albicantes.

*Circulatory System.*—The pulse is normal in rate, but the tension is low.

An apical systolic murmur is frequently present, due to dilatation rather than to valvular disease. Acute dilatation due to alcoholic excess may so alter the aspect of the case that cirrhosis is only revealed at the autopsy.

When there is ascites the heart may be considerably displaced upwards, and the apex beat may be in the 3rd interspace. This displacement tends to produce a slight kink in the pulmonary artery, and thus accounts for a systolic murmur over the artery which may disappear after paracentesis abdominis.

The Blood.—There is no leucocytosis, and as a rule no special anæmia. In the late stages the blood becomes toxic, as shown by hæmorrhages into the skin and elsewhere, and by œdema of the feet.

Urine.—The urine is diminished in amount, of high specific gravity, high-coloured, reddish-orange in appearance, and deposits a copious sediment of lithates. It is highly acid in reaction, and the amount of uric acid is increased. The amount of urea is diminished, while the ammonia is increased.

The amount of urobilin is increased, while occasionally urohæmatoporphyrin and indican have been met with. Bile pigment is only present when there is definite jaundice.

If the liver be regarded as an important factor in preventing sugar passing into the circulation, it would be natural to expect to find glycosuria in cirrhosis. But, as a matter of fact, though it is sometimes reported, alimentary glycosuria is rare in cirrhosis, and considerable doubt exists as to the value of alimentary glycosuria as a reliable sign of hepatic insufficiency. If the view be taken

that the liver is a sugar-producing organ, the comparative rarity of glycosuria in cirrhosis, and its absence where the liver is undergoing extensive disorganisation, as in acute yellow atrophy, can be understood. In hæmochromatosis the liver becomes cirrhotic, and diabetes mellitus is present in the great majority of the recorded cases (*diabète bronze*), but it is due not to the hepatic change, but to a concomitant and extensive fibrosis of the pancreas.

Albuminuria is not present, as a rule; it may be due to organic disease of the kidneys, such as granular or lardaceous change. In some instances it appears to be due to changes in the renal cells set up by toxæmia, and in such cases albumosuria has also been found. In some few instances albuminuria may be due to chronic renal congestion following dilatation of the heart, or possibly to a combination of the last two factors. It has been thought that albuminuria is more often seen in small cirrhotic livers than in larger ones, but no dogmatic decision as to this is at present justified.

When acute changes in the liver-cells are superadded to cirrhosis, leucin and tyrosin may appear in the urine. The urotoxic coefficient has been found to be increased.

Œdema of the feet is often referred to the intra-abdominal pressure of ascites impeding the flow of blood through the inferior vena cava. But this mechanical explanation will not, at any rate, fit all cases, for œdema of the feet may come on before and independently of ascites. In such cases the toxic origin of œdema may very reasonably be invoked; it has been suggested that a poison with a lymphagogue action is produced, and that the œdema is due to this factor. In some cases œdema may be cardiac, and the result of dilatation of the heart and mitral regurgitation. In an alcoholic subject the heart may dilate after a debauch. Another possible cause for œdema of the legs is alcoholic neuritis; a certain degree of alcoholic neuritis is probably commoner in cirrhosis than is generally recognised.

General œdema is very rare in cirrhosis, but œdema may creep up on to the abdomen and appear on the back.

Symptoms.—The early symptoms in cirrhosis are referable to the alimentary canal, and indicate gastro-intestinal catarrh. This catarrh is partly due to portal obstruction, with resulting venous engorgement of the stomach and intestines. There is often, in addition, dyspepsia of an alcoholic type with morning sickness, showing that the abuse of stimulants has a good deal to do with the symptoms. Chronic pharyngitis and laryngitis, with their attendant symptoms, are minor but frequent accompaniments of cirrhosis.

Digestion is slow and assimilation is impeded, so that the patients lose flesh and get thin. Flatulent dyspepsia is not uncommon, and the bowels are irregular; diarrhœa may alternate with constipation. Late in the disease, when there is toxæmia, diarrhœa may set in and carry the patient off.

Generally the temperature is not raised; but in cases where the disease advances rapidly there may be continued fever, while active tuberculosis or other complications produce a similar effect.

*Hæmatemesis* often comes on without any evidence of gastritis; at other times it is immediately preceded by pain, heaviness in the abdomen, and malaise. The patient feels faint, and shortly afterwards brings up a large quantity of blood, often partially clotted. The blood is darker in colour than that brought up in gastric ulcer, but not so altered as the "coffee-ground" vomit of carcinoma of the stomach. Usually there is a single large hæmatemesis, but it may be followed by a second. If hæmatemesis is repeated several times at short intervals there is probably a bleeding varicose vein at the lower end of the œsophagus, or a small abrasion of the mucous membrane of the stomach; these are the cases that may prove fatal. In 60 cases of fatal gastro-intestinal hæmorrhage in cirrhosis, Preble found that in no less than a third of the cases death took place on the first occasion, and in 80 per cent of the cases there were varicose veins in the œsophagus.

Ordinarily the hæmatemesis of cirrhosis does not give rise to such severe collapse as that of gastric ulcer, and is rarely fatal. While there may be some general tenderness over the stomach due to gastritis, there is no localised area where pressure gives rise to severe pain as in gastric ulcer. Gastric and duodenal ulcer are very rare accompaniments of cirrhosis. The bleeding may be due to gastritis, to small erosions or abrasions, or to rupture of varicose veins in the œsophagus, or, in rare instances, in the stomach. It is commonly assumed that there is a general and gradual capillary oozing of venous blood from the rupture of capillaries in the gastric mucosa, but it is probable that some inflammatory or destructive change in the mucous membrane is necessary to allow of this.

Hæmatemesis would be much more frequent were it merely the mechanical result of increased pressure in the portal circulation.

A cause of gastritis that is often overlooked and may lead to hæmatemesis, is bad teeth with pyorrhœa alveolaris; the pus teeming with micro-organisms is swallowed, and readily gives rise to changes in the gastric mucous membrane.

With hæmatemesis there is generally melæna; melæna may occur without hæmatemesis when the amount of blood poured out into the stomach is not excessive.

Hæmatemesis is usually a comparatively

early symptom of cirrhosis, and is often the first indication and warning of grave disease that the patient receives. But it may occur late in the disease, and even prove fatal when there is ascites.

For the diagnosis of hæmatemesis from different causes the reader is referred to the article "Hæmatemesis" in vol. iv. p. 16.

The treatment of hæmatemesis is absolute rest to the stomach and perfect repose in bed. A hypodermic injection of morphia is often useful in keeping the patient quiet.

Feeding should be carried on by the bowel, suppositories being given every four hours, and five or six injections of 10 ounces of water in the twenty-four hours to relieve thirst. After three or fours days, if there is no recurrence of hæmorrhage, beef-tea and peptonised milk can be given by the mouth. On the second or third day, if there is no recurrence of hæmatemesis, a blue pill and a saline purge should be given to remove the blood from the intestines.

Recurrence of hæmatemesis should be treated by return to rectal feeding, and by the administration by the mouth of a drachm of Ruspini's styptic (which is largely composed of gallic acid) in 1 ounce of water.

*Melæna*, besides being due to gastric hæmorrhage, may be due to similar oozing from the surface of the mucous membrane of the bowel.

A certain amount of blood may be mixed with the fæces as the result of small hæmorrhages, analogous to those seen in the skin, and due to hepatic insufficiency.

Piles are not infrequent in cirrhosis, and may give rise to hæmorrhage.

*Epistaxis* often occurs in the course of cirrhosis; it may pass backwards and simulate hæmoptysis. Like the small hæmorrhages into the skin, epistaxis is due to a toxæmic condition of the blood brought about by hepatic insufficiency; the poisons produced in the alimentary canal not being stopped by the liver, pass into the general circulation.

Oozing from the gums is due to the same cause.

Blood may be hawked from the back of the throat and be thought to have come from the lungs. Occasionally, bleeding occurs from the larynx. Hæmoptysis may be due to pulmonary tuberculosis, which is a well-recognised complication of cirrhosis; collapse of the bases of the lung, due to compression by ascites or by a pleural effusion, may also cause hæmoptysis.

In the early stages of cirrhosis in women metrorrhagia is often seen; later there is generally amenorrhœa.

*Jaundice.*—An attack of catarrhal jaundice may occur during cirrhosis; but continued or deep jaundice is very rare, and the black jaundice of malignant disease is never reached. A terminal jaundice, due to acute degenerative changes in the liver-cells, is sometimes seen.

A slight degree of icterus is, however, often seen, the conjunctivæ being tinged with light yellow for a time instead of their habitual dirty hue.

*Nervous Symptoms.*—In the late stages a toxæmic condition analogous to uræmia sets in, and the patients usually become drowsy, apathetic, and comatose; but sometimes there is delirium, which may be so active that there is considerable trouble in keeping the patient in bed. When drowsy and quiet the patient becomes careless, passes his motions under him, and it may be difficult to keep the skin of the back intact and prevent the onset of bed-sores. Cases have been described where children have presented symptoms like the juvenile type of general paralysis during life, with entire latency of advanced cirrhosis of the liver. The symptoms due to a toxæmic condition of the blood chiefly affect the brain, but slight degrees of peripheral neuritis are probably often overlooked.

*Complications.*—As has already been pointed out, pulmonary tuberculosis is often met with in the bodies of those dying from cirrhosis; often there are no clinical signs of the tubercle, but its presence should be suspected when there is fever without any satisfactory cause. Sometimes the progress of pulmonary tuberculosis is so rapid and emphatic that it throws into the shade the existence of cirrhosis, which is only revealed at the post-mortem. Pulmonary tubercle and alcoholic neuritis may be found as concomitant complications in cirrhosis, especially in women. Pulmonary tuberculosis is less often seen in children with cirrhosis than in adults with the disease, probably because alcoholism is less frequent in children.

Right-sided pleurisy frequently complicates cirrhosis; it has been suggested that infection may spread through the diaphragm. It should be remembered that considerable dulness at the base of the right lung may be due to a large cirrhotic liver, or to upward displacement of the liver by abdominal distension without any pleural effusion. In rare instances pleural effusions in cirrhosis are hæmorrhagic; this is due to tubercle.

Tuberculous peritonitis is another complication that is especially liable to occur in the course of cirrhosis, and it may very easily escape detection, the effusion being naturally regarded as that due to cirrhosis.

A number of acute infections may occur, such as erysipelas, pericarditis, pneumonia, infective endocarditis, and especially peritonitis.

When an acute infection attacks the liver itself icterus gravis results from the acute degenerative changes induced in the liver-cells.

Thrombosis of the portal vein shows itself by the rapid development of ascites; if the clotting

extends into the mesenteric branches it may give rise to melæna and a paralytic state of the bowel imitating intestinal obstruction.

In some instances cardiac failure occurs, and may become so prominent that the existence of cirrhosis is obscured or not detected until after death. Occasionally sudden death results from this cause.

Diagnosis.—In the pre-ascitic stage, before hæmatemesis has occurred, cirrhosis may be suspected from enlargement and tenderness of the liver, and enlargement of the spleen, in an alcoholic subject, with dyspepsia.

When hæmatemesis has occurred it must be differentiated from gastric ulcer, and especially from that extensive and somewhat latent form of ulcer met with in men between forty and fifty years, and often associated with arterio-sclerosis. Though it is easy to recognise a gastric ulcer in a young woman with all the classical symptoms and signs, it may be very difficult in men, for extensive ulceration may exist without much tenderness. These patients are more anæmic than in cirrhosis, and complain more of pain, while the spleen is not enlarged.

In malignant disease of the stomach the tumour may not be felt; but, in that case, there are likely to be signs of pyloric obstruction, and hæmatemesis is scanty and like "coffee-grounds."

When ascites has supervened, the other conditions that may equally give rise to this must be considered and eliminated (*vide* article "Ascites"). The ascites of cirrhosis is peculiar in that it seldom requires tapping more than twice, while in chronic peritonitis, and most other forms of ascites, it may be called for again and again.

In the late stages of cirrhosis, with cachexia and emaciation, it may be very difficult to eliminate cancer of the liver until the fluid is removed by paracentesis. A large knobby liver with umbilication of the nodules points to malignant disease; a small liver with splenic enlargement, to cirrhosis.

Syphilitic disease should be suspected when there are signs of syphilis elsewhere in the body, and a vigorous course of antisyphilitic remedies should be prescribed. But outward signs of syphilis may be wanting, and the proof of syphilitic disease of the liver may only be found in recovery under iodides.

When the patient first comes under observation, with some complication such as cardiac failure, phthisis, pleural effusion, and so forth, the existence of cirrhosis may not be suspected at first.

Prognosis. — The prognosis of cirrhosis is extremely bad at a late period of the disease, and when emaciation and ascites have developed, the patient's days are, as a rule, numbered.

Cases of undoubted cirrhosis, in which tapping has been followed by improvement and latency of the disease for years, have, it is true, been met with. If the patient's general condition and nutrition remain good, ascites is more likely to be recovered from than in the ordinary run of cases, where the patient is cachectic by the time ascites has appeared. The prognosis is very much better in the early stages of the disease, and a patient who has suffered from hæmatemesis may, by strict obedience to medical treatment and directions, escape from any further symptoms. On the other hand, the terminal symptoms of cirrhosis, such as ascites, œdema of the legs, and toxæmia, may come on suddenly, and sometimes without any very apparent cause.

The latency of symptoms depends on compensatory mechanisms: (1) the collateral circulation; and (2), probably most important, hyperplasia of the liver-cells.

When this compensatory hyperplasia has occurred the liver becomes larger, while the spleen becomes smaller. There is, however, the danger that the areas of hyperplastic liver-cells may undergo degeneration, or become invaded by fibrosis, and, by sharing in the cirrhosis, lead to a recrudescence of the symptoms.

The prognosis depends in great part on the patient's method and conduct of life, and is, of course, made worse by any complication, such as phthisis or renal disease, diseases which may kill the patient without any marked hepatic symptoms. The activity of the kidneys, or renal permeability, is an important element in the prognosis. As long as the kidneys carry off the toxic bodies that the cirrhotic liver allows to pass into the general circulation, the patient is in a fairly satisfactory state; but failure of the renal excretion entails hepatic toxæmia, which is analogous to urinary toxæmia.

Treatment.—The fibrotic condition of the liver cannot be removed by the administration of drugs, such as iodides or chloride of ammonium. Although the disease cannot be cured, it may become latent. The objects of treatment, therefore, should be (1) to allow the development of the compensatory mechanisms which enable the disease to become latent; and (2) symptomatic.

In the first place, any factors that lead to or favour cirrhosis must be removed. Alcohol must be cut off entirely; on the patient's power of will to become a total abstainer his future will largely depend. Medicines, if necessary, should not contain tinctures, alcoholic extracts, or be flavoured with spirituous compounds. If the patient's condition absolutely demands alcohol, it should be taken largely diluted after meals.

The diet should be restricted to milk, of which three or four pints should be taken daily; it may be mixed with Vichy, Apollinaris, Vals, or soda water. When improvement occurs, fish

diet may be taken. Abstinence from spicy, rich, and irritating food is most important, as fermentation and absorption of toxic products are thus minimised. Milk fulfils these essentials, and is, moreover, a good diuretic. Fatty and sugary foods have the disadvantage that they may lead to dyspepsia and the production of fatty acids.

Meat and much proteid food, tea and coffee, are also harmful.

Intestinal catarrh and fermentation should be prevented; although antiseptics, such as salicylate of bismuth, salol, β-naphthol, may be employed with this object, it is probably better to use a simple saline, such as magnesium or sodium sulphate with small doses of calomel, scammony, or euonymin. Water should be taken freely, so as to stimulate diuresis and excretion of toxic products.

Plenty of fresh air, and, unless there is ascites or some other deterring element, moderate exercise should be recommended, so as to improve the general health and resistance. A course at Carlsbad, Vichy, or Marienbad may be taken with benefit.

*Drugs.*—Iodide of potassium is usually given, and, no doubt, does good in syphilitic disease of the liver simulating cirrhosis. Many writers believe it does good in genuine cirrhosis. It is better to give the less depressing iodide of sodium. Chloride of ammonium is an hepatic stimulant and may be given a trial, but it is not more successful than iodides.

Arsenic should be avoided. There is no drug that has the power of stimulating the compensatory hyperplasia of the liver-cells.

Symptomatic treatment is necessary in hæmatemesis (*vide* p. 458), ascites, and in the terminal toxæmia.

*Ascites.*—When ascites gives rise to any embarrassment of respiration, to collapse of the bases of the lungs, or to hæmoptysis, the abdomen should at once be tapped. Formerly tapping was postponed as long as possible, because peritoneal infection was sometimes thus set up; but with strict antiseptic precautions this objection no longer holds, and paracentesis should be done, since the mechanical or pressure effects of ascites are harmful. The trocar should be a small one of Southey's pattern; the large trocars formerly employed removed the fluid very rapidly, and thus sometimes produced collapse. The trocar is usually introduced in the linea alba between the umbilicus and the pubes in a dull area; care should be taken to avoid puncturing a distended urinary bladder. It has been suggested that the trocar should be introduced to the left of the middle line, so as to avoid wounding the cæcum or liver, but this is hardly necessary. The fluid should be allowed to drain away through an india-rubber tube for twelve to eighteen hours. During this operation a bandage or binder should be applied to the abdomen and tightened from time to time; it should be kept on for some days after paracentesis. Continuous drainage has been tried, but is not successful, and may be dangerous.

When the ascites is comparatively small, it is worth while trying to remove it by purgatives and diuretics. It is probable, however, that they largely do good by removing toxic bodies from the organism, which are the cause of the ascites.

Saline purges, such as magnesium sulphate or jalap powder, have been commonly employed. Strong purgatives are not without the danger that they may set up or increase intestinal catarrh and exhaust the patient's strength, and so do more harm than good. Calomel in $\frac{1}{4}$ to $\frac{1}{8}$ grain doses, euonymin, and scammony may be tried.

*Diuretics.*—Copaiba is often successful, but has the disadvantage that it may set up gastric disturbance. A pill containing mercury, squills, and digitalis is a good preparation and may be safely employed. Digitalis, caffeine, bitartrate and acetate of potash, and spirits of juniper have been recommended, but, with the exception of the first, are of rather doubtful benefit. Recently urea, extract of liver substance, and asparagus have been said to have had some success. Personally I have found urea very disappointing.

The diuretic action of milk has already been pointed out. No attempt to restrict the amount of fluid taken should be made.

At the same time that moderate purgation and diuresis is being induced, iodide of potassium should be persisted in on the chance of the disease being in reality syphilitic.

The surgical treatment of ascites has recently been introduced by Morison and Drummond; the abdomen is opened, and artificial peritoneal adhesions set up by rubbing the opposed surfaces of peritoneum, and fixing the omentum to the parietal peritoneum. The avowed object was to increase the collateral circulation between the portal and the general systemic veins (*vide* p. 454), but it may act by increasing the nutrition of the liver-cells, and allowing them to undergo compensatory hyperplasia. The operation is on its trial; of 15 cases submitted to this treatment 5 have been cured, while improvement for a time has taken place in a few of the remainder.

When toxæmia becomes marked, very little can be done. The bowels should be kept freely open, the kidneys should be stimulated by diuretics, and plenty of water should be given by the mouth or by enemata. Intravenous transfusion is followed by temporary improvement.

Hæmorrhages and itching of the skin may be combated by calcium chloride, grs. xx. given for a few doses.

### On the Occurrence of the Various Forms of Cirrhosis in Early Life

The various forms of hepatic cirrhosis that may be met with in children have been described elsewhere, but it may be useful to summarise the facts briefly here.

The pericellular cirrhosis of hereditary syphilis and the lesions of tardive hereditary syphilis are fully dealt with (p. 476), and it is there pointed out that after recovery from pericellular cirrhosis the liver is probably left with its resistance so diminished that it may readily become affected by ordinary portal cirrhosis, the resulting change being neither due to syphilis nor curable by antisyphilitic treatment, but disposed to by the influence of former syphilis, and therefore parasyphilitic and comparable to locomotor ataxia and general paralysis of the insane. Some cases of marked portal cirrhosis in early life may thus be distantly related, though not directly due, to syphilis.

In rickets some slight fibrosis may occur in the liver, but it is never marked, probably transitory; it is hardly worth while to speak of rickety cirrhosis.

Both portal and hypertrophic biliary cirrhosis are met with in children, and the symptoms conform fairly well to those seen in adults.

In atrophic cirrhosis there is occasionally an absence of all hepatic symptoms and the presence of marked nervous manifestations, so that the existence of cirrhosis is quite unsuspected during life. Such cases have been recorded by Ormerod and Homén, but it is possible that the cases were juvenile general paralytics with a parasyphilitic cirrhosis of the liver.

Pulmonary tuberculosis is, I believe, much rarer in portal cirrhosis of children than in adults; but, on the other hand, tuberculous peritonitis is not an infrequent complication in children.

Hypertrophic biliary cirrhosis, which, under ordinary conditions, occurs earlier in life than portal cirrhosis, may present special features when it occurs in infants. The biliary cirrhosis of Brahmin infants around Calcutta, and the juvenile type of hypertrophic biliary cirrhosis described by Gilbert and Fournier, have already been referred to. It is noticeable that some cases of hypertrophic biliary cirrhosis in children run a very protracted course, and that sometimes the type changes and eventually presents many of the features of portal cirrhosis.

Marked monolobular cirrhosis accompanies congenital obliteration of the bile ducts (*vide* vol. iii. p. 372), but the symptoms are those of biliary obstruction. Cases of hepatic pseudo-cirrhosis, cardiac cirrhosis, and cardio-tuberculous cirrhosis (*vide* chronic venous engorgement of the liver), are chiefly met with in children.

### Degenerations and Infiltrations

Fatty Liver . . . . . 462
Lardaceous Liver . . . . . 464
Pigmentary Change . . . . . 464
Calcareous Infiltration . . . . 465
Leukæmic Infiltration . . . . 465

### Fatty Liver

Fatty Infiltration and Degeneration . 462
Causation . . . . . 462
Morbid Anatomy . . . . . 463
Signs . . . . . 463
Symptoms . . . . . 463
Diagnosis . . . . . 463

This includes the two conditions of (i.) fatty infiltration, and (ii.) fatty degeneration. Although this article is only concerned with a pathological increase of fat in the liver, it may be well to state what is meant by the terms fatty infiltration and fatty degeneration.

Fatty Infiltration or accumulation is an exaggeration of the physiological storage of fat in the liver-cells; fat is normally present in small amounts in the liver-cells of young children, sometimes in healthy persons who have died from the effects of accidents, and commonly in obesity. Histologically, the cells at the periphery of the lobules of the liver are first and chiefly affected, and contain globules of fat of considerable size; the protoplasm and the nucleus of the cell are mechanically displaced by the fat, and are not chemically altered or degenerated. When the fat is removed the cells return to their normal state.

In Fatty Degeneration the protoplasm of the liver-cell degenerates and undergoes a retrograde metabolism; as a result, globules of fat, at first of small size, appear scattered throughout the cell substance. The nucleus remains in its normal position; after removal of the fat the cell appears shrunken, its protoplasm granular, and the nucleus fragmentary. Fatty degeneration may occur in any part of the hepatic lobule, and may begin first in the central zone.

These two conditions run into each other, and in practice it is in many cases difficult to draw a hard and fast line between fatty infiltration and fatty degeneration. It is therefore better to speak of pathological fatty change in the liver.

Causation. — Pathological fatty change in the liver is met with in a number of conditions.

(1) *Poisons.*—Alcohol. Fatty change is very definitely related to alcoholic excess, and experiments show that alcohol may be regarded as a protoplasmic poison. Phosphorus, arsenic, antimony, iodoform, the mineral acids, oxalic, tartaric, and other acids, lead to marked fatty change.

Phloridzin gives rise to fatty change, which like that due to phosphorus has been regarded as an infiltration and not a degeneration.

(2) In certain intestinal diseases where toxins are absorbed from the alimentary canal and pass into the portal vein, fatty change in the liver is often found; thus it is frequently seen in gastro-enteritis and intestinal affections of children; in these conditions it may be said to be due to auto-intoxication.

(3) It also occurs in hæmic infections and intoxications as a further stage of cloudy swelling. It is seen in grave anæmias, where the deficient blood-supply and want of oxygen are, as well as toxic bodies in the blood, factors of importance in the production of the fatty (degeneration) change. It is also seen in some acute infections, in diabetic coma and the status epilepticus (Mott).

(4) In pulmonary tuberculosis a fatty liver is frequently found, and is a striking feature in the emaciated bodies of the victims of this disease. It is probably due to the action of toxins absorbed from the lungs. Peron's experiments showed that the intravenous injection of sterilised cultures of virulent tubercle bacilli lead to extensive fatty change in the liver; Carrière's results directly opposed Peron's, and suggest the possibility that in man the fatty change is the result of secondary streptococcal infection. It is at any rate unlikely that (i.) the fatty liver is due to the cod-liver oil so constantly given now, since the condition was noticed before its introduction (Wilson Fox); or (ii.) entirely to a deficient supply of oxygen, inasmuch as there is no special degree of fatty change in emphysema and allied conditions.

(5) Deficient blood-supply, as in anæmia, lardaceous disease, etc., probably leads to fatty degeneration; but in grave anæmia the presence of toxic bodies in the blood must also be considered.

Morbid Anatomy. — The liver is usually enlarged, sometimes, as in phosphorus or iodoform poisoning, very markedly; considerable fatty change may, however, be present in a liver of normal size. The surface is smooth and the edges are rounded; its consistency is usually firmer than normal, though sometimes from post-mortem changes it is very soft.

The specific gravity is diminished, and in some instances the liver may actually float in water. On section the knife becomes greasy, while pieces of the liver held in a flame may splutter and burn from the large amount of oil in the organ.

The cut surface is anæmic, yellowish-white in colour, and may show exaggeration of the lobular arrangement, suggesting fine cirrhosis. A microscopic examination is sometimes required to settle the question whether cirrhosis is present. There is often slight apparent fibrosis from atrophy of the liver-cells.

Fatty change frequently complicates other lesions of the liver, such as portal cirrhosis, nutmeg liver, and lardaceous disease. The histological changes have already been described (p. 462).

Signs.—In cases where there is general obesity the liver may be made out by percussion to be enlarged; but it may be difficult to feel the edge distinctly, both because the abdominal walls are overloaded with fat, and because during life the enlarged fatty liver is soft.

The skin is greasy, the tension of the pulse probably low, and the heart sounds distant or feeble. Fat women often have remarkably small chests, and in the dead-house the contrast between the enormous fatty covering and the size of the thorax is striking.

In cases where a fatty liver is associated with definite disease, such as pulmonary tuberculosis, the liver is enlarged and smooth, but is less firm than in lardaceous disease or cirrhosis, and therefore not so easily felt.

The spleen is not enlarged. There is no ascites or jaundice.

Addison laid stress on the condition of the skin accompanying fatty liver—bloodless, looking like fine polished ivory, almost semi-transparent, and exquisitely smooth, like satin. This change was earliest seen and best marked on the backs of the hands.

Addison also referred to recurring attacks of œdema in cases of fatty liver, especially when the patients were alcoholic. Possibly the œdema was due to peripheral neuritis or cardiac dilatation.

An excess of glycero-phosphoric acid in the urine derived from lecithin, which is present in large amounts in fatty livers, has been described (Lepine et Eymennet).

*Symptoms.*—The symptoms met with in cases of fatty liver are those of the condition or disease responsible for the secondary change in the liver. No doubt the various functions of the liver are not so well performed as they would be if the cells were healthy, but there is no constant or pre-eminent failure of function. If the degeneration is very acute and at the same time extensive, the symptoms would approach those of acute atrophy, although actually the liver is much larger than normal. But the condition then ceases to be one of ordinary fatty liver.

The stools are light, and the biliary secretion, though it does not cease, is probably deficient. Jaundice does not occur in uncomplicated cases, and there is no portal obstruction, so that ascites does not occur, and there is no enlargement of the subcutaneous or retro-peritoneal veins.

Piles are said to occur, but the diarrhœa that was formerly thought to depend on fatty liver is probably the cause rather than the effect.

There is no pain associated with fatty liver.

*Diagnosis.*—Fatty liver may possibly be mistaken for—

(1) Leukæmic infiltration of the liver.—Here examination of the blood settles any doubt.

(2) Lardaceous disease.—The liver is much firmer than in fatty liver, and there may be signs of lardaceous disease of the kidneys (albuminuria), splenic enlargement, or diarrhœa.

(3) A cirrhotic liver, especially for an enlarged cirrhotic liver, with latency of the symptoms.—When there are no symptoms the diagnosis is very difficult, and turns chiefly on the surface of the liver. If it is smooth, fatty change is probable; while if irregular, cirrhosis is indicated. In numerous instances fatty change is associated with cirrhosis.

(4) A displaced liver, if movable, is at once recognised; but if displaced by some cause, such as a pleural effusion or pneumothorax that is not detected, a further mistake is not improbable, and it might be regarded as a large fatty liver.

(5) Enlargement of the liver due to a deep-seated hydatid cyst or abscess. Here the liver is much more prominent and more easily felt and mapped out, while there may be signs of pressure or fever.

*Treatment.* — The primary cause, such as obesity or pulmonary tuberculosis, and not the liver, should be treated.

### Lardaceous Liver

In lardaceous disease the liver is less frequently affected than the spleen and kidneys. Thus combining the statistics of Birch-Hirschfeld, Loomis, Dickinson, and Turner, in 645 cases of lardaceous disease the spleen was affected in 486, the kidney in 429, and the liver in 314.

The liver is uniformly enlarged, smooth, and painless; the edge is firm and regular. The enlargement may be very considerable, and even reach to the level of the iliac spines. No symptoms can be referred to lardaceous affection of the liver apart from the general symptoms of the disease.

It has been thought that ascites may be set up by lardaceous lymphatic glands in the portal fissure; ascites, however, is rare in uncomplicated lardaceous liver, and when it does occur is probably part only of universal œdema. Under treatment the hepatic enlargement has been known to diminish considerably.

Lardaceous change may, however, be associated with cirrhosis, gummata, syphilitic cicatrices, perihepatitis, or abscess. Jaundice, ascites, and pain may be due to such conditions complicating lardaceous disease.

Lardaceous disease of the liver may be expected in a patient with signs of lardaceous disease of other organs, such as enlarged spleen, albuminuria with a low tension pulse and no cardiac hypertrophy, dropsy, anæmia, and diarrhœa, where the liver is smooth and enlarged.

Signs of past suppuration, of syphilis, or chronic phthisis are important in concluding that in a given case hepatic enlargement is due to the lardaceous change.

A lardaceous liver must be distinguished from other causes of painless and uniform enlargement.

In the absence of anæmia, of some degree of wasting, and of the antecedents of the lardaceous change—prolonged suppuration and syphilis—of evidence of concomitant lardaceous change in the kidneys and intestines as shown by albuminuria, œdema, and diarrhœa, the probabilities are against the lardaceous change.

Fatty liver in phthisis may imitate lardaceous change in the liver; but the organ is not so firm, and other evidences of lardaceous disease are wanting.

A deep-seated hydatid cyst may push the liver forward, and give rise to enlargement like that of the lardaceous organ; but the general health is good, no cause for the change is forthcoming, and the other symptoms of lardaceous disease are absent.

A large fatty cirrhotic liver will probably be tender, or accompanied by pain or definite symptoms of cirrhosis, such as hæmatemesis.

In leukæmia the liver is often considerably enlarged, painless, and smooth; this is more frequently seen in the lymphatic than in the spleno-medullary form. Examination of the blood will at once settle any question between these two diseases. In the rare event of lymphadenoma giving rise to considerable hepatic enlargement, evidence of enlargement of the lymphatic glands elsewhere in body will probably be forthcoming, and the temperature may be hectic.

When lardaceous disease is combined with gummata or syphilitic cicatrices, the diagnosis from malignant disease may be difficult, and depends on the effect of antisyphilitic treatment, the evidence of syphilis elsewhere, and the more chronic course of the disease.

The treatment of lardaceous liver is that of lardaceous disease generally; when combined with syphilis or cirrhosis the lines of treatment are those of the complicating disease.

For the pathology, morbid anatomy, and other points the reader is referred to the article on "Lardaceous Degeneration," p. 316.

### Pigmentation of the Liver

In Hæmochromatosis . . . . . 464
In Anthracosis . . . . . 465
Microscopic Pigmentation in Various Conditions . . . . . 465

Hæmochromatosis. — In the condition described by v. Recklinghausen as hæmochromatosis there is very extensive destruction of the red blood corpuscles and deposit of pigment in various parts of the body, especially in the liver, pancreas, and skin. It has been suggested by Adami that this destruction of the red blood corpuscles is due to bacterial infection, and by Meunier that there is a toxic factor analogous to toluylenediamine at work. The deposit of

pigment in the liver and pancreas sets up chronic interstitial fibrosis. When the fibrosis in the pancreas has reached a certain stage diabetes is set up; the cases of bronzed diabetes described by Hanot and Chauffard, of which Anschütz has collected twenty-four examples, all in men, are therefore the final result of hæmochromatosis.

The liver is usually enlarged, presents the naked-eye and microscopic appearances of multilobular cirrhosis, and is pigmented. The pigment occupies the liver-cells, which become degenerated, the cells in the periphery of the lobule are chiefly infiltrated, but the entire lobule may be affected. The pigment is also found in the fibrous tissue of the organ. The pigment is of two kinds—(i.) iron containing hæmosiderin; (ii.) a yellow iron-free pigment, termed by v. Recklinghausen hæmofuscin.

The hepatic artery shows endarteritis obliterans.

Anthracosis, Silicosis, etc. — In rare instances particles of carbon and other foreign substances are found in cirrhotic livers. Cases have been described in coal and copper miners and stone-masons. Particles of silver have also been detected in the liver after its medicinal administration.

These conditions are all very rarely seen, and have no clinical importance.

Microscopic Pigmentation of the cells of the liver is seen in a number of conditions:—

(i.) Pernicious Anæmia.—The cells of the peripheral zones of the lobules contain free iron. When acted upon with ferro-cyanide of potassium and dilute hydrochloric acid, the pigment turns of a bluish-green colour.

(ii.) In some causes of leukæmia a similar infiltration of the cells of the peripheral parts of the lobules of the liver with free iron is seen. I have also seen the same pigmentation in lymphadenoma.

(iii.) In chronic venous congestion the "nutmeg" liver shows hæmoidin in and around the cells surrounding the intralobular vein. This deposit of pigment must be distinguished from that of pernicious anæmia, from which it differs both in its situation and micro-chemical reactions.

(iv.) In malarial cachexia—a condition approaching that of hæmochromatosis—the liver-cells may become pigmented and atrophied. This pigmentary change may be associated with cirrhosis.

(v.) In some cases of cirrhosis and of new growth in the liver the cells may show pigmentation without the existence of any general deposit of pigment in the body. Possibly this may be due to local chronic venous congestion, hæmorrhages, or hæmolysis.

(vi.) In biliary obstruction the liver-cells are degenerated, and occupied by granules of bile pigment.

(vii.) Local pigmentation with blood pigment occurs around the scars of old abscesses, gummata, and sometimes in the immediate neighbourhood of innocent nævi—"melanotic angioma."

*Calcification of the Liver.*—This condition is of no clinical importance, though pathologically interesting. It may be briefly referred to under two heads:—

(1) Primary Calcification.—This is extremely rare in man, but it is not infrequent in horses to find primary calcification of the branches of the hepatic artery. It has, however, been seen around the hepatic arteries in chronic interstitial nephritis (Brill and Lebman), and in a case of tuberculous hip disease, where it was thought to be due to the deposit of salts absorbed from the affected bones (Babes).

(2) Secondary calcification in gummata, in the cicatrices of old abscesses, and in the walls of hydatid cysts, is by no means uncommon. Calcification also occurs in the walls of chronically inflamed gall-bladders. A remarkable case of diffuse calcification of the liver, which had to be cut with a saw (Targett), was possibly secondary to syphilitic change.

Carrel has recorded a case where laparotomy was undertaken with the diagnosis of calcified gall-bladder, and a calcified psorospermial tumour was found.

Sometimes hard masses are found embedded and encysted in the liver substance. They are generally intrahepatic biliary calculi due to drying up of the contents of cystic dilatations of the bile ducts.

### Liver in Leukæmia or Leucocythæmia

The liver is frequently very greatly enlarged in cases of leukæmia; 5 or 6 lbs. is a common weight for the organ, but it has been found to weigh more than double this. Enlargement of the liver chiefly occurs in lymphatic leukæmia, the rarer form of the disease. There may be very advanced spleno-medullary leukæmia without any manifest hepatic enlargement.

The liver is smooth and uniformly enlarged; the increase in size depends on infiltration of the portal spaces with leucocytes; the infiltration can sometimes be easily seen around the larger portal spaces with the naked eye. In addition the individual lobules become separated from each other by crowds of leucocytes, so that the lobules are definitely outlined. The leucocytic infiltration is not limited to the portal spaces or, indeed, to the peripheral parts of the lobules, for the capillaries inside the lobules become stuffed with leucocytes, and in some cases the leucocytic infiltration of the lobules is very widespread. The liver-cells, especially in the centre of the lobule, show the effects of impaired nutrition, and may be fatty or atrophied. At the periphery of the lobule the liver-cells are sometimes seen to be infiltrated with free iron,

as in pernicious anæmia. Cirrhosis does not occur as the result of leukæmia. In some instances small white nodules like tubercles are seen scattered through the liver; microscopically they are composed of accumulations of leucocytes.

In the later stages of leukæmia ascites is not uncommonly present. It has been suggested that this may be due to pressure of leucocytic infiltration on the intrahepatic branches of the portal vein, or to pressure of enlarged glands in the portal fissure on the portal vein; but it seems to me more probable that it is due to some concomitant chronic peritonitis, and to the cardiac debility and altered blood state. It is possible that ascites might be in some degree determined by thrombosis in the terminal branches of the portal vein in the liver.

*The diagnosis* of leukæmic infiltration of the liver depends on an examination of the blood. This should be done in a doubtful case of painless hepatic enlargement, in order to prevent the disease being regarded as lardaceous disease, and treated with iodide of potassium.

*Prognosis.*—As leukæmic enlargement of the liver is a more constant result of the lymphatic form, and as this is more rapidly fatal than the spleno-medullary variety, the prognostic value of hepatic enlargement in leukæmia is of bad omen.

*The treatment* is, of course, that of leukæmia.

### Innocent Tumours

Adenoma of the Liver—
(i.) *True* . . . . . 466
(ii.) *Multiple Adenoma in Cirrhosis* . 466
Angioma . . . . . 468
Lipoma, Teratoma, etc. . . . . 468

### Adenoma of the Liver

This subject is divided into two—(1) true adenoma; (2) so-called multiple adenomata, which may be considered as a compensatory hyperplasia of the liver-cells occurring in cirrhosis of the liver.

*True Adenoma.* — An innocent encapsuled growth of epithelial cells may occur in the liver, but is decidedly rare; pathologically they are of great interest, but clinically they seldom attract attention.

True adenomata may, theoretically, be divided according to their structure into—

(i.) Those composed of liver-cells, or of cells derived from the ordinary cells of the hepatic parenchyma.

(ii.) Those derived from the bile ducts.

(iii.) Those due to the inclusion of adrenal "rests."

(i.) An adenomatous tumour composed of liver-cells, apart from the multiple growths of this kind seen in association with cirrhosis, is very rare. Such growths may be spoken of as acinous adenomata in contradistinction to those derived from the bile ducts. Mahomed described a localised collection of cells surrounded by a fibrous capsule embedded in the liver, which was "nutmeg"; the tumour did not share in this general change. I have seen one similar specimen. Hale White refers to an adenoma 1¼ inch in diameter projecting from the surface of the liver; the specimen is in the Guy's Hospital Museum. Specimens have also been described by Engelhardt and others.

Possibly these tumours, which are pathological curiosities, may be due to some piece of liver substance separated during fœtal life from the main mass of the liver becoming subsequently embedded in the organ. Not infrequently small projections of liver substance, miniature lobes, are seen on the under surface of the liver; if these became implanted in the substance of the liver, the appearance of an encapsuled adenoma, composed of liver-cells, would be produced. Cristiani refers to the existence of multiple nodules of hepatic tissue embedded under Glisson's capsule, which have been explained as congenital, and due to the inclusion of tiny lobes.

Multiple areas of hyperplasia of the liver-cells or adenomatous formations have been seen in a nutmeg liver without any cirrhosis (Jacobi).

(ii.) An adenomatous tumour derived from the bile ducts. A papilloma springing from the inside of the extra-hepatic bile ducts would come under this heading.

At present tumours arising from the bile ducts, indenting and displacing but not invading the surrounding liver substance, concern us; they may be described as tubular adenomata. They may be single or multiple.

*Single.*—A single adenoma of the bile ducts may reach a size sufficient to imitate a tumour such as a hydatid or floating kidney. Cases have been recorded by Keen, Koenig, and Schmidt. Keen removed a cystic adenoma thought to be derived from the bile ducts from a women aged thirty-one, in 1891; as she was alive in 1899, the exceptional nature of the growth cannot be explained by supposing it to be a carcinoma. Clinically it was thought to be a floating kidney.

*Multiple.* — In rare cases multiple small tumours are met with, imitating the structure of bile ducts (v. Hippel).

It has been thought by some that the condition of multiple cystic disease of the liver is really a fibro-adenoma derived from the bile ducts.

(iii.) Possibly an included adrenal rest might give rise to a tumour that would be best described as an adenoma.

### Multiple Adenomata in Cirrhosis

Synonyms: *Cirrhosis complicated with adenoma, Cancer with cirrhosis; Nodular cirrhosis.*

*History.* —This condition was studied by Rokitansky, Kelsch and Kiener, Sabourin, Cornil and Ranvier, Hanot, and others.

*Nature of Multiple Adenomata.*—Cornil and Ranvier regarded the development of the adenomatous tumours as a complication of pre-existing cirrhosis. Hanot and Gilbert, on the other hand, believed the growths to be a special form of carcinoma, and in common with Lancereaux regarded the fibrosis of the liver as secondary to the irritation set up by these growths. Brissaud speaks of multiple adenoma as being a kind of half-way house between primary carcinoma and cirrhosis, while other writers (Sabourin, Engelhardt) regard the production of adenomata as due to the same causes that give rise to cirrhosis, but acting on the epithelial instead of on the fibrous part of the organ. In dogs, tumours due to hyperplasia of the liver-cells, and probably set up by infection, are far from rare; a few cases of a similar nature without any cirrhosis have been described in man.

These multiple growths are, I believe, practically always part of cirrhosis; they are exaggerations of the hobnails seen in ordinary cirrhosis, and further represent an attempt at compensation on the part of the more healthy liver-cells which have undergone multiplication (compensatory hyperplasia), and thus account for the increased size of the hobnails.

It is when these hobnails undergo fatty degeneration and necrosis, and appear white on section, that they are particularly liable to attract attention; for when this change has occurred they do not, unless bile-stained, suggest cirrhosis, but resemble multiple new growths. Fatty change and necrosis of the hyperplastic nodules are particularly likely to occur when thrombosis of the portal vein is superadded to cirrhosis; hence the frequency with which portal thrombosis is recorded as associated with multiple adenoma, cancer with cirrhosis, etc. Thus in 15 cases of so-called adenoma of the liver that were analysed by Dr. Ll. Powell, no less than 9 had thrombosis of the portal vein.

Those who regard the condition as one of primary carcinoma of the liver adduce the presence of hepatic cells in the portal vein and thrombosis as further proof of its malignant character. But the presence of hepatic cells in the portal vein does not prove that the growth is malignant; for the hobnails being poorly nourished, and having by rapid proliferation outgrown their blood-supply, soften down, and by discharging into the portal vein or hepatic veins may induce thrombosis.

The proliferation of the liver-cells may be due to one of two causes—very possibly to each of the causes at different stages of the disease.

(i.) The multiplication of the hepatic cells in the hobnails may be due to the same poison that stirs up the connective tissue of the liver to proliferation; this would be the case especially in the early stages of the disease. In cases of poisoning by mussels similar nodules are produced, evidently directly due to the irritation exerted by the poison.

(ii.) The multiplication of the liver-cells may be an attempt at compensation to make good the functional activity of the liver as a whole, which has been greatly reduced by the destruction of hepatic tissue.

It is *a priori* very reasonable to believe that the hyperplasia of liver-cells might become so vigorous as to pass into carcinoma. Probably this does occur, and so accounts for cases of multiple primary carcinoma of the liver. My own belief is that the cases described as multiple adenoma are all primarily cirrhosis, and that many of the cases of "cancer with cirrhosis" recorded by the French are not necessarily anything more than advanced cirrhosis, with hyperplasia of the liver-cells in the hobnails and a terminal thrombosis of the portal vein.

*Morbid Anatomy.*—The appearance of the liver is very striking, and suggests multiple secondary new growths, gummata, or even caseous tubercle. The surface of the liver shows numerous projecting nodules, which, however, are not umbilicated. They are white on section, usually dry and friable, but may, especially when there is associated venous thrombosis present, be softened. The surrounding liver substance may be deeply congested, so that the contrast between the hobnails and the rest of the liver still further suggests secondary malignant disease. The liver is usually somewhat enlarged in size, but may be smaller than natural.

The portal vein is frequently thrombosed, and microscopic examination of the clot may show liver-cells due to the discharge of one of the softened hobnails into the vein. Sometimes similar thrombosis is seen in the hepatic veins.

The lymphatic glands in the portal fissure are not enlarged.

Microscopically the liver shows marked cirrhosis, the masses that to the naked eye suggested new growth being seen to be altered liver-cells surrounded by a fibrous capsule; the interstitial tissue shows the appearance of pseudo-bile canaliculi, advancing cirrhosis, and sometimes extravasated blood. The normal trabecular arrangement of the liver-cells is lost; the cells are concentrically arranged, the more external layers being often flattened as if from pressure. The liver-cells vary somewhat in size; often they are large, occasionally they are multinuclear; the active karyokinesis of the nuclei is an important evidence of hyperplasia. Fatty degeneration of the cells and hæmorrhages may be met with, especially when thrombosis of the portal is present.

The symptoms of multiple adenomata are those of the disease of which it is only an epiphenomenon, viz., common cirrhosis. It is found in a high proportion of those cases where,

at the autopsy, cirrhosis with thrombosis of the portal vein is revealed.

### Angioma

The liver is more often the site of angiomata than any other viscus, but their occurrence is not common. Lancereaux, in an extensive experience, has seen twenty-five examples. They are more frequent in cats' livers.

They are usually single, but, like other innocent tumours, they may be multiple, and other organs besides the liver may be involved.

Though they may be congenital they are more often seen in patients of advanced years, and are then probably due to a combination of congestion and atrophy of the liver-cells. In early life they have occasionally reached a considerable size.

Angiomata are found immediately under the capsule of the liver, and most often on the convexity of the right lobe near the falciform ligament. They are of a deep red colour, at first sight like hæmorrhagic infarcts. After death they are collapsed, and somewhat depressed below the rest of the organ. As a rule the angioma fades gradually into the surrounding liver substance, but sometimes it is encapsuled by fibrous tissue. The surrounding tissue may be stained by blood pigment (melanotic angioma). The fibrous trabeculæ sometimes become much thickened (fibrous angioma), and so tend to lead to obliteration of the angioma. Thrombosis and organisation of the blood-clot in them may occur. It is possible that in some instances degenerative changes may result in an angioma becoming transformed into a serous cyst. Structurally they are cavernous angiomata.

They can be injected from the hepatic artery and from the hepatic and portal veins.

In a few cases an angioma of the liver is sufficiently large to give rise to signs of its presence. In a table of 75 cases compiled by Keen, where resection of the liver for various neoplasms had been performed, 4 were angiomata.

No distinctive signs or symptoms can be put down to their presence. It has been suggested that murmurs or venous hums heard over the liver may be due to them, but there is little proof in support of this view.

The only satisfactory treatment for the rare cases where there is a definite tumour, is removal by the surgeon.

### Lipoma, etc.

Genuine fatty tumours are not met with in the liver, but detached appendices epiploicæ may become indented on the convexity of the liver by the pressure of the diaphragm, and appear to be incorporated with the organ.

Localised areas of extreme fatty change in the liver-cells are sometimes seen as the result of vascular disturbances and microbic activity, but they have no resemblance to real fatty tumours.

A few instances of myxomatous tumours in the liver have been described, but it appears probable that they were really myxo-sarcomata and not pure mucous tumours.

Multiple congenital fibromata on the sympathetic nerves have been observed in the liver. The other recorded fibromata in the liver are probably either syphilitic, especially the remarkably fibrous formations described in hereditary syphilis by Marchant, or fibro-sarcomata.

*Teratoma.*—A unique specimen of a primary teratoma of the liver has been recorded by Musick. Implantation of a dermoid cyst on the surface of the liver, due to rupture of an ovarian dermoid, was described by Hulke.

In 10 cases of malignant abdominal teratomata collected by Montgomery there were 4 in which secondary growths occurred in the liver. This subject, however, belongs rather to the section on secondary malignant disease of the liver (see p. 481).

### Cysts of the Liver

Various kinds of cysts are met with in the liver.

(1) Parasitic cysts, hydatids, etc.

(2) Simple serous cysts, usually single, or present in small numbers. In size they are generally small; exceptionally, they are sufficiently large to be detected clinically. Possibly some of these latter are in reality sterile hydatid cysts. The walls of these cysts are smooth, and are made up of a fibrous capsule lined by epithelium. In the larger cysts the epithelium may be wanting; in the smaller ones it may be columnar, cubical, or flattened; ciliated epithelium has been met with, and has been thought to point to their origin from embryonic bile ducts (Musick). The cysts often contain the remains of dissepiments, showing that two or more originally separate cysts have united.

The cysts are probably due to local obstruction and distension of bile ducts; it is noticeable, however, that cysts are very rare in cirrhosis.

In the early stage bile is probably present, but disappears as time advances; the fluid may be clear, straw-coloured, green, or, from hæmorrhage into them, reddish-brown.

Other possible, but not very probable, origins for cysts are changes in the mucous glands of the bile ducts, dilatation of lymphatics, or degenerative changes in nævi.

The fluid is albuminous, and may contain blood or epithelial cells, hæmatoidin, bile pigment, cholesterin, or tyrosin.

Sometimes a few serous cysts in the liver are found to be associated with granular and cystic kidneys. Such cases form a transitional step to the multilocular cystic disease of the liver described below.

When, as is very rarely the case, a serous cyst is sufficiently large to give rise to clinical signs, it is indistinguishable from a hydatid cyst, and should be treated in the same way.

(3) In long-standing biliary obstruction the bile ducts in the liver become greatly dilated; at first they contain bile, but after a time they are found to be distended with clear mucous fluid.

(4) *Pseudo-Cysts.*—By the softening of adenomatous masses in cirrhosis cystic cavities containing degenerated liver-cells may result. Pseudo-cysts may also be produced by degenerative processes in masses of secondary malignant disease. In some instances of softened masses of growth the appearances are exactly like those of cysts. This has been observed in squamous-celled carcinoma and in sarcoma.

(5) Cystic adenomata of the bile ducts have in very rare instances been described (*vide* Adenomata, p. 466).

(6) In tuberculous disease involving the bile ducts cavities formerly spoken of as cysts may occur.

(7) Primary dermoid cysts do not occur in the liver; but from rupture of an ovarian dermoid, implantation on the surface of the liver has been known to occur (Hulke).

*Multilocular Cystic Disease.*—In this disease the liver shows multitudes of cysts, and thus differs from the serous cysts which are few in number or even solitary.

It is usually met with late in life; in 26 cases collected by Still, 17 were over fifty years of age, 4 over seventy, while the youngest adult was thirty-nine. A very few cases (3 or 4) have been met with in infants or still-born children who may be the subject of numerous malformations.

It occurs more often in women than in men, according to Still in the proportion of 3 to 1; of 28 cases 21 were in females.

Cystic disease of the liver is always accompanied by a similar and nearly always more advanced change in the kidneys. Cystic kidneys, however, are often met with without any manifest cystic change in the liver.

*Pathogeny.*—The mechanism by which cystic disease of the liver is brought about has given rise to a good deal of discussion. Space does not admit of a *résumé* of the subject, but the following views may be mentioned:—

(1) That it is an irritative or inflammatory process leading to pericholangitic fibrosis, and to dilatation and proliferation of the ducts themselves.

(2) That there is a diffuse new formation—a fibro-adenoma of the ducts.

(3) That vacuolation of the liver-cells occurs and, by fusion, forms cysts.

(4) That the condition is a malformation (Still). This view is analogous to Shattock's theory that congenital cystic disease of the kidneys is due to persistence and cystic dilatation of the mesonephrosis, the real kidney substance being included in and compressed by the fœtal persistence. Still believes that the cysts are derived from columns of hypoblast cells forming part of the original duodenal diverticulum, and not from the bile ducts.

Personally, I regard cystic disease as due to an irritative or inflammatory process around the bile ducts which gives rise to their dilatation.

*Morbid Anatomy.*—The liver may be greatly enlarged, though this is by no means always the case. The organ is riddled with cysts of various sizes up to that of a hen's egg; the larger ones are probably due to union of previously separate ones. The contents of the cysts may be clear or blood-stained, but do not contain bile pigment. The larger bile ducts and the gall-bladder are normal. In infants the cysts may be so small as to be overlooked unless a microscopic examination is made; the liver in these instances is not enlarged, but the fibrous tissue of the portal spaces is manifestly increased.

Microscopically the cystic spaces are lined by columnar or cubical epithelium, with an underlying layer of well-formed fibrous tissue. In children, branching tubes surrounded by fibrous tissue can be well seen spreading out from the portal spaces; in adults the fibrous tissue is old. The liver-cells may show an appearance suggesting vacuolation.

Clinically, cystic disease of the liver is usually overshadowed by the accompanying renal disease, and only discovered at the autopsy. The symptoms are those of chronic renal disease and arteriosclerosis. In some instances the liver may be recognised as enlarged, and has even simulated an ovarian cyst. If enlargement of the liver be found in a case where the kidneys are palpable as cystic tumours, cystic disease is highly probable. The renal enlargement is very likely to be regarded as hydronephrosis.

The treatment and prognosis are those of the renal disease.

INFECTIVE GRANULOMATA

TUBERCLE . . . . . 470
SYPHILIS . . . . . 472
*Acquired* . . . . . 472
*Hereditary* . . . . . 475
*Tardive* . . . . . 476
*Parasyphilitic Multilobular Cirrhosis* 477
LYMPHADENOMA . . . . . 477
ACTINOMYCOSIS . . . . . 477

HEPATIC TUBERCULOSIS

INTRODUCTION . . . . . 470
*Miliary Tuberculosis* . . . 470
*Local Tuberculosis* . . . . 470
(*a*) *Involving the bile ducts. Tuberculous Cavities or Tuberculous Cholangitis* . . . 471
(*b*) *Not involving the ducts.* "*Solitary Tubercle*" . . . 471

Tuberculous disease of the liver is of little clinical importance. Inasmuch as it gives rise to no characteristic symptoms it cannot be diagnosed during life, except in generalised tuberculosis, and then only on the grounds that the liver is in most instances affected along with the rest of the body.

The infrequency with which tuberculous lesions, other than miliary tubercles in the course of generalised tuberculosis, are found in the liver might suggest that the liver is specially inimical to the growth of the tubercle bacillus. It has, however, been shown experimentally by Sargent that the bile is not more antagonistic to tubercle bacilli than to other micro-organisms.

The most probable explanation why tubercle of the liver is comparatively rare, except as part of generalised tuberculosis, is that the liver does not lie in the direct line of the lymphatic vessels carrying lymph and tuberculous infection from the intestines. If it were the recipient of the lymphatics of the intestines it would probably suffer as frequently as the mesenteric glands. The lymphatic vessels in the portal fissure convey lymph out of the liver towards the lymphatic glands at the hilum, hence tuberculous infection from the intestine would have to extend against the lymph stream. This does take place in rare instances.

*Hepatic tuberculosis* may be divided into—

(i.) Miliary tuberculosis, part of a general hæmic infection.

(ii.) Local tuberculosis

(*a*) Involving the bile ducts.

(*b*) Solitary tubercle, not involving the bile ducts.

*Miliary Tubercles in Generalised Tuberculosis.* —In generalised tuberculosis the liver is practically always affected, though sometimes the miliary tubercles are few and difficult to detect. More recent and careful observations tend to show that the liver is very frequently affected in tuberculous disease of other parts of the body; thus it is said that miliary tubercles are present in the liver in 50 per cent of the fatal cases of phthisis (Zehlen); this roughly corresponds with the frequency of tuberculous ulceration of the intestines in phthisis.

In generalised tuberculosis the bacilli reach the liver by the hepatic artery, and give rise to a widespread eruption of grey miliary tubercles.

These grey tubercles, which are better seen on the surface of the liver than on section of the organ, are situated inside the lobules, and thus differ from the local tuberculous formations found in the portal spaces.

The liver is rather increased in weight and somewhat swollen. There may be some recent inflammation of the capsule of the organ due to the irritation of miliary tubercles.

There are no clinical signs or symptoms that can be relied upon to indicate the presence of miliary tubercles in the liver. Jaundice has occasionally been observed to coincide with the development of miliary tubercle in the liver in the course of phthisis and generalised tuberculosis; but this is so rare, and miliary tuberculosis so common, that it is an interesting rather than a valuable observation. The onset of jaundice in tuberculosis would certainly suggest hepatic infection, but the absence of jaundice would not contraindicate its existence. When tubercles are present on the capsule auscultation may reveal a friction rub.

*Local Tuberculosis.* — Under this heading come the cases where tuberculosis is more chronic, and leads to more advanced changes than in miliary tuberculosis.

A few words may be said about the sources of infection.

The weight of evidence is in favour of the view that the bacilli are derived from the intestinal tract, and carried to the liver by the portal vein. Sargent insists on the occurrence of tuberculous pylephlebitis and thrombosis in the portal spaces as a prelude to the development of tuberculous foci.

It has been suggested that tubercle bacilli from the duodenum pass up the bile ducts, work their way through the mucous membrane of the ducts into the portal spaces, and there give rise to the formation of caseous tubercles. This view, which on the face of it was improbable from the absence of motility on the part of the tubercle bacilli, has been disproved by Sargent's experiments of injecting tubercle bacilli into the bile ducts; these showed that unless the walls of the ducts were previously damaged, as by ligature, they did not allow tubercle bacilli to pass through them. It is noticeable that the extrahepatic ducts are not affected by tubercle except in the rarest instances, and that there is no condition of ascending or descending tuberculous cholangitis to correspond with tuberculous disease of the ureter.

A tuberculous gland in the hilum of the liver has been known to burst into the common bile duct.

It is doubtful whether tubercle is often conveyed into the liver by means of the lymphatic vessels, though tuberculous lymphatic glands in the hilum of the liver are, it is true, sometimes seen in cases of tuberculous enteritis.

It is also unlikely that tubercle bacilli pass in through the capsule, in cases of tuberculous peritonitis, sufficiently far to set up the tuberculous deposits.

In some cases tubercle bacilli in small quantities reach the liver by the hepatic artery—just as they are conveyed to bones that later become affected with tuberculous osteitis, without any accompanying acute generalised tuberculosis—and produce a local caseous focus of a chronic character in the liver.

*Tuberculous Cavities in the Liver*

SYNONYMS: *Local Tuberculosis in connection with the bile ducts, Tuberculous Cholangitis, Tuberculous Pericholangitis.*

This condition is probably not nearly so rare as the recorded cases lead one to suppose. The tubercle bacilli reach the liver by the portal vein, being obtained from the intestines, which in most of the cases show tuberculous ulceration. Sargent states that the intrahepatic branches of the portal vein show tuberculous pylephlebitis and thrombosis, and that, at a later stage, tubercles develop in the portal spaces.

The tubercles inside the portal spaces after reaching a fair size caseate, soften down, and eventually eat their way into the bile duct, into which they discharge their caseous contents in the same way that a pulmonary vomica opens into a bronchus. A local tuberculous cholangitis is thus secondarily brought about by the invasion of the duct from without; the tuberculous change does not spread to the large extrahepatic ducts. The communication between the duct and the emptied caseous cavity allows bile to enter into and stain its walls.

The liver is usually somewhat larger than natural, and on section shows a number of white caseous areas or of bile-stained cavities with caseous walls. In the earlier stages, before the tubercles have opened into the ducts, the tuberculous material is firm, and resembles and is therefore sometimes regarded as lymphadenoma; in the later (excavitation) stage, when it has opened into a bile duct, its walls have a greenish-yellow colour from bile-staining, and exceptionally of a purple colour from hæmorrhage. In their early stage the tubercles may be $\frac{1}{6}$-$\frac{1}{4}$ inch in diameter, while the cavities subsequently developed are larger, and may measure as much as an inch or even two inches across.

Structurally the masses are enclosed in a fibrous capsule representing the fibrous tissue of the portal space, and containing caseating granulation tissue surrounding a space which in its turn can be seen opening into a bile duct; the epithelium of the bile duct is usually well preserved except at the point where it has been destroyed by the perforation from without. The tuberculous process is therefore pericholangitic, not cholangitic.

*Symptoms.*—Since biliary obstruction to some extent must exist, it is remarkable that jaundice does not appear to occur. In some cases attacks of pain resembling in their character biliary colic, but without jaundice or bilious urine, have been noticed. Ascites does not occur, and nothing further is known as to the clinical results of this tuberculous lesion of the liver.

*Primary Tuberculosis of the Biliary Tract.*—As stress has been laid on the bile ducts being secondarily involved in the tuberculous disease of the liver, it ought to be mentioned that Lancereaux has described a case of tuberculosis of the common bile duct, gall-bladder, and cystic duct in a woman aged thirty-two years, which he regards as directly due to infection from the duodenum.

*Local Tuberculosis not involving the Bile Ducts. Solitary Tubercle*

Under the title "solitary tubercle" it will be convenient to describe caseous tuberculous masses embedded in the liver substance without any connection with the bile ducts Masses of this kind are often met with in the livers of animals, but are rare in man.

The fact that the masses do not open into the bile ducts suggests the probability that the tubercles have arisen in the substance of the liver as the result of bacilla conveyed to the liver by the hepatic artery—much in the same way as tuberculous foci are started in bone—and that the tubercle bacilli are not carried to the liver by the portal vein as in tuberculous pericholangitis, where the morbid process occupies the portal canals.

The recorded cases are curiously few. Moore in a recent paper only admits five; and of these two cases were of peculiar interest, in that in both caseous masses were found in the livers of patients dying of carcinoma of the pylorus. In both these cases the hepatic lesions resembled tubercle microscopically except in the absence of tubercle bacilli. It was thought that the absence of acid in the gastric juice had favoured the absorption of tubercle bacilli through the ulcerated surface of the stomach. As no tubercle bacilli were found, the possibility arises whether the caseous masses may not have been due to the activity of the pseudo-tuberculosis baccillus described by A. Pfeiffer and by Klein. Klein has recently shown that this bacillus, obtained from the water of the rivers Thames and Lee, produces caseous masses in the liver, lungs, and lymphatic glands of animals. It is, therefore, possible that some of these solitary caseous masses are not tuberculous.

My own belief is that solitary tuberculous masses are not nearly so rare as the recorded instances would suggest; I have seen at least two myself. These localised caseous masses of tubercle must be carefully distinguished from gummata, and are hardly likely to be imitated by actinomycosis.

Sometimes these solitary masses may soften down and form abscesses, and may then set up localised suppuration in the neighbourhood of the liver. In rare instances, as in a case related by Dr. T. L. Anderson, where there was a mass the size of a tangerine orange in the left lobe of the liver, they may be readily felt through the abdominal wall.

*The diagnosis* of these caseous masses is usually impossible; if these nodules are felt in

the liver of a patient with tubercle elsewhere, their true nature might be suspected. If they softened down and presented as a fluctuating swelling, the signs would be indistinguishable from those of an ordinary abscess.

SYPHILIS OF THE LIVER

ACQUIRED SYPHILIS . . . . . 472
HEREDITARY SYPHILIS . . . . . 475

ACQUIRED SYPHILIS

SECONDARY MANIFESTATIONS.—In the secondary stage of acquired syphilis jaundice is occasionally seen at the same time as the roseola; it appears to be due to the syphilitic infection, for it is amenable to mercurial treatment, and if untreated, does not pass off in the way that an accidental or intercurrent catarrhal jaundice would do. It is benign, and must be distinguished from the jaundice of icterus gravis, which sometimes supervenes in the secondary stage of syphilis. As to the cause of this jaundice there is considerable doubt; it has been variously suggested that it is due to an eruption on the mucous membrane of the bile ducts corresponding to that of the skin, to enlarged glands in the portal fissure, or to a generalised toxic disturbance of the liver, which may or may not lead to the generalised intercellular cirrhosis characteristic of the congenital form of the disease.

It is generally believed that diffuse pericellular cirrhosis is peculiar to congenital syphilis, and that it does not occur in the acquired disease. This is probably too absolute a statement, and its accuracy may well be questioned. It is true that it is very seldom seen, partly because the liver is less often affected in acquired syphilis than in the hereditary disease, and partly because opportunities for examining the liver during the secondary period only occur in rare and accidental instances. A diffuse pericellular cirrhosis is certainly present in some instances of acquired syphilis, even without any gummatous change, and is constantly seen around gummata that are not very old.

To sum up, in the secondary stage the liver may be so affected as to give rise to jaundice. This is rare; the jaundice is usually benign, but it may be due to acute degenerative changes supervening in the cells of a liver already affected, and its resistance impaired, by the baneful influence of the syphilitic toxin. Further, it is probable that a diffuse pericellular cirrhosis like that seen in congenital syphilis does occur; but apart from the possibility that it may in part be responsible for icterus, no clinical symptoms can be correlated with it.

THE TERTIARY MANIFESTATIONS OF SYPHILIS IN THE LIVER.—1. *Lardaceous Disease.*—Since the advent of antiseptic surgery, prolonged suppuration has become so comparatively infrequent that syphilis is now responsible for a much larger proportion of the cases of lardaceous disease. The subject is considered elsewhere (*vide* "Lardaceous Degeneration").

Here it may, however, be pointed out that lardaceous disease may coexist with gummata and cicatrices in the liver, and may thus lead to increase in size of the organ. Occasionally the lardaceous change is limited to an area round a gumma, thus suggesting its dependence on a toxin whose action is concentrated in and near the gumma.

*Gummata and Cicatrices.*—The liver is more often affected by these lesions than any other abdominal viscus. Their characters are so well known that there is rather a tendency to regard them as commoner than they actually are. Dr. J. L. Allen at my suggestion critically examined the post-mortem records of St. George's Hospital for forty-two years (1857-1898), during which period 11,629 autopsies were performed; he found 37 cases of undoubted gummata, and 27 additional cases in which cicatrices were present. There is, therefore, a contrast between the frequency of hepatic lesions in hereditary and its incidence in acquired syphilis.

*Disposing Conditions.*—Men are more often affected than women. Thus in a collection of 83 cases, 60 were males, and 23 females (Allen). It has been thought that any factor such as traumatism, alcoholism, malaria, or a past attack of jaundice, that would diminish the vital resistance of the liver, would render the organ more prone to gummatous disease.

The greater frequency of gummata on the anterior surface of the liver, which is more exposed to blows, the fact that gummata are not infrequently found close to the falciform ligament, where strain from falls must tell, and the increased incidence in the male sex, though this may be merely due to their being more often syphilitic, are points in favour of traumatism playing a part in the localisation of tertiary syphilitic lesions in the liver.

It is reasonable to believe that alcohol being a protoplasmic poison, syphilitic lesions would be commoner in the livers of the drunken than in temperate persons suffering from syphilis. It is curious, however, to note how seldom cirrhosis and gummata are found in the same liver.

*Morbid Anatomy.*—In its earliest stage the future gumma is a mass of syphilitic granulation tissue or a syphiloma; it is of a pink-grey colour, and does not show any central necrosis. As a result of impaired blood-supply depending on syphilitic endarteritis of the vessels, and probably also from an increase in the amount of the syphilitic toxin, the cells in the centre of the syphiloma die and undergo caseation. The term gumma or gummy tumour is now applicable, the caseous contents when softened having some resemblance to gum. In the

caseous material, crystals of cholesterin and stearic acid and granules of fat may be seen. In stained sections the caseous area has a homogeneous appearance, and takes the dye badly or not at all.

Near the caseous material giant cells are sometimes seen; their function is to absorb the debris; they are absent in old gummata, and are rarely so well developed or so numerous as in tuberculous formations.

The granulation tissue surrounding the caseous debris undergoes organisation, and forms a fibrous capsule around it, in which there may be found fresh elastic fibres. Spreading out from this into the liver tissue are seen small cell infiltration (intercellular cirrhosis) and bands of fibrous tissue, while the arteries enclosed in the capsule of the gumma show proliferation of the intima and narrowing of their lumen (endarteritis obliterans). In the immediate neighbourhood of the fibrous capsule pseudo-bile canaliculi are often seen, while the liver-cells are flattened and pressed out of shape. There are thus three zones in a gumma: (1) the central caseous material; (2) the fibrous capsule; (3) the extension of inflammation into the surrounding liver tissue.

In young gummata the fibrous capsule is indefinite, and there is extensive infiltration of the surrounding tissues, which may spread to the capsule of the liver, setting up perihepatitis and adhesions, and even infiltrate the diaphragm or the abdominal wall. In an old gumma there is little intercellular cirrhosis around it, the fibrous capsule is thick and is contracting on its caseous contents; these may gradually undergo absorption, and a scar is left. When undergoing absorption as the result of treatment with iodides, gummata may soften down; but this may also be due to secondary infection, and such a gumma may resemble a chronic abscess and even open into a bile duct.

Calcification of a gumma sometimes occurs, either of the caseous centre or of its capsule. A remarkable case of diffuse calcification of the liver recorded by Targett was probably secondary to gummatous infiltration.

*Cicatrices* are generally regarded as the remains of old gummata which have contracted up and undergone absorption; but it is probable that they may develop from masses of syphilitic granulation tissue without any preliminary necrosis and caseation. They are seen on the surface of the organ, especially on its convexity, as white depressions invading the substance of the organ for a short distance, being often conical in shape and tapering towards the interior of the liver.

*Situation and Results.*—Gummata are usually multiple, though one may be much bigger than the rest; in 86 cases of hepatic gummata collected by Dr. Allen only 11 were single. They are much commoner on the anterior surface of the liver than elsewhere, and are said, though this is not my experience, to be specially apt to occur near the falciform ligament. They are very rarely seen embedded in the substance of the organ away from the surface. They are more often met with in the right than in the left lobe. On section they have a dead white colour, and sometimes closely resemble secondary carcinomatous masses.

In well-marked cases the liver is much deformed from the contraction that gummata and their cicatrices induce, and its surface may be depressed and furrowed so as to resemble the lobulation of fœtal kidneys. A combination of gummata and cicatrices may, indeed, practically destroy a part or the whole of a lobe; usually, however, gummata and cicatrices are circumscribed, and the intervening liver tissue is healthy, thus contrasting with the diffuse pericellular cirrhosis of congenital syphilis. They often set up local chronic perihepatitis—very rarely universal chronic perihepatitis. When combined with lardaceous disease a gummatous liver is larger than natural; as a rule, it is about the normal size, and where greatly deformed may be smaller than natural.

*Signs and Symptoms.*—Gummata and cicatrices are frequently latent and give rise to no disturbance during life. The factors that determine the development of symptoms are: (1) their size and extent; (2) their position.

(1) If a gumma is large it will give rise to the signs of a tumour, and, by irritating the capsule of the liver, to perihepatitis and pain; while the morbid metabolism going on inside it may lead to the production and absorption of poisons which will lead to constitutional symptoms, such as anæmia, asthenia, and perhaps fever.

(2) A cicatrix or small gumma on the convexity of the liver need give rise to no symptoms, but if situated in the portal fissure jaundice and ascites may follow.

There is a great difference between the relative importance of a caseous gumma and an old cicatrix; for symptoms due to the pressure of an adjacent caseous gumma may be relieved, or disappear under the influence of iodide of potash, whereas it is highly improbable that an old cicatrix will be altered by such treatment.

*Onset.*—A fair proportion of the cases manifest themselves within three years of the primary infection; sometimes hepatic manifestations occur much earlier and with great rapidity. On the other hand, a long interval may occur between the infection and the appearance of any symptoms; they may be postponed for thirty or forty years, so that, as in tuberculosis, it might be said that no man should be regarded as cured of syphilis until his autopsy had been thoroughly performed.

*The clinical manifestations* of the tertiary

syphilitic lesions of the liver may be present themselves under the following aspects:—

(1) Resembling common cirrhosis and simple chronic peritonitis and perihepatitis.

(2) Presenting the features of lardaceous disease, with albuminuria, œdema, and perhaps diarrhœa.

(3) Resembling tumour of the liver or of the neighbouring parts.

(4) Suggesting hepatic abscess.

(1) If a gumma presses on the portal vein or its branches, the symptoms of portal obstruction —hæmatemesis, dilated abdominal veins, ascites, asthenia, wasting, etc.—will follow.

These are the cases that recover under iodide of potassium, and probably account for some of the reputed cures of common cirrhosis. If the gumma is large, absorption may be imperfect, and a cicatrix will be left behind which may permanently compress the portal vein and bile duct in the hilum of the liver and not yield to antisyphilitic treatment. These cases then closely resemble cirrhosis in the symptoms. Jaundice is a rare event in syphilitic disease of the liver; ascites is much more frequent.

The presence of gummata on the surface of the liver sets up local perihepatitis, and thus gives rise to discomfort, dragging, and even pain in the hepatic region which may radiate up to the right shoulder. The perihepatitis is very seldom universal; when this is the case it may account for ascites; ascites may also be due to extension of the chronic inflammation to the peritoneum, while a rare cause is narrowing and stricture of the hepatic veins by gummatous infiltration, or the contraction of cicatrices near their opening into the inferior vena cava.

*Diagnosis from Cirrhosis.* — A history of infection and manifest signs of syphilis are indications for active antisyphilitic treatment that should never be neglected. If it is palpable, the syphilitic liver will probably be felt to be irregular; and if enlarged, the increase in size is not uniform, or shared in by the left lobe as it is in large cirrhosis livers.

Enlargement of the spleen in the absence of lardaceous disease, which itself suggests syphilis, points to cirrhosis. An alcoholic history and long-continued dyspepsia are also in favour of cirrhosis.

*Diagnosis from simple Chronic Peritonitis and Perihepatitis.*—Cases of syphilitic disease of the liver in which ascites recurs will closely resemble cases of simple chronic peritonitis, of which chronic universal perihepatitis is only a part.

Chronic and recurrent ascites only occurs in a small proportion of the cases of syphilitic disease of the liver, while it is constant in cases of simple peritonitis and perihepatitis. In order, therefore, to regard a case of recurrent ascites as due to syphilitic disease of the liver, there must be undoubted evidence of syphilis in the body, and of enlargement and irregularities on the surface of the liver, such as would be produced by gummata and not by chronic perihepatitis.

Treatment by iodides, if successful in a doubtful case, would point to syphilis.

(2) When gummata in the liver are associated with widespread lardaceous disease, the albuminuria and œdema of the legs may render the aspect of the case that of lardaceous disease, and no symptoms may be found suggesting gummata in the liver.

(3) *Gummata imitating Hepatic Tumours.*—When, as they usually are, gummata are situated on the anterior surface of the liver, the irregularities they give rise to may be readily felt through the abdominal wall. The elevations of the liver substance due to the contraction of cicatrices are also easily palpable. These nodules, however, are not umbilicated as the secondary carcinomatous nodules are; but no stress can be laid on umbilication, for it may be felt over a gumma projecting from the surface of the liver.

When gummata are associated with lardaceous change in the same liver the enlargement may be very considerable, and the resemblance to carcinoma very considerable. The irregularities produced by cicatrices in a lardaceous liver have a similar resemblance to malignant disease. In such cases albuminuria points to lardaceous disease, and is therefore in favour of syphilis. Jaundice and ascites, especially together, are more likely to be met with in malignant disease; other points in favour of growth are rapid increase in the size of the liver, marked constitutional symptoms, and, of course, any signs of a growth elsewhere. In a syphilitic subject, enlargement and irregularity of the liver may be due either to gummatous disease or to new growth; for syphilis, of course, in no way protects against malignant disease. The vigorous administration of iodides and mercury should decide the question, diminution in size of the liver settling the diagnosis in favour of gumma.

Difficulty sometimes arises in deciding between gummatous infiltration of a lobe of the liver and a hydatid cyst covered over by a layer of liver substance. The general health in hydatid is unaffected unless suppuration has occurred, and the liver is smooth, whereas in syphilis other signs of the disease and irregularity of the liver should be present. In any doubtful case iodides should be given at once.

It can very rarely happen that a gumma imitates a distended gall-bladder, but this has occurred.

(4) Occasionally an irregular or hectic temperature accompanies gummatous change in the liver, and might suggest ordinary suppuration, malaria, tuberculosis, or even typhoid fever: it usually yields to iodides.

As a result of secondary infection a gumma

may soften down, and may present as a fluctuating swelling either anteriorly or by perforating through the intercostal spaces laterally or posteriorly.

*Prognosis.*—When adequately treated with iodides the prognosis of syphilitic disease of the liver is much better than in most of the conditions that have been referred to as sometimes resembling it, viz., malignant disease, cirrhosis, perihepatitis, and chronic peritonitis.

Gummata undergo absorption, and the bad effects due to their mechanical pressure are relieved; but cicatrices are left behind, and if they compress the portal vein or bile ducts the symptoms will remain practically unaffected. Antisyphilitic treatment does not affect them, so it is not fair to assume that the failure of iodides proves the condition to be non-syphilitic.

The prognosis of hepatic enlargement or tumour due to syphilis is thus much brighter than that of ascites or jaundice thought to depend on some other factor.

*Treatment.*—Iodides should be given in combination with mercury. Iodide of potassium should be combined with iodide of sodium, and with an ammonium salt such as spiritus ammoniæ aromaticus, so as to prevent the depressing effect of the potash. To begin with, a dose containing 10 grains of the combined iodides should be given three times daily, and should be increased so that in a fortnight's time 30 grains are taken for a dose. The medicine should be taken shortly before meals; if taken on a full stomach, dyspepsia may result from liberation of iodine by the action of the hydrochloric acid of the gastric juice.

Mercury may be given in the form of hydrargyri c. creta combined with compound ipecacuanha powder to prevent diarrhœa.

In cases where gummata develop rapidly and early after infection, the subcutaneous or, better, intramuscular injection of soluble mercurial salts, such as the benzoate, should be employed.

*The Surgical Treatment of Gummata.* — In cases where a softened gumma of the liver has begun to work its way out through the abdominal wall, incision and removal of some of the caseous debris has had a good result in diminishing septic absorption. In other cases in which exploratory laparotomy revealed a hepatic gumma partial removal has seemed to accelerate the subsequent action of iodides.

It is not likely to be employed except in the event of a gumma simulating an abscess, or where the diagnosis has been at fault.

### Hereditary Syphilis

The changes in the liver that depend on hereditary syphilis may conveniently be considered under three heads:—

(i.) The lesions met with in the livers of babies, manifesting the other ordinary evidences of hereditary syphilis.

(ii.) Tardive or delayed hereditary syphilis.

(iii.) Multilobular cirrhosis supervening in children, the subjects of hereditary syphilitic infection.

The first of these categories is the most important, and refers to the lesions ordinarily known as the liver of congenital syphilis.

*The Ordinary Hepatic Manifestations of Congenital Syphilis.*—The liver is found to be affected in a very large proportion of the fatal cases of hereditary syphilis; this contrasts with acquired syphilis, where the liver frequently escapes. The frequency with which the liver is affected in hereditary syphilis is an argument in favour of the view that antenatal infection of the fœtus is maternal, and that the infection passes through the placenta and umbilical vein, thus damaging the liver on its way to the fœtus. If the ovum was primarily infected by a syphilised spermatozoon the ovum would probably not survive; and further, if it did, the syphilitic toxin would reach the liver, as it does in acquired infection by the hepatic artery, and should therefore only be affected in the same proportion as in acquired syphilis.

*Morbid Anatomy.*—The appearances vary very considerably. In slight cases the organ may show little change except some pallor. In other cases its colour may be brown, yellowish, or violet, and may in advanced cases look like flint. To livers of this type Gubler applied the term "*foie silex.*"

The organ is enlarged and heavier than normally, weighing $\frac{1}{16}$th to $\frac{1}{12}$th instead of $\frac{1}{25}$th of the normal body-weight at birth. Evidence of past perihepatitis in adhesions to the diaphragm are sometimes seen, but usually the surface is smooth, though there may be irregularities and projections due to the changes being more advanced in certain areas of the liver.

On section the liver tissue is firm and tough, and appears marbled or mottled from the presence of pale, whitish-yellow areas, where there is increased fibrosis, with congestion around them. The lobular markings are obscured or lost, and the appearances may suggest lardaceous disease or diffuse sarcoma.

As a rule the changes are diffuse, and thus contrast with the circumscribed lesions of tertiary acquired syphilis; but exceptionally the change may be so localised as to imitate a tumour.

On carefully looking at the cut section, small millet-seed nodules resembling tubercules are often detected. These are minute syphilomata, composed of granulation tissue, and have been spoken of as miliary gummata, though the term gumma is better reserved for the further stage where central necrosis and caseation have supervened.

In rare instances well-marked gummata, comparable to those met with in acquired syphilis, are found in the liver of infants, or even in still-born fœtuses.

Another and a rare appearance is a localised fibrosis of part of a lobe; this may indeed imitate a tumour, and cases described as fibroma of the liver are probably of this nature.

*Histologically.*—The essential change is that seen in the secondary stage of syphilis, viz., a diffuse small cell infiltration. The individual liver-cells are separated from each other by young connective tissue, the result of proliferation (*a*) of the pre-existing connective tissue cells of the organ; (*b*) of the endothelium of the capillaries and lymphatics in the lobule of the liver. Kupffer's star-like cells, which are intimately connected with the endothelial lining of the vessel walls, share in this change According to the duration and activity of the process there may be small round cells, spindle cells, or fairly well-formed fibrous tissue. This diffuse fibrosis is variously spoken of as a monocellular, unicellular, intercellular, or pericellular cirrhosis. When the process is seen in an early stage the small round cells may suggest sarcoma; when a number of these cells are collected together a syphilitic granuloma or miliary gumma is formed.

The liver-cells are compressed, shrunken, granular, and sometimes undergo necrosis and disappear. They do not undergo fatty change. When compressed, they may appear in rows like the so-called new bile ducts.

The fibrous tissue of the portal canals is increased in amount. The hepatic artery is normal, and although in exceptional instances changes in the branches of the portal vein and bile ducts have been described, they are, practically speaking, always healthy.

In different stages of the disease the appearances vary; thus pericellular cirrhosis alone, combined with miliary syphilomata, with fibrous tissue, and even with well-defined gummata, may be found.

The diffuse monocellular cirrhosis is like the secondary lesions elsewhere in the body, a curable condition if treated by mercury; but it may pass into the tertiary manifestations and lead to gummata, cicatrices, diffuse fibrosis, and lardaceous disease.

*Clinical Features.* — As a rule, symptoms pointing to the liver are entirely absent; the ordinary signs of congenital syphilis are found, with enlargement of the liver and spleen.

The liver is smooth, firm, and tender; in exceptional instances part of it may be so prominent as to feel like a tumour.

Jaundice is rare; it is like the jaundice occasionally seen in the secondary stage of acquired syphilis, and may be referred to one of the following causes: enlarged glands in the portal fissure exerting pressure on the ducts, inflammation of the small ducts, and *intime* changes in the liver-cells and minute bile ducts. In some of the cases the jaundice is terminal and due to secondary infections falling on the liver, a form of icterus gravis.

Ascites is rarely seen except in still-born children. It may be due to peritonitis, which, as shown by perihepatic adhesions, may occur in this disease.

The hepatic enlargement corresponds to the other manifestations of the disease, and hence may be taken as an index of the severity of the infection. In some cases the liver may reach down as far as the crest of the ilium. In connection with this it should be borne in mind that the liver is not only relatively larger in infants than in adults, but that it normally projects farther down below the ribs, so that slight apparent enlargement of the liver is not of any importance.

*Treatment* is that of congenital syphilis by mercury either by inunction or by the mouth. Mercurial ointment should be rubbed into the skin of the axillæ, groins, etc., with flannel; a different area of skin should be employed from day to day. The method of inunction is more rapid in its action and less likely to lead to salivation than the administration of mercury by the mouth. It should be practised daily for three months; after that it should be dropped for a week at a time at first, and then for two weeks. In the second year of treatment inunction should be practised for one month out of every three, and small doses of iodide of potassium given; this should be continued in the third year, the iodide being increased; in the fourth year the mercurial treatment should be stopped, but the iodide should be continued. By these means the development of tertiary manifestations should be presented.

When mercury is given by the mouth it is usually administered in the form of hydrargyr. c. creta; to an infant under two months old ½ grain should be given twice a day, the dose being increased to 1 grain after a time.

*Hepatic Lesions of Delayed or Tardive Hereditary Syphilis.* — Here hepatic manifestations develop very much later than in the last category, often coming on about puberty or even in adult life. The lesions are tertiary in character, and resemble those seen in the acquired form of the disease. What has happened is that hepatic lesions characteristic of hereditary syphilis (pericellular cirrhosis) have persisted, and instead of being cured by treatment have passed on into the tertiary stage.

Since the lesions are the same as those of the tertiary stage of acquired syphilis, there must be some other evidence of the hereditary form, such as interstitial keratitis or Hutchinson's teeth, in order to be certain that the case is one of delayed hereditary syphilis, otherwise the disease might have been acquired in early life —for example, from a wet nurse.

The liver may be greatly deformed from contracting cicatrices, and may be divided up into numerous lobules; in fact, some of the

recorded examples of abnormal lobulation of the liver are of this nature. In some cases there may be extensive lardaceous disease, giving rise to albuminuria, diarrhœa, and enlargement of the spleen. The pressure of a gumma or contraction of its cicatrix may involve the portal vein, or, more rarely, the bile duct, giving rise to ascites and jaundice.

From cirrhosis of the liver and new growths this condition may be distinguished by the presence of syphilitic lesions in the skin, bones, and sense organs, eye, nose, ear; and by the effect of antisyphilitic remedies.

From acquired syphilis it is distinguished by the presence of stigmata of the congenital form, such as nebulæ on the cornea from former interstitial keratitis, or Hutchinson's teeth.

The clinical characters and treatment are the same as those of the acquired disease.

*Multilobular Cirrhosis developing in the Subjects of Hereditary Syphilis.*—The diffuse pericellular cirrhosis of infants the subjects of congenital syphilis is, like the lesions of the secondary stage of the acquired disease, a curable lesion. Microscopic examination of the livers of children formerly affected with well-marked hereditary syphilis may show no disease. On the other hand, every now and again the liver of a child who bears undoubted stigmata of congenital syphilis in the body is found to show ordinary cirrhosis. The arrangement of the two lesions is so dissimilar that pericellular cirrhosis cannot be thought to be transformed into multilobular cirrhosis; it would rather tend to diffuse fibrosis or gummatous change. It seems probable that the pericellular cirrhosis undergoes absorption, but that some vulnerability and diminished resistance of the liver is left behind. If causes then arise that tend to produce ordinary cirrhosis, this change will be readily produced. In other words, the multilobular cirrhosis is a parasyphilitic lesion, and is comparable to general paralysis of the insane, in that though not syphilitic it is favoured by syphilisation of the soil.

In some instances there is very diffuse cirrhosis, suggesting that multilobular cirrhosis has supervened before the pericellular cirrhosis had receded, and that some of the fibrous infiltration was due to organisation of the pericellular formation.

Occasionally in multilobular cirrhosis occurring early in life in the subjects of congenital syphilis, there is early lardaceous change in the organ.

What proportion of small cirrhotic livers in children have a substratum of syphilitic taint, it is difficult to say. Statistics of reported cases of cirrhosis in children make it clear that direct evidence of syphilis is often not forthcoming.

The clinical features of these cases of cirrhosis is much the same as those of common (small liver) cirrhosis, viz., those of portal obstruction, ascites, wasting, etc.

It may be very difficult to differentiate between these cases of cirrhosis in individuals with other manifest signs of congenital syphilis on the one hand, and cases of tardive hereditary syphilis with hepatic lesions and ascites on the other hand. In the latter there may be excessive lardaceous disease, as shown by albuminuria. Iodide of potassium and mercury should be tried, and improvement will point to hepatic gummata and cicatrices due to tardive hereditary syphilis, and must then be pushed.

*The prognosis* of these cases is very bad.

*The treatment* is that of ordinary cirrhosis, viz., milk diet, no alcohol or irritating food. The prevention of constipation and auto-intoxication by intestinal antiseptics such as calomel, salicylate of soda, β-naphthol, is important.

Iodide of potassium should be given constantly, as is often done in common cirrhosis of adults, to prevent if possible any further progress in the disease. But as the lesion is parasyphilitic rather than syphilitic, iodide of potassium can hardly be expected to remove the fibrosis.

### Lymphadenoma

In generalised lymphadenoma the liver not infrequently contains nodules of growth. As a rule they are small, and rarely give rise to much enlargement of the organ.

In exceptional cases the liver is considerably enlarged, and if the superficial lymphatic glands available for clinical examination are little affected, the clinical aspect may suggest hepatic abscess as in a case under my care, or even malignant disease of the liver (Suchard et Teissier). For the morbid anatomy and other details *vide* article on "Lymphatic System, Physiology and Pathology."

### Actinomycosis of the Liver

When actinomycosis occurs in the liver it must always be conveyed from some absorbent surface, such as the intestines, or spread to the liver by continuity. In 30 cases of hepatic actinomycosis collected by Aribaud, the growth was derived from the alimentary tract in 20, spreading by direct extension in 8 cases and by metastasis in 12. The liver may be affected by extension from the base of the lung, the infection spreading through the diaphragm, or possibly the primary lesion may be in the skin of the abdominal wall.

Sometimes the primary source of inlet is not found; thus Taylor, Shattock, and Boari have described primary actinomycosis of the liver.

*Morbid Anatomy.* — The liver is enlarged. The actinomycotic abscess has a characteristic honeycombed aspect, and has been compared

to a sponge soaked in pus. The alveolar appearance is due to the coalescence of a number of small abscesses. The suppurative process spreads by continuity, and is accordingly more or less localised, but sometimes small abscesses are seen away from the main collection. The abscesses vary in size from a pin's head to that of a walnut; the pus contains the characteristic granules composed of the ray fungus—or actinomyces colonies—and numerous pyogenetic micro-organisms. Around the areas of suppuration there is fibrosis, with pigmentation of the walls of the small abscesses. The remainder of the liver may be congested and fatty. Microscopically there is intercellular cirrhosis in the immediate neighbourhood, with atrophy of the liver-cells.

For the nature and characters of the fungus the reader is referred to Professor Delépine's article, vol. i. p. 53.

There is a great tendency to get inflammation of the capsule of the liver and adhesions to adjacent organs. If the actinomycotic lesion is situated anteriorly it readily extends to the abdominal wall, and may lead to an abscess. This may be the first evidence of disease, so that caution is required in assuming that the hepatic lesion is secondary to an abscess of the abdominal wall.

The actinomycotic abscess may spread through the diaphragm to the pleura or into the lung, and may first appear as an empyema of chronic character and obscure origin.

In rare cases (Israel, Kanthack) actinomycosis may be pyæmic, and spread by the blood-vessels. In Kanthack's case it was not clear whether the abscess originated in the right lobe of the liver or at the base of the right lung; from this it had spread by continuity into the right suprarenal body, and then given rise to secondary pyæmic abscesses over the body.

In Boari's case there were secondary pyæmic abscesses due to pyogenetic cocci, and not containing actinomycosis.

*Clinical Aspect.*—The first evidence may be that of an empyema, of an abscess in the abdominal wall, or, when the portion of the liver near the kidney is involved, of a perinephritic abscess.

The liver may be enlarged, and with a slight degree of fever and some pain over the liver the suspicion of an hepatic abscess may arise. Jaundice is extremely rare.

*Diagnosis* depends on finding the fungus in the pus, either from the liver or from a discharging abscess elsewhere. Before this has been done the condition is hardly likely to be thought of, and recorded cases show that the disease has been regarded as empyema, phthisis, sarcoma of the kidney (Leith), perinephritic abscess, hepatic abscess, suppurating hydatid, or gumma of the liver.

Latimer and Welch describe a case of actinomycosis of the liver combined with myelogenous leukæmia.

*The prognosis* depends on the disease being recognised and vigorously treated with iodide of potassium, and on freedom from secondary infection with pyogenetic micro-organisms.

*Treatment.*—The effect of iodide of potassium, introduced by Thomassen, in actinomycosis is extremely marked, and does fully as much good as it does in tertiary syphilis. It should be given in large doses—as much as a drachm daily.

Locally iodoform may be employed, and antiseptics to minimise septic infection.

### Malignant Disease of the Liver

Malignant disease may be primary in the liver, but more commonly new growth in the liver is secondary to a growth elsewhere. It will be convenient to consider the subject under these two heads:—

Primary Malignant Disease of the Liver—
Etiology . . . . . 478
Morbid Anatomy . . . . 478
*Carcinoma* . . . . . 479
*Sarcoma* . . . . . 479
Physical Signs and Symptoms . 480
Diagnosis . . . . . 480
Treatment . . . . . 481

### Primary Malignant Disease of the Liver

Malignant disease, when it occurs primarily in the liver, most frequently starts in the gall-bladder. This subject has already been described (vol. iii. p. 386), and here primary disease of the liver itself will be considered.

*Frequency.*—Primary malignant disease of the liver is a rather rare disease, and although clinically it is common to meet with cases where the manifestations are those of malignant disease in the liver without any definite evidence of a primary growth elsewhere, the majority will be found to be secondary to a latent growth elsewhere.

The ratio of the incidence of primary to secondary malignant disease of the liver has been stated to be as 1 to 20.

*Sex.*—It is commoner in men than in women, thus contrasting with primary cancer of the gall-bladder, which is four times commoner in women.

*Age.*—It is met with in or after middle life, and seldom occurs under the age of forty years. It may, however, occur in quite early life. I have notes of twenty-nine cases of primary sarcoma in children under ten years of age, and congenital examples have been described.

*Nature.*—Primary carcinoma is much more frequent than primary sarcoma of the liver. Very considerable variation exists in the forms of carcinoma and sarcoma met with in the liver.

Carcinoma may be—

(1) Massive, a large growth expanding the liver around it, the surface of which is smooth, though secondary nodules may arise away from the main mass. This form of growth may for a time imitate an abscess or hydatid. Ascites, jaundice, and perihepatitis are rare.

It is usually a rapidly growing spheroidal-celled carcinoma derived from the liver-cells, or from the cubical epithelium of the smaller bile ducts; exceptionally it is a columnar-celled carcinoma starting from one of the larger intra-hepatic ducts. In a few instances giant multinuclear cells are found. A carcinoma starting in the gall-bladder and completely replacing it may at first sight be mistaken for a primary massive carcinoma of the liver.

(2) Infiltrating Form.—The greater part or even the whole of the liver may be uniformly saturated with carcinoma; sometimes the growth is slow, and a great quantity of fibrous tissue is formed, with the result that the organ is hard, like a small atrophied liver, and not necessarily increased in size. In other cases the liver is widely infiltrated with active growth, and is much increased in size and weight. Histologically, this form is generally spheroidal-celled carcinoma.

(3) Nodular.—The appearance of the liver is like that seen in secondary carcinoma, the chief difference being that there is no primary growth elsewhere in the body. The tumours grow rapidly, are prone to degenerate, and sometimes become hæmorrhagic. Possibly some of these cases are, like carcinoma of the inguinal lymphatic glands, in sweeps without primary carcinoma of the scrotum, examples of what has been termed secondary growths without any manifest primary focus. It is compatible with the parasitic theory of cancer to suppose that the hypothetical parasite might, once having gained an entrance through the alimentary canal, set up multiple lesions in the liver.

Possibly some cases of primary nodular carcinoma are due to growths arising in accessory suprarenal bodies that have become embedded in the liver.

It may be that one of the multiple nodules of growth was primary, and that the others are secondary, but have grown more rapidly and so rivalled it in size.

These growths are usually spheroidal-celled, but may be columnar-celled, or show a transition from the latter to the spheroidal type.

Just as anatomically, so clinically this form resembles secondary carcinoma of liver, in the frequency with which perihepatitis, pain, jaundice, and ascites are met with.

(4) The condition termed *carcinoma with cirrhosis* somewhat resembles the nodular form on the one hand, and cirrhosis with adenoma on the other. It has been chiefly described in France; Hanot and Gilbert say that it is the form met with in more than one-third of the total number of the cases of primary carcinoma of the liver. There are multiple growths associated with cirrhosis of the liver; it is supposed that the compensatory hyperplasia of the liver-cells that gives rise to multiple adenoma passes on into a malignant activity, and that carcinoma develops. This form is frequently associated with thrombosis of the portal and hepatic veins, the growth being said to invade the veins. Secondary growths in the portal lymphatic glands or elsewhere are rare. When they do occur, no doubt can exist about the nature of the change in the liver; but in their absence it seems to me probable that many of the cases described as carcinoma with cirrhosis are merely nodular cirrhosis, or cirrhosis with multiple adenoma (*vide* p. 467).

Histologically the carcinomatous structure is described as being trabecular, and resembling the pseudo-bile canaliculi seen in so many conditions where compensatory hypertrophy of the liver-cells is required.

It is noteworthy that the symptoms of these cases correspond with those of cirrhosis.

*Secondary growths* in primary carcinoma of the liver occur in the liver itself, in the glands in the portal fissure, and sometimes in the lungs, but the course of the primary disease is so rapid that secondary metastases have not time to become of importance.

Gall-stones are rarely found in primary carcinoma of the liver itself. This contrasts with primary carcinoma of the gall-bladder, where the association is present in 95 per cent of the cases.

*Primary sarcoma* of the liver is much rarer than primary carcinoma. It may occur, as already mentioned, in early life, but a caution should be thrown out not to regard as sarcoma the lesions of congenital syphilis.

The following forms of primary sarcoma may be mentioned:—

(1) A massive tumour which may soften down and imitate an abscess or a cyst.

(2) A diffuse infiltrating form, as seen in cases occurring in early life, and in the rare cases, of which about ten are on record, of primary melanotic sarcoma of the liver.

(3) A multiple form without any primary growth.

The growth may start from the general connective tissue of Glisson's capsule, from the perivascular sheaths, from Kupffer's star cells, or from the endothelium of the vessels.

The histological characters of the primary sarcomata met with include small round-celled, spindle-celled, mixed and irregular-celled, angio-sarcoma, and melanotic growths. Difficulty not infrequently arises in deciding whether a primary hepatic tumour should be labelled carcinoma or sarcoma; this depends on the

tendency of the sarcomatous growth to spread along the capillaries, and so to assume an alveolar appearance.

Physical Signs of Primary Malignant Disease.—The liver is nearly always enlarged; it may be smooth or nodular, but in either case it increases progressively and often rapidly in size. The enlarged liver may displace the diaphragm upwards, and give rise to dulness at the base of the right lung; sometimes it is further complicated by pleural effusion.

Ascites and jaundice are not so frequent as in secondary malignant disease of the liver. Ascites is said to be present in about half the cases, and not to be found in the massive form of primary carcinoma. Jaundice very rarely shows itself in the massive form; when present, it is not of the dark green or black colour seen in some instances of secondary malignant disease.

The patient's facial aspect is usually that of grave disease, and wasting occurs, but the progress of the disease is so acute as compared with that of secondary malignant disease that emaciation has barely time to become marked. The tumour growth may be so rapid that the body-weight actually increases in spite of general loss of flesh. Œdema of the feet may develop in the late stages. The temperature may be raised, and bacterial infection of the liver or the bile ducts may take place, and thus exceptionally rigors may be met with.

There may be albuminuria due to toxic substances in the circulation reaching the kidneys and damaging the delicate epithelium covering the glomerular tufts. When there is jaundice, bile pigment will be found in the urine.

The chief symptoms are loss of strength, loss of appetite, gastric disturbance, and pain over the liver. Vomiting may be reflex in origin. Pain and tenderness depend on stretching of the capsule, or on local perihepatitis set up by the growth involving the capsule.

In the late period of the disease hepatic insufficiency may be developed; the patient passes into a drowsy, semi-comatose state, and hæmorrhages may appear.

The course of the disease is more rapid than that of secondary malignant disease, and few cases last more than four months; sometimes the disease may justify the adjective acute, and its duration may be counted in weeks rather than months.

*Diagnosis.*—Under this head the diagnosis of malignant disease in the liver substance, whether primary or secondary from other conditions, will first be considered, and then the distinction between primary and secondary and malignant disease will be referred to.

In a few instances of primary malignant disease of the liver the existence of hepatic disease is not even suspected; but this is exceptional, and enlargement will usually be detected.

In the massive form, where the surface is smooth, it must be distinguished from lardaceous disease; in the latter, attention must be directed to the history of past suppuration, or of syphilis, and to signs of lardaceous change elsewhere. In the enlargement due to a deep-seated hydatid the patient's general health and strength remain good, while in carcinoma his powers rapidly fail.

Multilocular or alveolar hydatid has often been mistaken for malignant disease, both clinically and even when found after death. It has not been described as occurring in England, and it is rare anywhere; in most cases of the disease the spleen is enlarged, thus differing from malignant disease.

The large and tender liver of the terminal stage of mitral disease has been known to resemble malignant disease, but the history of the case and the signs of cardiac and circulatory disturbance should prevent any mistake.

In rare instances the rapid growth of the tumour may give rise to fluctuation, while the raised temperature that is not infrequently seen may further increase the resemblance to various forms of intrahepatic suppuration, such as abscess, pylephlebitis, cholangitis, etc. Sometimes an exploratory incision is the only means of deciding the question. It may indeed happen that secondary infection either of the growth or of the ducts occurs, and that suppuration is thus superimposed on new growth.

From the large liver of hypertrophic biliary cirrhosis primary malignant disease differs in its more rapid growth, in the absence of splenic enlargement, and in the character of the jaundice. In malignant disease it is either absent or, if present, obstructive, so that no bile passes into the bowel. In biliary cirrhosis jaundice is constantly present, but bile colours the fæces. Hypertrophic biliary cirrhosis is met with much earlier in life than malignant disease.

In the late stages of ordinary or portal cirrhosis, if there be ascites and jaundice, the resemblance to cases of multiple nodular malignant disease of the liver is considerable; after paracentesis, the condition of the liver, comparatively small in cirrhosis, large or extremely nodular in new growth, will generally render a definite decision possible.

A large gumma of the liver may be accompanied by considerable cachexia, but should be recognised by the signs of syphilis elsewhere, and by the effect of vigorous treatment with iodides.

Occasionally fæcal accumulation in the transverse colon may imitate malignant disease; here the tumours may vary in position from time to time, can be indented by pressure, are capable of removal by purgatives or abdominal massage, and when a careful examination is made, if need be under an anæsthetic, other

masses can be made out in the course of the colon.

A renal tumour may appear to be in connection with the liver, but a bimanual examination should be sufficient to show that it bulges into the loin, while the presence of bowel in front of the tumour points to its renal origin.

Inflammatory thickening around the gall-bladder is often palpable as a hard mass, and thus may give rise to physical signs resembling carcinoma. The history of gall-stones, and the fact that the patient's general state is not so grave as in carcinoma, are important points to bear in mind.

*The diagnosis of primary from secondary malignant disease* of the liver is very difficult, inasmuch as, in perhaps as many as 50 per cent of those cases of secondary malignant disease of the liver that give rise to symptoms, the existence of a primary growth elsewhere cannot be satisfactorily determined during life. When there is evidence of a growth in situations such as the stomach, colon, or pancreas, the malignant disease in the liver is evidently secondary. But when the only clinical evidence is of growth in the liver, it is very difficult to come to a satisfactory conclusion as to whether it is primary or secondary. Multiple growths, and the association of jaundice and ascites, are rather in favour of secondary malignant disease, while rapid growth of the liver without marked emaciation points to a primary growth. Deep jaundice is in favour of secondary growths.

Malignant disease of the gall-bladder is usually preceded by biliary colic, and shows itself as a tumour in the region of the gall-bladder.

*The prognosis* is, of course, absolutely hopeless, except in those very rare instances where the tumour has been completely removed by the surgeon.

*Treatment.*—In a few exceptional instances a primary malignant tumour of the liver has been removed. In most cases, however, this is impracticable from the extent of the tumour and the frequency with which secondary growths are found in other parts of the liver.

Apart from this the treatment is merely palliative, and consists in relieving symptoms as they arise. Vomiting should be met by ice, bismuth, dilute hydrocyanic acid, etc.; dyspepsia by carminatives, ascites by paracentesis, and pain by the hypodermic injection of morphia.

A milk diet is most suitable, tea and coffee may be given, and stimulants are usually necessary.

SECONDARY MALIGNANT DISEASE OF THE LIVER

ETIOLOGY . . . . . 481
MORBID ANATOMY . . . . . 481
*Site of Primary Growth* . . . 481
*Carcinoma* . . . . . 481
*Sarcoma* . . . . . 482
CLINICAL FEATURES AND COURSE . . 482
DIAGNOSIS (*vide* Diagnosis of "Primary Malignant Disease") . . . . . 483
TREATMENT . . . . . 483

*Frequency.*—The liver is the organ most frequently affected by secondary malignant disease. Thus it is involved in half the total cases of malignant disease, and in 3 per cent of all bodies examined after death (Hale White). In a large number of the cases collected for statistical purposes secondary growths in the liver have given rise to no sign during life. It appears that malignant disease is becoming more frequent, especially in the abdomen.

*Sex.*—Secondary malignant disease is rather commoner in women, from the frequency of malignant disease in the breast and internal organs of generation, than in men.

*Age.*—It usually occurs after forty years of age.

*Site of Primary Growth.*—The primary growth is latent in a large number, perhaps in half of the cases presenting evidence of secondary malignant disease of the liver during life. The stomach and colon are most frequently sites of the primary growth, but fatal cases of carcinoma of the breast are very frequently found to have secondary growths in the liver. Other situations in which the primary growth may occur are the pancreas, gall-bladder, œsophagus, uterus, kidney, and uveal tract.

Secondary growths are usually carcinomatous; sarcoma is comparatively infrequent. This is probably due to the fact that it only rarely occurs primarily within the area drained by the portal vein. Secondary melanotic sarcoma of the liver is a striking but rather uncommon form of growth; it is much more marked after melanotic sarcoma of the uveal tract than of the skin.

*Morbid Anatomy.*—Secondary growths are usually multiple and nodular, but sometimes, *e.g.* when secondary to carcinoma of the mamma or to sarcoma of the uveal tract, there may be diffuse infiltration of the organ. The two forms may be found in the different parts of the same liver. The growths are frequently found on the surface of the liver, and are rarely present inside when absent externally.

Carcinomatous growths are white, yellow, bile-stained, or streaked with blood, and when of some standing become cupped or umbilicated. This depends partly on cicatricial contraction taking place in the older portions, and in part on the more exuberant growth of the peripheral and more recent portions.

The nodules on the surface of the liver may set up perihepatitis and adhesions to adjacent parts, while exceptionally the growth may grow directly into the diaphragm or abdominal parietes.

The growths may soften down, and occasion-

ally may suppurate as the result of infection. In secondary squamous-celled carcinoma, cysts containing clear fluid have been seen.

All three forms of carcinoma — spheroidal, columnar, squamous-celled—are met with; and not infrequently, when the secondary growths are increasing rapidly, there is a transition from the columnar-celled to the spheroidal-celled type.

Secondary colloid carcinoma may occur; and sometimes, like other forms of carcinoma, colloid carcinoma may spread by continuity into the portal fissure.

Secondary carcinoma and sarcoma both begin inside the capillaries of the liver, and hence a sarcoma often has an alveolar arrangement.

Pressure on the bile ducts and branches of the hepatic veins gives rise to local bile-staining and chronic venous congestion of the liver substance.

Carcinoma may be spread directly into the liver substance, especially from primary carcinoma of the gall-bladder. Carcinoma of the stomach may grow directly into the liver, or pass up the lesser omentum to the portal fissure, and incidentally compress the bile duct and portal vein.

Sarcomatous growths are very rarely umbilicated; they are prone to be more hæmorrhagic than carcinomatous nodules, and, like them, may soften down and form pseudo-cysts. As the result of hæmorrhage taking place into the growths the size of the liver may suddenly increase. Rupture of a hæmorrhagic nodule of growth may give rise to severe collapse from hæmorrhage into the peritoneum.

*Clinical Features.*—The liver is enlarged and progressively increases in size, the right lobe being more affected than the left. Its surface is irregular and nodular, and the projections may, if the abdominal wall be thin, be felt to be cupped in the centre; this is a point of importance in distinguishing it from the hob-nailed liver of cirrhosis. Outlying nodules of growth may be felt at the umbilicus or along the line of the falciform ligament. The liver may be both painful and tender, from stretching of its capsule and local perihepatitis, which may reveal itself to the stethoscope by a friction sound. The pain may spread from the right hypochondrium to the back, and be felt in the loins.

The spleen is not enlarged, hæmatemesis does not occur, and enlargement of the abdominal veins, if present, is due to obstruction to the inferior vena cava, and is not seen chiefly around the umbilicus, as it would be in portal vein obstruction.

The patient is emaciated, more so than in primary malignant disease of the organ, both because he is suffering from new growth in at least two situations, and because the course of secondary malignant disease of the liver is more protracted. The patient progressively loses strength, and gradually passes into a condition of cachexia. The cachexia may be accompanied by a certain amount of fever.

Gastric disturbance, nausea, vomiting, and loss of appetite, with marked distaste for meat, are commonly seen. The bowels are usually confined; very occasionally there is diarrhœa.

Jaundice and ascites occur in about half the cases, and may be met with together. The jaundice may be catarrhal, but is often due to gross obstruction of the ducts in the portal fissure, and is then progressive, and becomes of a dark green colour. Bile disappears from the fæces and is present in the urine. Pruritus may be troublesome; and from the development of cholæmia, hæmorrhages into the skin, and bleeding from the nose, gums, and mucous surfaces may result.

The jaundice, which does not last sufficiently long to allow of the development of xanthelasma, is more likely to occur when the primary growth is near the liver, as in the gall-bladder or stomach, whence a direct continuity of the growth may spread to the larger ducts.

The pressure of growth in the portal fissure on the ducts may extend to the portal vein, and give rise to ascites. In some cases the portal vein itself is not involved, the ascites is due either to chronic peritoneal oedema or malignant infection of the peritoneum, or to widespread infiltration of the liver with new growth obstructing the branches of the portal vein.

The ascitic fluid may be clear, bile-stained, or more rarely chyliform or hæmorrhagic. In melanotic sarcoma it has in rare instances been found to be of a dark colour, from the presence of the pigment melanin.

The urine may be lithatic and contain indican; in secondary melanotic sarcoma of the liver the urine sometimes darkens on standing, from the presence of melanin. The pigment is usually passed in a colourless form—melanogen—and when oxidised darkens. This can be rapidly demonstrated by adding nitric acid, ferric chloride, or bichromate of potash. Occasionally the urine is already dark when voided from the bladder. Urine containing indican darkens with nitric acid, but not with perchloride of iron; the latter reaction is useful in distinguishing melanuria from indicanuria.

Albuminuria and glycosuria are very infrequent in secondary malignant disease of the liver.

*Termination.*—Unless life is cut short by some complication, death occurs from gradually increasing weakness passing into coma, which may be extreme when the patient is jaundiced or suffering from cholæmia.

*Duration.*—After the liver is known to be involved, life is seldom prolonged for more than six months; sometimes the course of the disease

is more rapid. Much depends on the position and nature of the primary growth. If it be latent, have been removed, or, as in colotomy, be prevented from setting up obstruction, life may be carried on for a year, or even more. Sometimes the patient holds his own for a while and then rapidly goes down hill.

*The prognosis* is, of course, quite hopeless. Operative interference cannot be expected to do any good since the growths are multiple.

*The diagnosis* has already been discussed under the heading of Primary Malignant Disease.

*The treatment*—symptomatic and purely palliative—is in the main the same as that of primary malignant disease. For pruritus due to marked jaundice chloride of calcium may be given in full doses for a day or two and then stopped. If this fails, pilocarpine $\frac{1}{6}$-$\frac{1}{4}$ grain or morphia may be given hypodermically. Alkaline baths or sponging the skin with carbolic lotion 1 in 40 sometimes give relief.

### Icterus Gravis

Icterus Gravis—
*Nature* . . . . . 483
*Relation to Acute Atrophy* . . 483
Acute Yellow Atrophy—
*Incidence* . . . . . 483
*Etiology* . . . . . 483
*Relation to Phosphorus Poisoning* . 484
Morbid Anatomy . . . . . 484
Symptoms and Signs . . . . 486
Diagnosis . . . . . 486
Prognosis . . . . . 486
Treatment . . . . . 487

*Nature.*—Icterus gravis, or malignant jaundice, is a term somewhat loosely used for cases where there is extensive degeneration of the liver-cells combined with toxæmic jaundice, and a tendency to a fatal termination. It thus includes a number of different conditions, such as the most severe cases of febrile jaundice or Weil's disease, acute yellow atrophy of the liver, phosphorus and other forms of mineral poisoning, and other cases where an acute toxæmic or infective condition of the body falls on the liver and gives rise to widespread acute degenerative and necrotic changes in the liver-cells; for example, in yellow fever and in streptococcal and staphylococcal hæmic infections. The term icterus gravis may also appropriately be applied to cases where acute degenerative changes are superimposed on some pre-existing disease of the liver, such as cirrhosis or nutmeg liver.

Icterus gravis should therefore be regarded, not as a specific disease, but as a group of symptoms due to the rapid development of hepatic insufficiency, eventually becoming absolute, which may be due to many different causes.

Icterus gravis may be divided into—

(*a*) Those cases where the liver was previously healthy, *e.g.* in phosphorus poisoning, acute yellow atrophy, or yellow fever.

(*b*) Those cases where it supervenes as a terminal lesion on pre-existing hepatic disease, *e.g.* in cirrhosis or chronic venous engorgement.

*Relation to Acute Atrophy.*—Acute yellow atrophy is a special form of icterus gravis, and may be regarded as a typical variety, since it is uncomplicated by the presence of any other disease. The terms icterus gravis and acute yellow atrophy are not absolutely synonymous, for all cases of icterus gravis do not show the naked-eye appearances of acute yellow atrophy of the liver, though the essential change—acute degenerative changes in the liver-cells—is much the same in both. Under the microscope the appearances are so closely allied that, from a pathological point of view, they may be said to pass into each other.

Generally speaking, the liver is somewhat enlarged in icterus gravis, and the degenerative changes are not so markedly necrotic as in acute yellow atrophy.

Since some of the various conditions, such as acute yellow atrophy, phosphorus poisoning, and Weil's disease, that are or may be included under the generic term icterus gravis, will be separately described, the clinical features of icterus gravis do not require any further description than that found under the heading of Acute Yellow Atrophy.

### Acute Yellow Atrophy

Acute Atrophy of the Liver.—*Definition.*—An acute degeneration of the liver-cells with diminution in size of the liver, jaundice, hæmorrhages, nervous symptoms, and usually a fatal termination.

*Incidence.*—That this is a rare disease is shown by the fact that Osler has never seen a case; curiously enough some observers have met with a number of cases in a short time—Reiss saw 5 cases in three months. In a personal experience of eleven years I have met with 6 cases with autopsies. Up to 1894 W. Hunter was only able to refer to 250 published cases, and in the succeeding four years M'Phedran collected 29 more. In twenty-five years 7 cases occurred at St. Bartholomew's Hospital, which, according to Brunton and Tunnicliffe, is 1 in every 500,000 applications for treatment at that charity. In twenty-seven years there were 11 cases brought to autopsy at Guy's (Hilton Fagge).

*Etiology.—Age and Sex.*—It is commonest between the ages of twenty and thirty. According to Hunter's figures half the cases occur in this decade, and four-fifths between the ages of ten and forty. A certain proportion—I have collected 21 such cases—occur within the first ten years of life; exceptionally it has been seen within the first year, or even shortly after birth.

Females are more often attacked than males, the proportion between the two being nearly 2 to 1. Wilks puts the proportion higher—

two-thirds in women. This greater incidence of the disease in women seems to depend on a special association between pregnancy and this disease.

*Pregnancy.*—The influence of pregnancy is borne out by the fact that a large proportion of the cases occur in connection with this event. The liver is, it appears, peculiarly susceptible to morbid changes during pregnancy, and there is reason to believe that degenerative changes in the liver play a very important part in the production of puerperal eclampsia. As to the period of pregnancy at which acute yellow atrophy occurs, statistics show that it is commonest from the fourth to the seventh month.

Mental disturbance, shock, or fright has preceded the onset of the disease in a certain number of cases. The mental worry in persons with syphilis or in women that are pregnant, especially if unmarried, may tend further to depress the resistance of the body and so dispose to the disease.

In six fatal cases recorded by Hardie of acute yellow atrophy of the liver in Australia, importance was attached to the anxiety and fear with which women look forward to parturition in hot climates.

*Syphilis.*—The secondary stage of syphilis is sometimes accompanied by jaundice; this is usually harmless and yields to specific treatment. In rare instances acute yellow atrophy supervenes. This is said to be more often seen in women than in men. The syphilitic toxin would appear to attack the liver acutely just as it sometimes attacks the spinal cord, giving rise to acute myelitis.

*Alcoholic excess* in a few instances has apparently stood in a causal relation to acute yellow atrophy; in some instances the condition of acute red atrophy has been found after recent and undoubted excessive indulgence.

Inasmuch as alcohol is a protoplasmic poison, it is not improbable that the resistance of the liver being diminished by alcoholic excess, other causes making for acute yellow atrophy are thus enabled to become effective.

*The Influence of Pre-existing Hepatic Disease.*—The lesions of acute yellow atrophy may supervene in the course of morbid conditions of the liver such as cirrhosis, chronic venous congestion, or gall-stone obstruction. The onset is no doubt disposed to by the morbid condition of the organ. In these cases it is more convenient to describe the condition as icterus gravis rather than as acute yellow atrophy.

*Relation to Phosphorus Poisoning.*—Inasmuch as there is considerable resemblance between the clinical features of acute yellow atrophy and phosphorus poisoning, it has been thought that all cases of acute yellow atrophy are due to phosphorus poisoning. In support of this it might be urged that examples of what were for a time considered undoubted examples of acute yellow atrophy have, on further inquiry, turned out to be due to phosphorus poisoning.

Generally speaking, however, the differences between the two conditions are sufficiently marked to separate them and not to warrant the assumption that they are the same.

The differences are:—

(i.) In acute yellow atrophy the diminution in size is practically constant, whereas in phosphorus poisoning enlargement is the rule.

(ii.) In acute yellow atrophy the changes in the liver-cells lead to rapid disintegration with but slight increase in the amount of fat; while in phosphorus poisoning there is very extensive fatty change in the liver-cells, the amount of fat in the organ reaching 30 per cent as against 5 per cent in acute yellow atrophy.

It may be safely assumed that the two conditions are allied forms of icterus gravis, but there is no proof of the view that they are one and the same.

*Morbid Anatomy.*—The liver is greatly diminished in size and in weight; it may be half or even a third of its normal weight, often scaling 28 oz. instead of the normal 50 oz. It is uniformly atrophied in most cases, but exceptionally the less affected parts may form rather prominent projections. The changes are often more marked on the left lobe, where the morbid process probably often begins.

The capsule is wrinkled and loose, so that it can be picked up by the fingers, like the walls of a half-filled bladder. If a stream of water is turned on to the surface of the liver the capsule is thrown into folds and wrinkles by the jet of water. The outside of the organ has a greenish-yellow colour with red splashes; subserous hæmorrhages may be present under the capsule.

The liver is flabby and limp, and collapses and bends under its own weight; thus it readily doubles over on itself and is without the rigidity of a normal liver. This flabbiness of the organ allows it to drop back during life from the abdominal wall, its place being taken by the colon. As a result, the liver dulness may be entirely absent.

The liver cuts with the same kind of resistance that collapsed lung does, and, though very flabby, is not softer or more easily broken down by the finger than in health. Many writers, however, state that the liver is softened. Possibly this is more true in icterus gravis.

On section of the organ the surface is seen to be of a bright yellow colour. Usually, in addition to the more general yellow atrophy there are areas of red atrophy. As a rule, there is more of the yellow change; but in some rare examples of what have been called acute red atrophy, diffuse red atrophy greatly predominates or is universal. In the red areas the degenerative change is of oldest duration, while in the yellow areas it is more recent. It would appear that the longer the patient lives the

greater will be the extent of the red change found after death. Acute red atrophy is thus a further stage of acute yellow atrophy, and not a distinct condition. According to Hilton Fagge the red atrophy is often more extensive in the left lobe.

The outlines of the lobules are lost in the red areas, and with difficulty, if at all, discernible in the yellow areas; if visible, they are much smaller than in health.

The gall-bladder contains bile, but the larger bile ducts often only show mucus.

A scraping of the fresh section shows, under the microscope, blood corpuscles, degenerated liver-cells, and crystals of leucin, tyrosin, and xanthin. Leucin and tyrosin may be found in the blood of the veins of the liver, in the kidneys, and in the spirit in which portions of the liver have been preserved. In the alcoholic extract of the liver of acute yellow atrophy that had been kept for two years, Delépine found Charcot-Leyden crystals.

Histologically the appearances vary with the intensity of the change; for, as pointed out, the liver may suffer unequally in different parts. The liver-cells are disorganised, shrunken, angular, and yellow from bile; they stain badly, the nuclei being obscured. The protoplasm of the cells is granular and often contains pigment. It may indeed be difficult to recognise the tissue as liver except for the remains of the portal spaces, the appearances being chiefly those of cell debris, small-cell infiltration, and nuclei. There is small-cell infiltration in the portal spaces, starting from the portal vein and spreading into the lobules between the columns of liver-cells. Small-cell infiltration may also be seen around the intralobular veins. In chronic cases, or where the acute change supervenes on cirrhosis, considerable fibrosis may be present.

There is an increase in the amount of fat that can be extracted from the liver, some 5 per cent; but this by no means compares with the very considerable amount found in the liver of phosphorus poisoning.

In fresh sections crystals of leucin and tyrosin are seen. In places blood corpuscles are seen extravasated among the disorganised liver-cells. The smaller bile ducts show signs of proliferation, cholangitis, thus explaining the jaundice.

The appearances known as pseudo-bile canaliculi, consisting of columns of small cubical cells, are prominent in the small portal spaces, and may be regarded as an attempt at compensatory hyperplasia on the part of the remaining, comparatively healthy, liver-cells. The liver-cells divide, and thus small cells resembling minute bile ducts are produced. These regenerative processes are better marked when the disease is prolonged, and may not have time to develop when its course is very rapid. This regenerative process in acute yellow atrophy has been specially studied by Meder, Marchand, and Stroebe. In parts where the changes are less marked the liver-cells may be seen forming columns of larger size than the pseudo-bile canaliculi, as if the organ was reverting to the embryonic type of liver.

Micro-organisms have been found in some cases, but not in others, and no definite causal connection can be said to exist between any micro-organism and the changes found. Probably several different kinds of micro-organisms, as well as several poisons, are capable of producing the acute inflammatory and degenerative changes characterising acute yellow atrophy of the liver.

The kidneys are swollen, soft, bile-stained, and show small hæmorrhages. Microscopically, the epithelium of the tubules shows degeneration.

The spleen is softened, as in infective diseases, and often enlarged.

The heart is softened and swollen, and shows cloudy swelling. The blood, as in other toxic and septic conditions, stains the walls of the vessels and coagulates imperfectly. Hæmorrhages are found scattered through the body on the cutaneous, mucous, and serous surfaces. Meningeal and cerebral (Lafitte) hæmorrhages have been known to occur. Toxic changes in the vessel walls allow extravasation to take place. Brunton and Tunnicliffe point out that viperine poison has the same effect when applied locally to the mesentery of a frog.

The intestinal tract shows catarrhal inflammation and degeneration, while patches of necrosis in the stomach have been met with.

The body thus shows widespread degeneration due to a virulent poison. Changes of this nature have been described in the spinal cord.

*Nature of the Change.*—The essential factor is a very acute necrotic degeneration of the liver-cells with evidences of inflammation in the supporting fibrous tissue of the organ. The condition is a very acute hepatitis; chronic or protracted cases have been regarded as acute cirrhosis. It is analogous to, but more acute than the toxic changes seen in the liver, in phosphorus, iodoform, arsenic poisoning, or in lupinosis. In Germany many sheep die with jaundice, hæmorrhages, delirium, and acute yellow atrophy of the liver as a result of eating certain lupins. This disease—lupinosis—which is not met with in man, is thought to be due to a poison—ictrogen or lupinotoxin—produced by the agency of fungi in the husks of the seeds.

Where the poisons that lead to acute yellow atrophy are primarily produced is not known. But whether produced in the liver or elsewhere, the body suffers as a whole. In some instances the change in the liver may be a local manifestation of a general infection or intoxication, while in other instances the liver may be

primarily involved and the body secondarily affected.

*Symptoms.*—At the onset there may be nothing to distinguish the disease from ordinary catarrhal jaundice. It is true there is generally some rise of temperature, but this is often seen in the innocent jaundice, and is not enough to justify a gloomy prognosis. There is malaise, vomiting, constipation, bilious urine, and not uncommonly muscular pains.

This stage usually lasts five or six days, but may be prolonged for several weeks; it is succeeded by signs of mental disturbance, headache, delirium, screaming, restlessness, coma, and occasionally convulsions. The jaundice becomes more marked; it is due to obstruction in the smaller bile ducts, the result of inflammatory lesions in their walls produced by the same poison that is responsible for the acute degenerative changes in the liver-cells. In some exceptional cases of acute yellow atrophy there is no jaundice.

With the onset of these grave symptoms vomiting becomes urgent.

The tongue is usually dry, brown, and tremulous, and the teeth become coated with sordes. Dilatation of the pupils has been regarded as an important sign, and has been so extreme as to suggest belladonna poisoning; with the onset of grave symptoms the pulse quickens and becomes feeble and of low tension. The respiratory rate tends to be quickened or to become irregular.

The temperature varies, but is more often depressed than raised; it has been observed to rise before death. The presence or absence of fever has theoretically been correlated by Hanot with different microbic poisons, infection with the colon bacillus leading, like phosphorus poisoning, to a depressed temperature, while streptococcal and staphylococcal infections lead to pyrexia. Occasionally a red rash appears on the skin. Petechiæ and hæmorrhages occur under the skin, and blood may be passed in the motions; occasionally epistaxis and hæmaturia are observed, and in women metrorrhagia. Pregnant women abort.

The fæces may be darkened by blood so as to resemble bile; in the later stage it is improbable that bile passes into the duodenum, inasmuch as the bile ducts contain nothing but mucus. But as constipation exists throughout the disease, some of the fæces may contain bile excreted into the bowel at a very early stage of the disease. The dejecta are often extremely offensive. Diarrhœa is exceptional.

*Urine.*—The amount is somewhat diminished; it is high-coloured from bile pigment, and possibly in some instances from excess of urobilin. Albumin and tube-casts may be present. The amount of urea is greatly diminished.

Leucin and tyrosin, to which great importance is attached as replacing the urea and signifying the functional failure of the liver, are not invariably present, hence their absence does not disprove the existence of acute yellow atrophy. Sometimes one is present without the other. Leucin and tyrosin are sometimes spontaneously deposited from the urine.

On the other hand, leucin and tyrosin may be present in the urine in diseases where the liver is not affected in any way comparable to acute yellow atrophy, *e.g.* in erysipelas, typhoid fever, leukæmia variola.

*Liver Dulness.*—At the onset of grave symptoms the liver may or may not be found to be enlarged; this may be due to pre-existing disease such as cirrhosis, but it has been noticed in cases where this explanation does not hold. This enlargement is succeeded by diminution of the liver dulness, which may proceed rapidly until it entirely disappears. The complete disappearance is due to the atrophied and flabby liver falling away from the abdominal wall and allowing the colon to take its place.

The liver is tender on pressure.

The spleen may be made out to be enlarged.

Some degree of ascites may be present.

The stage of severe symptoms usually lasts for two days, and is followed by death in coma. In some instances the stage is prolonged; acute, subacute, and protracted classes have been made to embrace cases of varying severity. The protracted cases show changes which perhaps justify the term acute cirrhosis.

Diagnosis. — From phosphorus and allied forms of poisoning; the absence of any evidence that phosphorus or other poison has been taken or vomited is, of course, all-important. The progressive diminution in the hepatic dulness and the diminution in the amount of urea in the urine are strongly in favour of acute yellow atrophy. The presence of leucin and tyrosin is not conclusive, as they may be absent on the one hand in acute atrophy, and, on the other hand, be present in phosphorus poisoning and in other conditions, such as typhoid fever, erysipelas, and even occasionally in leukæmia.

In phosphorus poisoning there is an interval between the severe symptoms due to its irritant action and the onset of jaundice with severe constitutional symptoms; there is no interval between the first and second stages of acute yellow atrophy. There is more gastric irritation in phosphorus poisoning.

In biliary cirrhosis the progress of the disease is very chronic, while the liver is enlarged.

Prognosis.—When the disease has fully declared itself the prognosis is most gloomy; in fact, doubt must always arise as to the nature of cases that recover, and where an opportunity for examining the liver is not provided by death later. Some of the cases, of which a good number are on record, may have been examples of infective jaundice or Weil's disease of a severe character.

I have had such a case under my own care

where the diagnosis of acute atrophy, and death, the patient being in a condition of coma, seemed equally certain, but where recovery followed. Fagge refers to a case where a subsequent post-mortem showed the changes of acute yellow atrophy in a patient who recovered from the acute symptoms.

V. Kahlden reports a case in which death occurred some months after the acute symptoms, and where cirrhosis was in process of development as a result of the changes. This case is open to the explanation that it was one of acute hepatitis and icterus gravis rather than one of acute yellow atrophy.

Although doubt may arise as to the real nature of the lesion in the cases that recover after manifesting the characteristic symptoms, there are ample grounds for the statement that this does occur.

TREATMENT.—There is no means known of curing the disease; theoretically, free purgation in the early stages of the disease, to eliminate the toxins before their degenerative effects have been produced, might be recommended. Intestinal antiseptics, such as salol and β-naphthol, to reduce auto-intoxication as far as possible, may be given.

The excretion of the kidneys should be increased by the administration of citrate of caffein and free draughts of water. Intravenous transfusion has been performed with transient improvement.

Milk diet only should be given.

Vomiting may be combated by bismuth, dilute hydrocyanic acid, bimeconate of morphia, and effervescing mixtures.

## WEIL'S DISEASE


HISTORY . . . . . 487
NATURE . . . . . 487
ETIOLOGY . . . . . 487
BACTERIOLOGY . . . . . 487
MORBID ANATOMY . . . . . 487
SYMPTOMS . . . . . 487
DIAGNOSIS . . . . . 488
PROGNOSIS . . . . . 488
TREATMENT . . . . . 488


SYNONYMS: *Infective Jaundice, Bilious Typhoid.*

HISTORY.—In 1886 Weil described a condition of febrile jaundice associated with nephritis and enlargement of the spleen. It occurs in epidemics, one of which had previously been described by Weiss in 1866 as infectious jaundice. The disease was called after Weil of Heidelberg by his compatriots, but the French school did not consider it was different from icterus gravis or infectious jaundice. This unwillingness to acknowledge it as a new disease distinct from other forms of infectious jaundice is shared by Hunter in his article in Allbutt's *System of Medicine.*

NATURE.—Weil's disease is an excellent example of acute infective jaundice secondary to a hæmic infection, the nature of which has not been satisfactorily established. The jaundice is toxæmic, and has close analogies with that induced experimentally by means of toluylenediamine. It is allied to, but less acute than, acute yellow atrophy of the liver, and cases formerly recorded as examples of recovery from acute yellow atrophy would probably be regarded now by many as Weil's disease.

ETIOLOGY.—It usually occurs in males between the ages of twenty and forty, but children are sometimes affected. It is more likely to attack sewermen, butchers, soldiers, and others who follow certain occupations that expose the workers to infection. The onset of the disease is sometimes attributed to poisoning by bad meat.

Most of the cases occur in the summer months, and are met with in epidemics. It may arise repeatedly in the same place.

BACTERIOLOGY.—Jaeger and Banti have described a proteus bacillus in the blood. The former observer found the same organism in ducks dying of jaundice that frequented the water where his patients had bathed and presumably had been infected; the bacillus he described as B. proteus fluorescens.

Further observations are required on this point.

MORBID ANATOMY.—The tissues of the body show the effects of a general toxic process. There is cloudy swelling of the cells of the kidney, liver, and heart muscle, going on to the further change of fatty metamorphosis. The changes in the liver may progress further and resemble those in acute yellow atrophy; the mucous membrane of the bile ducts becomes swollen and degenerated.

Hæmorrhages may be present in the skin, mucous and serous membranes.

The spleen is swollen.

SYMPTOMS.—The disease begins with malaise, headache, fever, pains in the limbs, and, generally speaking, resembles influenza at its commencement. The pulse is rapid (120), but becomes slower after the onset of jaundice.

Jaundice begins on the second or third day, is generally slight, and lasts about two weeks; the motions may be clay-coloured, but usually contain bile, and are often loose.

The liver becomes enlarged and tender, and a marked feature of the disease is the splenic enlargement.

Fever reaching 103°-104° F. lasts for about a week; the temperature then falls and becomes normal at about the tenth day.

The urine is albuminous, contains bile pigment, and sometimes blood and bile acids. The presence of casts shows that there is tubal nephritis.

The pains in the limbs are especially marked in the calves; there is great prostration, giddiness, and some delirium at night.

Epistaxis, purpura, and various cutaneous rashes such as herpes, erythema, and urticaria may be met with.

A relapse may occur a week or so after the temperature has become normal; its occurrence may be suspected if, after the end of the first attack, the spleen remains enlarged. The relapse lasts about a week. Chauffard describes Weil's disease as "relapsing infectious jaundice," but in Germany relapses are comparatively infrequently described; thus in 84 cases, of which 73 were collected from German literature, Tymowski found that relapses were mentioned in 19.

DIAGNOSIS.—From epidemic catarrhal jaundice it is distinguished by its greater severity and evidence of its being not a local disease limited to the bile ducts, but a general infection, as shown by albuminuria and hæmorrhages, with secondary implication of the liver. The association with albuminuria would at once differentiate it from simple or from epidemic jaundice, or from the epidemic form that is sometimes seen in association with influenza.

From enteric fever the Widal's reaction would distinguish it. It was formerly described as "bilious typhoid" by Griesinger; but the lesions of typhoid fever are not found in the body after death, and further, it is extremely rare to see jaundice associated with typhoid fever.

The more severe examples of Weil's disease approach icterus gravis and acute yellow atrophy; the difference is one of degree, as far as our present knowledge goes.

Relapsing fever should be recognised by examination of the blood and the presence of the spirillum Obermeieri.

Hæmoglobinuric fever should be recognised by examination of the blood, by the history of exposure to malaria, and by the vomiting.

THE PROGNOSIS is fairly favourable, but convalescence may be protracted.

TREATMENT.—The patient should remain in bed until after the temperature has become normal, and should be restricted to a milk diet. All alcoholic drinks should be interdicted, and the patient should be encouraged to drink freely of water.

Intestinal antiseptics, such as salol, salicylate of bismuth, or β-naphthol, should be given.

## Liver—Tropical, Parasitic, and other Diseases

The physiology and general medical affections of the liver have already been considered. This section mainly treats of so-called "tropical" and parasitic affections of the liver, viz.:—

PERIHEPATITIS . . . . . 488
ACUTE HEPATITIS—MALARIAL HEPATITIS . 494
DERANGEMENTS OF CIRCULATION—
*Active Congestion* . . . . 496
*The Tropical Liver* . . . 497
*Passive Congestion* . . . . 498
SUPPURATION IN THE LIVER—
*In Pyæmia* . . . . . 501
*Tropical Abscess* . . . . 502
*Pylephlebitis* . . . . . 515
PARASITES . . . . . 519

### PERIHEPATITIS

*Definition and Synonyms* . . . 488
*Anatomical Considerations* . . . 489
*Varieties and Causation* . . . . 489
*Pathology and Pathological Changes* . 490
*Signs and Symptoms* . . . . 491
*Diagnosis and Prognosis* . . . . 493
*Treatment* . . . . . 494

*Synonyms.* — Capsulitis: adhesive or plastic hepatic inflammation.

*Definition.*—Perihepatitis signifies inflammation of the peritoneum covering the liver.

*Discussion of Terms employed.*—Although the term capsulitis is frequently employed to denote inflammation of the peritoneum covering of the liver, this use of the word is apt to, and does, cause confusion. The peritoneum is frequently considered as the capsule of the liver; but there is a capsule of subperitoneal (fibrous) tissue, enveloping the liver independently of the peritoneum, best seen at the several points where the peritoneum is wanting, but also present beneath the peritoneum itself.

The surface capsule must not in turn be confused with Glisson's capsule, which is the fibrous investment surrounding the vessels at the gate of the liver and penetrating with them into the liver substance, where it becomes continuous with the connective tissue of the liver itself. It is well to differentiate between the so-called capsulitis and perihepatitis. The former is a secondary affection resulting from congestive and chronic inflammatory changes commencing within the liver, or it may be secondary to, or occur coincidently with, primary perihepatic inflammation; the latter (perihepatitis) may be a primary ailment, but may also result from changes commencing in the parietal or visceral peritoneum around the liver, or may be but part of a general peritonitis.

The term capsulitis would be useful clinically were its meaning definitely understood. The (fibrous) capsule of the liver is a definite structure seen best where the peritoneum is wanting; but it exists also as a subperitoneal tissue, although thinned and stretched to a degree. The capsule is apparent in the hypertrophied condition met with in liver congestions due to cardiac regurgitation and cardiac and pulmonary obstruction, when it will be found as an opaque, white, tight-fitting covering to the liver. Capsulitis should be regarded as a condition the result of passive hepatic congestion or of chronic inflammatory changes, and although perihepatitis is apt to supervene, the thickened liver capsule may remain as such without perihepatic inflammation.

Perihepatitis, the result of general peritonitis, is dealt with under "Peritonitis," and is not considered in detail in this article.

*Anatomy.*—The peritoneum envelops the liver in a closely-fitting covering, everywhere present except between the layers of the coronary ligament, where the gall-bladder touches the liver, and at the transverse fissure.

The peritoneal reflections connecting the liver with the parietes and other viscera are arranged in three groups.

(1) The suspensory or falciform ligament slings the liver to the abdominal wall and the under surface of the diaphragm; in the fœtus the umbilical vein finds its way along the falciform ligament, but after birth the vein is obliterated and remains as the round ligament of the liver.

(2) The coronary ligament passes from the under surface of the diaphragm to the postero-superior aspect of the liver. A space is left between the two reflections forming it, which gives passage to the inferior vena cava and the lymphatics from the upper surface of the liver. At either end of the liver the two layers forming the coronary ligament approach each other and constitute the right and left lateral ligaments.

(3) The lesser or gastro-hepatic omentum passes from the stomach to the under surface of the liver in the neighbourhood of the transverse fissure; the lesser omentum gives passage to the portal vein, the hepatic artery, the ductis communis choledochius, and the lymphatics from the under surface of the liver. The peritoneum of both sacs touch the liver—the greater covering the upper surface wholly and the right side of the under surface as far as the antero-posterior fissure; to the left of the fissure the lesser sac of the peritoneum covers the under surface of the liver.

*The Relations of the Liver.*—The posterior and antero-superior aspects of the liver are in contact with the diaphragm for the most part, and only at the left anterior portion does the liver touch the abdominal parietes, in the region of the epigastrium. The under surface of the right lobe is in direct relation with the hepatic flexure of the colon, with the duodenum at the junction of its first and second portion, and with the right kidney in the order given from before backwards. On the under surface of the left lobe the liver overlaps the stomach. The gall-bladder occupies a groove on the under surface of the liver half-way along its anterior border. The gall-bladder differs from all the other structures in relation with the liver inasmuch as the peritoneum does not intervene between them.

VARIETIES.—*Primary perihepatitis* is by many authorities considered to have no place in the category of disease. It is frequently considered under the heading of hepatitis, or as part of the natural sequelæ of hepatic lesions generally, or as a part of general peritonitis only. Perihepatitis is, however, of considerable clinical importance, possessing both beneficial and deleterious properties sufficient to justify its position as a definite ailment.

*Secondary perihepatitis*, also, is too often considered of no clinical or pathological moment; its symptomatology is neglected and its sequelæ held of but little or no account. This neglect is not justified, for whether in its clinical indications or in its beneficial or pathological effects on the economy, secondary perihepatitis is of considerable moment.

*General perihepatitis* is the term employed when the serous covering of the liver is universally implicated. It is usually simply a part of general peritonitis, and seldom occurs as an isolated lesion.

*Local or patchy perihepatitis* signifies a limited inflammatory affection of the serous covering of the liver. The extent of the area may amount to a small patch only, but, on the other hand, it may approach universality.

*Local perihepatitis* may consist of a circumscribed patch of recent inflammatory exudation, as occurs in hepatic abscess where the pus, as it gains the surface of the liver, causes effusion of lymph. Perihepatitis arising in conjunction with pericarditis, pleurisy, fractured ribs, or surgical operations, is usually met with on the antero-superior aspect of the liver. In ulcer of the stomach, in malignant disease and inflammation of the stomach, duodenum, colon, pancreas, gall-bladder and bile ducts, kidney, etc., perihepatitis will occur at the part of the hepatic peritoneum in most direct apposition with the diseased organ. In cirrhosis of the liver, perihepatitis may be well-nigh universal; in syphilis, two or three large patches are the rule; in cancerous and tuberculous infiltrations recent lymph may be present, but more frequently numerous ill-developed, fibrous adhesions pass from the apices of the various nodules to the parietal peritoneum or to the peritoneum covering adjacent viscera. In every hepatic abnormality involving the surface or capsule (fibrous) of the liver, perihepatitis is the rule and not the exception.

*Induced Perihepatitis.*—Attempts are sometimes made to induce perihepatitis by applications of counter-irritants to the abdominal wall over the region of the liver. The object of this practice in threatening abscess of the liver is to provide a safe path by which the pus may reach the surface. Perihepatitis is also induced in cases of hydatids or liver abscess, etc., by the surgeon cutting down upon and exposing the liver, and if necessary stitching the visceral and parietal surfaces of the peritoneum together. In many cases of cirrhosis of the liver where the hepatic and parietal layers of the adjacent surfaces are made to adhere, a channel would be provided along which blood-vessels could

reach the liver and establish a freer vascularity of the organ by way of blood-vessels from the abdominal wall. A conservation of this nature is frequently established by pathological change, but there can be no doubt that surgical interference in this direction should be much more frequently practised than it is. The peritoneum over the liver is accredited with the power of withstanding rough handling better than any other part of the membrane, and short of stitching the liver to the wall of the abdomen, a mere ruffling of the adjacent serous surfaces would bring about the salutary adhesive process.

CAUSATION. — *Traumatic Perihepatitis.* — Inflammation of the antero-superior aspect of the liver, more especially in the neighbourhood of the costal margin, the result of a blow or pressure, is by no means uncommon. In the normal state the liver protrudes beyond the rib cartilages in the epigastrium, and here the result of bruising of the liver and of the peritoneal covering is most often found.

The simplest form of traumatic perihepatitis is that met with as the result of tight-lacing. The portions of the thoracic and abdominal wall covering the liver are held immovable, and at the same time pressed firmly against the liver by the tight garments; but the liver is moved to and fro by the contractions of the diaphragm, with the result that the opposite surfaces are bruised and chafed and a low form of circumscribed peritonitis is set up.

Injuries of the liver by fractured ribs must involve its peritoneal covering, which splits like a kid glove over a clenched fist.

When the liver is ruptured the peritoneum may or may not give way to an extent commensurate with the injury; the rent in the peritoneum may fall considerably short of the tear in the liver, causing the blood to escape into a more or less complete bag of hepatic peritoneum. The rectus muscle in the epigastrium protects the liver beneath from injury; but over the fundus of the gall-bladder, where the parietal tissues are wholly aponeurotic, little or no protection is afforded against a blow in this region, and the frequency of peritoneal adhesions round the gall-bladder, especially when the organ is distended, is thus partly accounted for.

The liver, when struck by a blow in front, may rupture posteriorly or on the under surface only, and a local peritonitis may resolve itself into firm fibrous adhesions to neighbouring organs.

*Primary perihepatitis* is, in the opinion of the writer, a condition met with especially in the Tropics, as the result of exposure to cold. Just as pleurisy may occur as a primary disease independent of pneumonia, so a localised or patchy perihepatitis may occur on the liver and from similar causes. In the Tropics it is the abdominal rather than the thoracic organs which are called upon to resist climatic influences, whereas in temperate climates the opposite is the case. Exposure of the abdomen, more especially at night, may end in an intestinal flux, or may give rise to acute pain in the right lower costal region, associated with increase of temperature, with hurried, shallow breathing, etc., signs and symptoms closely resembling pleurisy of the right side. We are apt to assume that the ailment is primarily pleuritic and secondarily perihepatitic, and even the friction heard through the chest wall may be peritoneal instead of pleuritic; yet are we slow to ascribe the friction sound to peritonitis. A careful investigation may elucidate the fact that perihepatitis, and not pleurisy, is the lesion, or that the inflammatory changes started together and not consecutively, as is so often assumed.

*Secondary perihepatitis* may be the result of lesions commencing within the liver. A hydatid tumour may excite a localised perihepatitis as it extends beyond the liver area; but this is not invariably the case, and in consequence of the absence of a benign perihepatitis the contents of the hydatid may find their way into the peritoneal cavity. In liver abscesses similar phenomena may develop—the presence or absence of adhesions determining the course of the pus and the safety of the patient. Cancerous and tubercular infiltrations of the liver seldom cause well-marked hepatic adhesions; there may be no appearance of the exudation on the liver in either of these conditions, and even when it does occur it usually consists of weak adhesions between merely the apices of nodules and the parietal peritoneum.

Syphilitic, amyloid, hyperæmic, and inflamed conditions of the liver may each be associated with or may set up a concomitant perihepatitis.

PATHOLOGY AND PATHOLOGICAL CHANGES.— Non-infective perihepatitis has in the light of recent pathology come to be regarded as an impossibility. Bacterial invasion is at the root of inflammatory changes in the peritoneum as well as of all other serous membranes.

In connection with the peritoneum certain bacteria are ever ready to launch themselves upon an altered or abnormal portion of its surface; of these the bacillus coli communis is the most commonly met with. Resident in a normal state within the whole length of the alimentary canal, with physiological but with no pathological powers, yet this very bacillus, in altered conditions, may become pathogenetic to a degree. Should the walls of the intestinal tract become diseased, the bacillus coli travels beyond its natural limits, bearing in or around it a toxic agency. On the other hand, should any portion of the abdominal contents depart from the normal, thither will the bacillus drift and set up inflammatory or destructive changes.

The escape of the colon bacillus from the intestinal tract need not be owing to direct injury of the intestinal walls, although, in the case of derangement of the liver, the intestinal functions will be necessarily upset. Reasoning from this basis, it is plain that a disturbance of nutrition of the endothelial lining of the peritoneal surface of the liver may attract the bacillus coli communis to the altered spot and inflammatory changes ensue. That the inflammation set up should remain limited, or that the fluids exuded should fail to set up general peritonitis by finding their way into the general cavity of the peritoneum, seems remarkable. Explanations of this limitation are to be sought for in the fact that the peritoneum is endowed with a certain power of counteracting and resisting the toxic power of such infective influences. Experiment and every-day experiences in abdominal surgery prove this to be the case, and the principle admits of application in the case of individual organs being attacked. Moreover, the general pressure maintained in all parts of the abdominal contents thwarts the precipitation of fluid exudations to the lower reaches of the cavity. Were this not the case, pelvic peritoneal inflammation subsequent to any and every inflammation starting within the abdominal cavity would be more common. Pelvic peritonitis, however, does not necessarily, or even usually, succeed—showing that the inflammatory fluids, issuing around the individual organ, are naturally primarily restricted to the peritoneum in the immediate neighbourhood of the diseased part.

With the bacillus coli communis other bacteria appear, and both streptococci and staphylococci may infect a local peritoneal inflammation.

The presence of the micrococcus lanceolatus (the pneumococcus of Fraenkel) does not seem more common in perihepatitis than in other peritoneal inflammations, although the proximity of the liver to the lung and intimate lymphatic association therewith might have implied that such would be the case.

The amœba coli is also accredited with causing perihepatitis in two situations at least—viz., where the colon touches the liver, and again, upon the upper aspect of the liver. The amœba coli, like the bacillus coli, is found at times in the intestines of healthy persons; but in several abdominal diseases and functional derangements it becomes endowed with pathological powers, as witnessed in the so-called amœbic dysentery. Not only so, but it may become extra-intestinal by finding its way through the intestinal walls and gaining the cavity of the peritoneum. When the amœba passes through the part of the colon in contact with the liver at the hepatic flexure the localised perihepatitis admits of explanation; but how the parasite gains the upper surface of the liver is not so apparent. It would seem that the infection of this region is determined by the general drift of lymphatic fluids towards the upper part of the abdominal cavity, and that here, between the constantly-moving liver and diaphragm, a perihepatitis is induced which binds the liver to the diaphragm and the adjacent parietal wall.

After perihepatitis the parts may return to their normal condition without any trace of the inflammatory disturbance being left. The lymph effused on the surface may undergo fatty degeneration and be absorbed together with the inflammatory fluids exuded. It is more usual, however, for the lymph to become organised and to remain:—(1) Of a *membranous* sheet of adhesions, closely binding the liver to the diaphragm and the abdominal wall or to the neighbouring viscera; the fibrous material may be as tough as leather—so much so that on the post-mortem table the parts cannot be torn apart. (2) The adhesions may have become stretched whilst in the pliant state, and, if so, they will appear as *filamentous* adhesions allowing of fairly free movement between the liver and the neighbouring structures. During convalescence from a perihepatitis, therefore, it is important that the patient should have massage, and take exercise calculated to expand the chest, for the purpose of lengthening the adhesions; for when the adhesions are of a membranous character the right side of the chest is hampered in its movement, the right shoulder drops, a sense of weight remains in the right chest, especially when any exercise is taken; therefore everything possible should be done to prevent "the liver sticking to the ribs" during the early stages of convalescence.

Signs and Symptoms.—Acute perihepatitis, occurring primarily with or without pleurisy, or secondary to acute affections of the liver, is usually ushered in by a definite train of symptoms. An initial rigor, should it occur, is no guide to the nature of the peritoneal lesion around the liver. Pain diffused over the epigastrium and the right lower ribs suggests a general perihepatitis, whilst a pronounced pain, referred to a more or less definite spot, betokens a localised perihepatitis. Respiration is hampered, and the right side steadied and fixed against movement. A short, catching, hacking, dry cough is a frequent symptom often provoked when the liver is being examined. The pulse is hard and small, becoming softer only when free exudation occurs. The muscles of the abdominal wall generally, and over the region of the liver especially, become tense and rigid the moment any attempt is made to examine the liver. Percussion below the costal margin is attended by pain. When the liver is grasped and moved between the hands, by placing one over the right lower ribs behind and the other over the region of the epigastrium, acute pain is usually elicited, and frequently a sharp, darting pain shoots up to the shoulder, causing

the patient to scream and remonstrate strenuously against the manipulation being repeated. The pain from perihepatitis is not usually diffused over the shoulder, as in the case in hepatic pain, but is frequently concentrated on the cervical aspect of the angle formed between the clavicle and the acromion process of the scapula; the patient will frequently press a finger firmly and deeply into the recess to relieve pain. Vomiting occurs especially when the stomach walls are in contact with the area of perihepatic inflammation. Hiccough is at times troublesome. The usual accompaniments of "feverishness," headache, foul tongue, etc., are less frequent in perihepatitis than in almost any other inflammatory state. The facial aspect in perihepatitis, as compared with hepatic ailments, is usually most marked. In lesion confined to the liver the patient is apt to despise his affection, can with difficulty be kept in bed or even indoors, and his aspects show little sign of suffering. When perihepatitis supervenes all this is changed, and the patient's drawn features and general condition of suffering indicate that the peritoneum is involved in the inflammation.

In every case of perihepatitis the genesis of the ailment must be sought for. The condition is so frequently secondary to some diseased condition of the liver, peritoneum, pleura, pericardium, lungs, heart, and kidney, that each of these ought to be systematically examined in turn. Well-nigh every affection of the liver may engender perihepatitis; it may be but a part of a general peritonitis, acute or chronic; pleurisy and pneumonia may coexist, may precede, or may follow a perihepatitis; pericarditis is a frequent concomitant and a possible etiological factor. Regurgitant venous conditions due to cardiac incompetency or cardiac and pulmonary obstruction are always associated with mechanical congestion of the liver, a tense capsule, and a tendency to perihepatitis. Interstitial nephritic changes with albuminuria, ascites, etc., are frequently the antecedents of chronic peritonitis, and therefore of general perihepatitis. In every case of chronic perihepatitis kidney mischief is to be suspected and sought for. Of the signs and symptoms observable in perihepatitis none are more characteristic than the position assumed by the patient. Lying on the left side is attended by pain of a severe and characteristic nature. The patient describes the pain as of a sharp, dragging character, whilst at the same time the breathing and the heart rhythm are interfered with. Lying on the right side is also frequently accompanied by pain, and the patient finds most comfort on the back with the knees drawn up and the head and shoulders well supported by pillows.

The friction sounds of perihepatitis differ to some extent from those heard in pleurisy. In recent pleurisy the friction note is high-pitched, continuous, and formed as it were by a number of short, rapid, consecutive crackles. The peritoneal rub is attended by a low note, is heard chiefly at the beginning of inspiration and at the end of expiration, and consists of a few slowly-emitted rubs.

*Prevalence of Perihepatic Adhesions.*—At 200 post-mortem observations made by the writer, 55 presented perihepatic adhesions in some form. The cases were not selected, the notes being taken from cases irrespective of the cause of death. Forty of the observations were made in Hong-Kong, and the remainder in London. In the Tropics, although the number observed was too small for positive opinion, Europeans presented hepatic adhesions more frequently than in the case of natives. In 30 of the 55 cases there was no suspicion of hepatic disease during life.

*Perihepatitis and Ascites.*—General perihepatitis, apart from hepatic lesions and general peritonitis, is so rare an affection that there are but few data to go upon as to its immediate effects and sequelæ. Such evidence as there is, however, would appear to indicate that perihepatitis, *per se*, is not directly provocative of ascites. That perihepatic infiltrations and inflammations cause constrictions of the vessels at the gate of the liver to any marked extent, and therefore become a potent factor in developing ascites, is not in evidence. Were it so, it is not likely that the bile passages would so frequently escape constriction, while the portal vein, with which the bile ducts are so closely allied in position, would alone suffer. And as we know jaundice to be a rare symptom in perihepatitis, we have no right to assume that ascites is likely to follow perihepatic thickening. With the liver in a state of even advanced cirrhosis, ascites when it occurs is a late development, and one which post-mortem evidence shows is most frequently associated with chronic peritonitis. The supervention of general peritonitis (with perihepatitis) in a case of cirrhosis appears to be the state most favourable to develop ascitic complications. In cirrhosis of the liver, especially when associated with interstitial nephritis, perihepatitis is a frequent lesion. The adhesions formed in such a condition may be confined to the liver, spleen, and colon, or they may be part of a general chronic peritonitis so commonly met with in cirrhosis with ascites. The immediate part played by a cirrhotic liver in causing ascites is not yet settled, for it would appear that ascites seldom becomes pronounced until a general peritonitic change supervenes. But few cases of universal perihepatitis without general peritonitis have been observed, and as in these ascites has not been recorded, it is fair to assume that perihepatitis by itself is not a primary determining cause of ascites.

The *sequelæ* of perihepatitis may be classified

into the beneficial and the harmful. The beneficial effects of perihepatitis are seen when the liver adheres to other organs as the result of disease commencing within its own boundaries or arising in neighbouring organs. Abscesses, hydatids, etc. extending from the liver may, by the perihepatitis they induce, be guided by channels to safe outlets. Pus may come to the surface, may be conducted through the diaphragm and lung, may reach the colon, stomach, etc., along tracts which prevent its bursting in the peritoneum or pleura. In the same way in hydatids and even in malignant disease, a barrier, the result of perihepatitis, may be formed which may check for a time, at any rate, serious results. Disease in neighbouring organs may also be arrested by a timely perihepatitis. Perforating ulcers of the stomach, disease in the colon and pancreas, may be arrested at the liver walls by perihepatitic barriers of inflammatory tissue. The beneficial results of perihepatitis are also seen in cirrhosis, where, by extensive adhesions, the vascular supply of the liver is benefited to a high degree. The detrimental action of perihepatitis is evident in the arrest of movement, caused by the liver and diaphragm being bound together, whereby the breathing is impeded, exercise is curtailed, the functions of the liver do not derive the full benefit of the respiratory movements, and in consequence the general health becomes impaired.

Induration of the liver tissue is another pathological state ascribed to perihepatitis. Recurrent attacks of perihepatitis, seen especially in residents in the Tropics, there can be no doubt, tend to infiltrate the liver with fibrous tissue; but that perihepatitis and subsequent capsulitis directly cause a general induration of the liver substance is a sequel of conditions not by any means constant. In many post-mortem examinations such a train of pathological changes would seem undoubted, but in others the liver retains a normal appearance, or it may even become softer than is natural. In yet another group of cases of perihepatitis the branches of the portal vein may be found surrounded by fibrous thickenings whilst the liver-cells retain their normal appearance. The information at our disposal is too limited to come to a definite conclusion in the matter; but the question seems to be mostly one of time and degree, as recurrent attacks of perihepatitis may no doubt bring about some degree of hepatic induration.

The post-mortem appearances of perihepatic inflammation may be (1) a general coating of recent lymph on the surface of the liver, which can be easily peeled off; (2) the lymph may become organised and form fibrous material, which may extend as a tough membranous layer binding the liver and the adjacent parts firmly together, so that the liver can only move during respiration with the diaphragm and the viscera in contact with it as a whole; or (3) the fibrous material may be stretched and thinned out into filamentous bands which allow of fairly free movement.

Diagnosis.—Acute perihepatitis is most apt to be mistaken for pleurisy of the right side. When we consider how often even the presence of a liver abscess is obscured by and mistaken for a right basal pneumonia, we are prepared for serious difficulty in this direction. Pleurisy and perihepatitis are, as shown above, frequently concomitant diseases. In almost every case of old pleuritic adhesions at the lower part of the right chest, perihepatic adhesions will be met with post-mortem, and the two conditions may have arisen, developed, and run their course in common. It is usual to assume that the affections are not coincident, and that the pleurisy is the primary ailment; this is frequently the case, no doubt, but in many instances the peritoneum may have been first affected, or the two conditions may have started together. The pleuritic signs and symptoms are apt to, and usually do, obscure the peritoneal. The friction sounds are ascribed to the pleural state, whereas the peritoneal rub may be the more marked. By grasping the liver by the hands placed front and back, and moving the liver backwards and forwards, the diagnosis may be cleared up. The sharp, excruciating pain shooting up to the clavicular angle can only be elucidated by this method, and the diagnosis of perihepatitis with or without pleurisy can be established. There are several other diagnostic features which are sufficiently obvious from a study of the signs and symptoms.

The diagnosis of perihepatitis in cases of interstitial nephritis, accompanied by cirrhosis and ascites, is of little clinical consequence, as the perihepatitis itself plays but a small part in hastening the end; and, again, perihepatitis is assumed to be present when the signs of chronic peritonitis are apparent.

The most important clinical point to settle is whether a recurring perihepatitis is or is not accompanied by cirrhosis. A patient who has cirrhosis or hepatic induration will in all probability have at the same time peritoneal adhesions over the liver. Exposure to chill, injury, etc. may, however, set up a subacute perihepatitis, supervening upon old perihepatic trouble, without marked symptoms of hepatitis or increasing induration.

If the liver tissue is not seriously involved—and it may not be—it is important to check the perihepatitis, however slight or subacute, as there is a danger of the liver becoming, in time, indurated and even cirrhosed. On the other hand, if cirrhosis is marked, a further development of perihepatic adhesions is calculated to do good by establishing relief

channels for the vascular and lymphatic communications of the liver. The practitioner is therefore in a dilemma concerning the prognosis, as, on the one hand, perihepatic adhesions by inducing induration may injure the liver tissue, whilst, on the other hand, given a cirrhosed liver, perihepatic adhesions will help the liver to acquire an additional blood-supply.

If jaundice is present with the other clinical evidence of cirrhosis, then is it almost certain that the liver is seriously cirrhosed; but when jaundice and ascites are absent, the pain, etc., in the region of the liver is in all probability of perihepatic origin and the liver tissue is not seriously involved.

Prognosis. — Perihepatitis may be a mere local condition brought on by chill and exposure, and may subside without further recurrence. Perihepatitis supervening in a case of cirrhosis may be beneficial in its effects by allowing an increased and alternative blood-supply to the liver. Recurrent attacks of perihepatitis in a person with a fairly normal liver may set up a capsulitis, and the fibrous change advancing into the liver substance will, in time, interfere with the nutrition of the liver-cells themselves, inducing atrophy. It is therefore of the utmost importance in prognosis to ascertain the condition of the liver, of the peritoneum, and of the kidneys before coming to a conclusion.

Treatment.—When the signs and symptoms of perihepatitis are pronounced, a definite line of treatment must be pursued. Rest in bed is of primary importance, whilst at the same time the warmth of the bed-clothes is beneficial. The diet is of consequence — red meats and alcohol being strictly withheld. Locally, the pain may be relieved by poultices and hot packs; or, when very severe, the right side may require strapping, so as to prevent movement. Counter-irritation may be applied over the hepatic area by means of oil of mustard, sinapisms, or the liniment of iodine. Eight or ten leeches applied over the region of the liver frequently checks the progress of the inflammation. Should ascites develop, paracentesis may become necessary.

In addition to local treatment the liver may be acted upon by therapeutic means. Sulphate of soda will best serve the purpose of a hepatic drain, the effervescing preparation being the more agreeable form in which to administer the drug. Recurrent perihepatitis must be treated, to begin with, in a manner similar to that employed for the acute form. As hepatic congestion is usually present with this condition, it is well to administer the chloride of ammonium in an alkaline mixture for some days after the more pronounced symptoms have disappeared. Iodide of potassium is to be recommended as a means of counteracting the local tendency to fibrous deposit, after a subacute attack of perihepatitis has subsided.

Old-standing adhesions, the result of perihepatitis, are to be treated by massage and by hot packs, followed by inunctions (of mercurial ointment). Systematic exercises, especially devoted to chest expansion and movements of the arms, especially the right, are of benefit in loosening the adhesions between the liver and the diaphragm or abdominal wall.

Perihepatitis may be induced in advanced cirrhosis by exposing the liver by abdominal section, and "ruffling" the peritoneum over the liver by scraping with a blunt instrument or other means. The beneficial effects of such treatment can only be expected in exceptional cases, where cirrhosis of the liver is marked and uncomplicated by kidney disease, by cardiac or pulmonary regurgitation, or by any of the deposits and infiltrations to which the liver is liable.

### Acute Hepatitis, Malarial Hepatitis

Definition.—Acute inflammation of the parenchyma of the liver.

Etiology.—By far the commonest cause of acute hepatitis is malaria. In temperate climates derangement of the liver, associated with a determination of blood to the organ, seldom passes beyond the stage of acute hyperæmia. The chief exception to this statement is to be met with in cases of alcoholic engorgement; but primary hepatitis is not usually of alcoholic origin. Alcohol induces a gradual induration of the connective tissue of the liver, the chronicity of which is occasionally interrupted by acute hyperæmia of the organ, or it may occasionally amount to an actual acute inflammation.

A primary hepatitis, however, is uncommon outside warm climates, and the cause, rightly or wrongly, is assigned to malaria. Exposure to cold or to the sun alone, or climate, may be factors in the genesis of hepatitis, even when the malarial parasite is present in the blood; and proofs are not wanting that from some such causes, independently of malaria, hepatitis may arise. The writer treated a case of acute hepatitis going on to suppuration on a man who had arrived in the Tropics but five weeks previously, who had never had dysentery nor an attack of malaria.

Signs and Symptoms.—The chief distinction between congestion and inflammation of the liver is the absence or presence of fever. It may be taken as a maxim that, given a temperature over 100° F. in derangements confined to the liver itself, hepatitis is present. The borderland between hyperæmia and inflammation of the liver is but a step, and the state of the temperature seems the most reliable factor in determining the distinction. Each possesses several points of individuality, but the signs and symptoms of hepatitis seem but an aggravated form of those met with in hyperæmia.

The pain in hepatitis is greater, both whilst the patient is at rest and during movement;

the shoulder-ache is more definite and concentrated; vomiting and retching are more pronounced and frequent; and blood is more often present in the vomit. The thoracic movements in hepatitis are shallow in the extreme; the abdomen is rigid; and even the weight of the bed-clothes causes discomfort. Restlessness, irritability, impossibility of lying on either side or flat on the back with a low pillow, aid in the diagnosis. The patient prefers to sit propped up in bed with a support to the back and with the knees flexed in a semi-sitting posture.

The tongue is usually red, except on the posterior part, where it is coated with a dry brownish-yellow fur; thirst is distressing; vomiting of bilious matter, becoming ultimately of a watery greenish appearance, attended by violent retching, may be incessant, causing exhaustion and at times collapse. The bowels may remain inoperative, probably from paresis, for days, or an acrid fluid diarrhœa prevails with frequent calls to stool. The contents of the bowel when expelled possess a foul smell, inducing frequently an attack of vomiting before the patient leaves the closet.

The urine is scanty, high-coloured, and loaded with urates; occasionally albumin is present, and the amount of urea varies from day to day, now increased, now diminished in quantity.

The pulse is quick, hard, and full; the breathing is shallow; and a dry cough, causing at times acute suffering, is always dreaded by the patient.

The liver, when examined, in primary hepatitis will be found increased in area in every direction. The upper limit of dulness may extend as high as the nipple; and far beyond the costal margin, it may reach to just above the umbilicus. In other cases the area of the liver dulness may not be so pronouncedly increased. In hepatitis associated with spirit drinking there may be little or no enlargement, owing, no doubt, to the presence of cirrhosis, but in primary hepatitis the increase in size is usually as described. On percussion, palpation, and movement of the liver (by a hand placed back and front) acute pain is complained of; and when perihepatitis is present, as is so often the case, the agony of pain caused by the examination causes positive dread of its being repeated. Jaundice, even in a mild degree, is quite exceptional.

When malaria is the cause of hepatitis the spleen will be found enlarged; but in non-malarial forms of the disease it is not usual to find the area of the spleen increased in size. Dysentery may precede, may appear simultaneously with, or may occur during an attack of hepatitis. Should any of these conditions prevail, there is great likelihood of the inflammation ending in suppuration. An attack of simple hepatitis usually lasts a week or ten days.

*Morbid Anatomy.*—Opportunities of examining the liver post-mortem in the initial stage of acute hepatitis seldom occur; but in post-mortem examinations of patients dead of acute malarial diseases, the following changes will be noted:—

(1) The liver may be considerably or only somewhat enlarged, and of a dusky, pigmented, slate-grey appearance. When cut into, the vessels will be found charged with blood, and the parenchyma of the liver markedly softened. Perihepatitis is commonly found to a greater or less extent, and the general capsule is thickened.

(2) When the blood of the liver is examined microscopically in the early stages of acute malarial disease, the capillaries will be found to contain numbers of pigment-laden cells clinging to the walls of the blood-vessels or accumulated in masses forming actual thrombi. The cells are leucocytes, phagocytes, or the large white cells derived from the splenic tissue, and containing, it may be, not only melanin, but incorporated infected blood corpuscles, free malarial parasites, and even the products of hæmoglobinic disintegration. These macrophages and pigment-laden cells are carried by the splenic vein from the spleen, where the cells laden with the malarial parasites seem to have deposited their burden, as but comparatively few malarial parasites are met with in the blood of the liver capillaries. At this stage the cells of the liver show a cloudy swelling.

(3) At a later stage of the malarial infection, the characteristic melanin will be found incorporated in the endothelium lining of the cells. The pigmented endothelial cells swell and still further tend to block the capillary channels and produce areas of stains, with the result that atrophy or necrosis of some of the liver lobules occurs and the vascular network dilates.

(4) At a still later period the melanosis is found to be extravascular and the pigment infests the whole lobule, but accumulates chiefly at the periphery. The phagocytes are meanwhile actively engaged in removing the detritus left in the necrotic patch, and a process of regeneration of the liver parenchyma commences.

(5) The melanin, in course of time, reaches the outer rim of the lobule and becomes peri- or inter-lobular and extravascular. A slate-grey coloured rim is seen around each lobule, the lobule itself appearing of a brownish tinge within its darker ring of pigment.

(6) Finally the pigment diminishes and then disappears, leaving the liver enlarged and granular, the lobules distinct and surrounded by a pinkish-tinged hyperplastic connective tissue.

The disappearance of the malarial melanin from the parenchyma of the liver is effected no doubt by the mononuclear and polymorphonuclear cells, which carry their burden to the lymphatics and the lymphatic glands. In the

glands the pigment is ultimately detained and stored, hence the dark colour of the glands at the gate of the liver and elsewhere in chronic malaria.

It will be observed that, alongside of atrophy, degeneration, and phagocytic removal of necrotic tissue, regeneration proceeds amain. In those parts where the stroma is destroyed by a hyperplasm no regeneration of liver tissue is possible; but in others, after the pigment in the endothelial cells lining the capillary walls has been got rid of, and the blood-current restored, young hepatic cells are seen occupying the stroma and arranging themselves in rows radiating from the centre in the manner characteristic of cells in a hepatic lobule.

The structure of a tropical liver can, from the account just given, be readily understood. The perilobular thickening, the destruction of liver-cells, and their place taken by areas of dilated vascular and lymphatic vessels in a connective-tissue stroma, the increase in size of some lobules and diminution in others, account for the large smooth-surfaced liver met with as the results of malaria.

HYPERÆMIA OR CONGESTION OF THE LIVER


A. ACTIVE CONGESTION . . . . 496
*Causation* . . . . . 496
*The Tropical Liver* . . . . 497
*Effects of "Climate"* . . . 497
*Increase of Physiological Functions* 497
*Impairment of Physiological Functions* . . . . 497
*Signs and Symptoms* . . . 498
*Diagnosis* . . . . . 498
*Prognosis* . . . . . 498
B. PASSIVE CONGESTION . . . . 498
*Mechanical Venous Obstruction* . . 498
*Physiological Passive Congestion* . 499
*The Part played by the Nervous System* 499
TREATMENT . . . . . 499


Under this title several hepatic ailments, differing considerably in their symptoms and wholly in their cause, are included. It is clinically unfortunate that such is the case; and although several would-be distinctive names have been bestowed upon each of them, the terms active and passive congestion continue to be the accepted nomenclature.

A. ACTIVE CONGESTION.—*Definition.*—When a large excess of blood is present in the liver owing to increased "inflow" from the portal vein or hepatic artery, the liver is said to be in a state of active congestion or hyperæmia.

*Causation.*—In health, after every meal, the quantity of blood brought to the liver by the portal vein is increased in amount; and as the digestion of the meal proceeds, the hyperæmia lessens and finally subsides. Owing to several causes, however, the hyperæmia created during digestion of a meal may be excessive, and may continue even after the meal has completely disappeared from the stomach. The failure of the physiological flow and ebb of blood to the liver may be owing to one of the following causes:—

(1) The amount of the solids and fluids ingested may be in excess of the power of the liver to deal with or assimilate. (2) The character of the food may be of so irritating a character, or the quantity of alcohol imbibed may be so excessive, that a hepatic engorgement ensues. (3) The gastro-intestinal tract may, owing to altered conditions or to disease, produce toxic elements which reach the liver by way of the portal vein and set up hepatic irritation and consequent hyperæmia. (4) The liver may be rendered hyperæmic through the blood of the general circulation becoming impregnated by toxicants, such as malaria, the poisons of several of the common fevers, yellow fever, etc. (5) Active congestion of the liver may result from exposure to cold.

Several of these causes give rise to what is popularly known as "biliousness," a term which, although elastic in its meaning, conveys fairly definitely what is implied. In consequence of over-stimulation by alcohol, by wines rich in salts, by excess of food, etc., the functions of the liver are interfered with to such an extent that assimilation and excretion are over-taxed; the blood is surcharged with salts, which may result in a gouty storm; the urine becomes loaded with urates, alternating with oxalates, or it may be with albumin or sugar. Slight jaundice may be present, caused either by pressure on the radicles of the bile ducts, or it may be the result of catarrh of the duodenum and common bile duct, induced by the dyspepsia which almost invariably accompanies hepatic congestion. Gastric catarrh, as distinct from intestinal catarrh, is often a feature in congestion of the liver. The gastric juice may fail in its function whilst the intestinal juices and the assimilation of food in the intestine continue to be normal. In such a condition the congestion of the liver is secondary to the gastric condition, and evanescent in character, and not the cause thereof. Were the liver primarily hyperæmic, the whole alimentary canal would be affected simultaneously.

In another group the intestinal tract is the primary seat of pathological change. Catarrh, ulcers, excoriations, inflammatory changes, or retained fæces give rise to abnormal chemical products which reach the liver by the portal vein and cause a local determination of blood. Auto-intoxication from this source reaches the system by way of the portal vein, and the liver is the organ disturbed in the first instance. The hyperæmia of the liver may be but the first stage of the sequence which afterwards induces hepatitis and then cirrhosis or suppuration. According as the products absorbed are wholly bacterial in their nature, or bacteria

accompanied by the products of their life and death, so will the hyperæmia end.

The enlargement of the liver which occurs in the febrile state connected with the eruptive and other fevers is due to a general blood infection. The spleen is involved in the same lesion, and frequently the kidneys, the lungs, the intestine, the brain, etc., show signs of functional disturbance. Malaria is a toxic agent belonging to this group, and yellow fever in its several forms probably affects the system in this fashion.

So-called "menstrual jaundice" is by some regarded as toxic in origin, and by others as a neurotic affection. At the grand climacteric, cases have been recorded in which congestion of the liver with slight jaundice has occurred, and even during suppression of the ordinary menstruation similar symptoms have been noted.

The jaundice which accompanies influenza in some outbreaks is accompanied by a hyperæmia of the liver. The attack lasts from five to eight days, and the divergence of the biliary secretion is chiefly observable in the urine, although the skin also may be slightly tinged.

Exposure to cold or sudden chill is popularly believed to be a cause of hepatic congestion, and one which will bear investigation. It is in the Tropics that "a chill upon the liver" is most frequently observed, and it is during two periods of the day that there are special dangers from this cause. One period is just as the sun is setting, when the earth rapidly cools, when the perspiration is suddenly arrested, and when the clothing, damp from perspiration, acts as a cold wet pack to the surface of the body, lowering the temperature, chilling the surface, and driving the blood from the skin to the thoracic and abdominal organs. At yet another period of the tropical day is "chill" apt to supervene —namely, in the early morning whilst yet in bed. The natural fall in temperature which occurs diurnally about 4 A.M. is a frequent source of abdominal trouble, and hyperæmia of the liver particularly. In great heat the bed-clothes are thrown off, or one may have retired without any covering. As morning approaches, however, the cold wakes the sleeper and he almost involuntarily covers the abdomen with a blanket, a sheet, a pillow, or whatever is at hand. The protection may have been applied in time and the effects of the chill prevented; but, on the other hand, diarrhœa or a congestion of the liver may result.

*The Effect of a Tropical Climate on the Liver.* —A young European when he first takes up residence in a warm climate finds that the continued heat acts as a stimulant. His physical energy for some months is increased, and his digestive organs enjoy an increased functional activity. The liver would seem to partake in the excitement, and the result is copious motions, deeply stained by the colouring matter of the bile. After a time, however, certainly within twelve months, the functional higher activity seems to become exhausted; in course of time the bile diminishes in quantity, until, judged by the pallor of the stools, it would appear that the liver action is imperfect. Other symptoms elucidate the same fact, and a form of dyspepsia supervenes, accompanied by epigastric fulness after meals and a sense of sinking when the stomach is empty. Buoyancy of spirits disappears, lethargy supervenes, and several minor ailments, such as occasional headaches, attacks of morning diarrhœa, etc., bring out the full meaning of the effects of "climate." In this state, if the liver is examined, it will be found increased in bulk. Percussion elicits a feeling of discomfort or actual pain, and all the signs and symptoms of hepatic congestion. In this state a chill, a wetting, wearing damp clothing, a cold bath, indulgence in alcohol, or exposure to the sun, may bring about a hepatitis.

Should, however, care in clothing and diet be exercised, combined with judicious medicinal treatment, the hepatic hyperæmia may subside, and a normal state of health be re-established. When recurrent attacks of congestion of the liver occur, owing largely to persistent departure from the hygienic necessities of a tropical climate, the liver will become permanently damaged. Chronic thickening of the capsule, perihepatitis, induration of the connective tissue of the liver, to a greater or less extent, will supervene, and true cirrhosis or hypertrophy may result. The liver in tropical climates, and in consequence of successive attacks of hyperæmia, may become permanently enlarged, even to a great extent; but, on the other hand, an atony or true atrophy with shrinking in bulk may ensue. It is by no means an uncommon—in fact, it may be styled a common—condition to meet with a small liver in old tropical residents. In many instances it is an atony rather than a true atrophy which occurs. In several intestinal lesions such as chronic diarrhœa, hill diarrhœa, and sprue, the liver is reduced in size to comparatively minute dimensions; and even independently of any marked ailment, shrinkage of the liver is to be met with. The cause of such a condition is frequently to be found in the reduction in diet incumbent upon persons inflicted with gastric or intestinal derangements. The quantity of food is reduced to a minimum, and the nature of the alimentation is frequently of a kind but little calculated to stimulate the liver to action. The effect is atony of the hepatic function, and if this be attended by intestinal flux, the portal system is kept permanently drained of its natural blood-supply and the organs of the chylopoietic system generally attenuated.

*Signs and Symptoms.*—In ACTIVE HYPERÆMIA

of the liver the patient complains of lassitude, loss of appetite, abdominal fulness, frontal headache, and inclination to lie down and sleep during the day, which is surely followed by sleepless nights or unrefreshing sleep. The tongue is furred, and the patient complains of a bad taste in the mouth. There may be nausea, retching, and vomiting at times of bilious matters; the feeling of sinking in the epigastrium is probably caused by an accompanying gastric catarrh; there may be an intestinal flux with bilious, acrid, and scalding stools, or the motion may be pale; constipation may succeed diarrhœa, or the constipated habit may persist from the first. Itching of the anus, especially if piles are present, and a moisture around the anus cause considerable annoyance; internal piles, if present, may enlarge and bleed. Jaundice to some extent is usual with yellow conjunctivæ, bile-colouring matter in the urine, and a greenish-yellow tinge of the skin. The urine tends to scantiness, and is dark in colour and loaded with urates. On percussion and palpation discomfort is elicited over the region of the liver, but there is seldom actual pain. In the right shoulder a "rheumatic" feeling may exist, and a definite tender spot on the posterior border of the scapula is not uncommon. The area of the liver dulness may be slightly increased, but is never markedly so unless the patient has been the subject of frequently recurring attacks of congestion. Lying on the left side is attended by discomfort and is unconsciously avoided. The patient, especially if the hyperæmia is alcoholic in origin, not infrequently states that "he feels the liver jump"; this peculiar symptom would appear to be caused by the descent of the diaphragm more sharply than usual on the tightly-distended liver capsule. The breathing is mostly thoracic and always shallow, and a "stomach" cough may occur spasmodically, especially on getting out of bed. Exercise is attended by marked shortness of breath, palpitation, and either increased pallor or purplish congestion of the face. Elevation of temperature is the exception, and when it does occur it seldom exceeds 100° F.

Tropical Liver.—*Signs and Symptoms.*—In the more common hyperæmia met with in persons who have resided in the Tropics, most of the general signs and symptoms above recounted are met with; but, in addition, there are a few that are characteristic of the condition. A history of malaria, of diarrhœa or dysentery, of long-continued dyspepsia, is usually present. Enlargement of the spleen is almost a constant accompaniment of the hepatic trouble. Anæmia and the peculiar skin tint, "the tropical mask," are always signs of long-continued liver congestion in the Tropics. Weakness, inability to take exercise, giddiness, and that condition of nervous irritability best expressed in the word "peppery" are very general concomitants. Oxalates may appear in the urine, which is pale in colour, of low specific gravity and containing urea in excess, and may alternate with dark-coloured, bile-stained urine containing urates. The action of the bowels is irregular; the stools may be frequent and inconstant as to the times they are passed, or constipation may prevail. Bile is usually absent from the stools, but at intervals it comes down in what seems an imperfect form, judging by the foul odour it engenders, and its irregular distribution in the fæces. The liver may be enlarged without much tenderness, or it may be small and shrunken, scarcely reaching the costal cartilages.

*Diagnosis.* — The "biliousness" associated with congested liver serves to direct attention to the source of the ailment. The increased size of the organ in all directions; the absence of, or but occasional evelation of, temperature; the local fulness, discomfort, or actual pain; the history of the case, especially as it relates to alcohol; the slight jaundice; and the general disturbance of the whole length of the alimentary tract, are usually sufficient to establish a correct diagnosis.

*Prognosis.*—An occasional congestion of the liver from indiscretion in diet, or from catching cold, is a condition which usually speedily yields to rational treatment. When the hyperæmia is due to alcohol, and recurs frequently and at short intervals, a more serious prognosis must be given unless alcohol is completely stopped. The recurrences betoken permanent changes in the connective tissue of the liver made especially manifest during subacute congestions of the organ.

B. Passive Congestion. — Under this heading several conditions differing in extent, in etiology, and in results, may be differentiated. The most marked of these are congestions arising from mechanical, from physiological, and from neurotic causes.

(1) *Mechanical obstruction*, or congestion, due to venous engorgement from obstruction, is a hepatic condition associated with impeded circulation of blood in the thorax. Cardiac disease, most frequently mitral regurgitation, is the genesis of the condition; but mitral stenosis, aortic obstruction or stenosis, emphysema, narrowing of the channel of blood-vessels by mediastinal tumours or by aneurysm, may each and all contribute. Displacement of the heart by fluid accumulations in the right or left pleura may cause the inferior vena cava to be so bent from its course that its lumen is encroached upon and its calibre diminished. In consequence, the exit of blood from the inferior vena cava is interfered with, and the venous return from the body everywhere below the diaphragm is hampered. Œdema of the lower extremities, piles, hæmorrhage from the bowel, ascites, fulness and hypertrophy of the liver and

spleen, and gastric disturbance ensue. The arrest in the freedom of exit of blood from the hepatic vein brings about alteration in the blood-current and in the tissue of the liver. To begin with, the sublobular and the interlobular veins—that is, the radicles of the hepatic vein—are engorged; the intralobular veins and the branches of the portal vein in time inevitably suffer from blood stasis. The liver thereby becomes swollen, its capsule is rendered tense, and a true chronic capsulitis, independent it may be of a perihepatitis, results. The connective tissue of the liver in time hardens, the nutrition of the liver-cells is so altered that a fatty infiltration or true fatty degeneration supervenes. Gastric and duodenal catarrh, giving rise to obstruction to the flow of bile, obtains, and some jaundice is the result. Examination of the liver in advanced cases shows that whilst its surface remains smooth, provided it is uncomplicated by syphilitic or other changes, the area of the liver dulness is increased in every direction, it may be to elephantine dimensions. The hepatic symptoms may become the most urgent of all the complications induced by thoracic venous obstruction, and may require attentive treatment.

The hypertrophy in such cases may temporarily subside, only to reappear in an aggravated form; or the hypertrophy may permanently disappear, and a contraction in size and true cirrhosis of the liver take its place. The diagnosis of hepatic hypertrophy due to retarded venous exit is usually sufficiently evident from the signs and symptoms.

*The prognosis* depends entirely on the curable or incurable nature of the affection which gives rise to the hepatic condition. The treatment is considered later.

(2) *Physiological passive congestion* is best exemplified by the hepatic condition associated with habitual constipation. The primary cause, however, of the constipation may be due to lessened functional activity of the liver itself, aggravated or caused by excess of food or insufficiency of muscular exercise, usually by both. Residence in cities, working in close and ill-ventilated apartments, more especially when the work entails a sitting posture, is a frequent cause of the trouble.

(3) What may be termed a *neurotic* form of *passive congestion* in the liver is witnessed in several paralytic states. In fracture of the lower part of the cervical spine or dorsal vertebræ, the thoracic and abdominal muscles are wholly or partially paralysed according to the seat of injury, and the movements of the chest wall are arrested or interfered with. The sympathetic nerves, more especially the splanchnics, may be involved in paralysis of either cerebral or peripheral origin, and the functional control of the liver disarranged. The dependence of the liver upon the nervous system for healthy action is illustrated in such conditions as sudden jaundice from fright, in the diabetic state induced by experimental research upon, and by disease of, the fourth ventricle. It is possible that the jaundiced state connected with influenza has its origin in a neurosis.

*Treatment.*—It is well at all times to treat active congestion of the liver seriously. The patient ought to be sent to bed and kept there until the jaundiced tint has worn off, and bile disappeared from the urine and reappeared in the stools. The diet should be reduced to a minimum in quantity, and given at three-hour intervals; it should consist chiefly of milk in some form. Plain milk is often repugnant to the patient at this time and may induce nausea or flatulence; whereas skimmed milk, soda or seltzer with milk, whey, junket or curds, are often well tolerated and easily digested. Calf's-foot jelly or any non-alcoholic jelly is agreeable and useful as a change. Chicken-tea freed of the fat, thin beef-tea, raw meat juice, raw meat sandwiches, tripe prepared in milk, etc., are a few safe forms of diet to select from. Some of the artificial foods, plasmon, Maggi, etc., are welcome as changes to a restricted diet, and agree well with patients suffering from hepatic and gastro-intestinal derangements. Alcohol must be denied altogether and the thirst satisfied by hot water; by weak tea, hot or cold, and with lemon in place of milk and sugar; lemon tea (not lemonade); rice tea (boiling water poured over roasted rice); toast-water, etc. When the biliousness disappears the diet is to be gradually increased; a speedy cure at this stage is often effected by a diet of finely minced beef, lightly cooked, and given in quantities of six ounces three times daily, with copious draughts of hot water between meals, and in the early morning and at bed-time. Intestinal catarrh is thereby reduced, and the function of the liver stimulated to act, as can be demonstrated by the quantity of bile in the stool.

*Medicinal treatment* is devoted to (1) allaying gastric or intestinal catarrh; (2) draining the liver by purgative medicines; (3) and restoring the hepatic functions, when acute symptoms have subsided, by the administration of hepatic stimulants.

During the acute stage of the disease, and when gastric irritation is a prominent condition, alkaline medicines are indicated. The bicarbonate of sodium (gr. xv.), iodide of sodium (gr. ij.), chloride of ammonium (gr. xx.), tinct. nucis vomicæ (♏ iij.), in a mixture with ʒj. inf. gentian, is a common and useful form of exhibiting alkalies, and should be taken immediately before food. Saline purgatives are to be given in the early morning. The sulphate of soda, in effervescing form by preference, in purgative doses in a small quantity of water, is the most satisfactory; it is an important constituent of Carlsbad, Hunyadi Janos, Friedrichshall, and

Marienbad purgative waters, any one of which may be employed instead of the plain salt. The sulphate of magnesia or any of the purgative soda or potassium preparations may be used. The popular remedies, seidlitz powder (pulv. tartarat. efferves.), Rochelle salts (soda tartarata), and cream of tartar (potas. tartras acida) are each and all efficient medicines. These saline preparations are invaluable in hepatic congestion. Their several actions may be classified as:—(1) Simply purgative—unloading the bowel and carrying off the bile. (2) Increasing peristalsis. (3) Hydragogue purgatives—draining the portal system of fluid. (4) Cholagogues—increasing the flow of bile by directly acting on the liver. (5) Alkalisers of the urine—a most important factor in counteracting the evils of the excess of uric acid in the blood and urine. (6) Prophylactic agents in the prevention of gout.

Mercurial preparations have been and are employed more extensively than any other drug in hepatic congestion. Blue pill, calomel, and pil. hydrar. subchloridi co. are the usual forms of exhibition. Mercurials are not direct cholagogues—that is, they do not directly stimulate the flow of bile; but by unloading the intestine, they carry off the bile acids by the bowel and necessitate functional activity on the part of the hepatic cells to supply the deficiency induced.

Cholagogues given in the stage of active congestion or inflammation of the liver can only do harm; their administration is an attempt to stimulate a diseased organ when rest is indicated. The chloride of ammonium in 20-grain doses is a drug of considerable value in hepatic ailments. It serves as a gastro-intestinal and hepatic stimulant, but it is also no mean diuretic; and whilst it actually increases the acid of the urine, it helps to free the system of urea and uric acid, a valuable element in treatment in such conditions of the liver. It possesses the advantage of retaining its activity in both alkaline and acid mixture, and may be advantageously exhibited with both. Cholagogues, when the acuteness of the illness has subsided, are of undoubted benefit. The liver excitement is followed by an inhibition of function which, unless overcome, will tend towards anæmia, dyspepsia, and debility. Rhubarb is one of the best of these; 3-grain doses of the rhubarb root, given thrice daily after meals, serves as a cholagogue and intestinal stimulant. It is better used thus than as a purgative. Direct cholagogues of vegetable origin and endowed with purgative or cathartic powers are such drugs as—podophyllin, aloes, scammony, jalap, colocynth, iridin, euonymin, and perhaps ipecacuanha. These are called for occasionally in liver derangements, especially of a chronic nature, but their habitual use is deleterious. Of the salts, the sulphates and phosphate of sodium are most distinctly direct cholagogues. Nitro-hydrochloric acid is one of the most favourite remedies in hepatic torpidity. Apart from its action as a sialagogue, and as a digestive adjuvant, the acid is a cholagogue of the first importance. Combined with strychnia, chloride of ammonium, and a vegetable tonic such as gentian, nitro-hydrochloric acid in 4 to 10 minim doses, given thrice daily in water, and given soon after, or sipped at, meals, is a cholagogue and digestive mixture of high value in torpidity of the liver, in the state of functional desuetude after congestion or hepatitis, and in cases of tropical liver. Nor is the action confined to the good gained by internal administration only. Sponging the body with an acidulated solution (8 fl. oz. of nitro-hydrochloric acid to 1 gallon of water), or employed as a foot bath or as a wet pack to the abdomen, are highly recommended as adjuncts to its internal use.

The acid bath at a temperature of 98° F. may be continued for half an hour morning and evening, or the wet pack may be kept on the abdomen for two hours, night and morning. Murchison directs that the same bath may be used on three or four successive occasions, a drachm or two of fresh nitro-hydrochloric acid being added on each occasion the bath is used to make up for the waste. For the bath, wooden or glazed earthenware should be used instead of metal baths, and all towels and sponges used in the bath should be wrung out, and kept in cold water to prevent the acid destroying their texture.

*Abstraction of Blood in Liver Derangements.*—In every form of active liver engorgement it is probable that abstraction of blood does good. Phlebotomy was extensively practised at one time, and the total neglect of this very rational mode of treatment is to be condemned. The writer has, in cases of hyperæmia and hepatitis, employed it with benefit. Ten to fifteen ounces of blood, drawn from the median basilic vein at the bend of the elbow, seems to be as efficacious as the smaller quantities usually drawn by local depletion. Eight or ten leeches applied over the region of the liver, and the bleeding further encouraged by hot fomentations, is also a potent remedy. A few leeches applied round the anus is perhaps more rational and physiologically correct, as thereby the portal system is presumed to be directly drained of its blood. Direct abstraction of blood from the liver by the hollow needle of an aspirator is by far the most effective and satisfactory method of blood-letting. For simple bilious congestion it is not required; but in hepatitis, in the acute congestion associated with alcoholism, and in congestions from mechanical causes associated with great hypertrophy, it is invaluable. The needle of the aspirator may have to be introduced into two, three, four, or more places before a vein

of sufficient size is struck to allow of a free flow. But even after several needle punctures are made and a small quantity of blood only is drawn into the bottle of the aspirator, a considerable amount of blood escapes from the punctured liver capsule into the cavity of the abdomen, and thereby effective blood-letting is obtained. The writer was enabled to prove the fact that blood does escape from the liver when perforated by the needle of an aspirator, as the following account shows:—A patient, the subject of mitral regurgitant disease, had an enormous hypertrophy of the liver and ascites. The ascites was so excessive that the fluid had to be withdrawn by paracentesis. Whilst the fluid was flowing through a cannula introduced between the umbilicus and pubes, the liver was punctured in several places by the needle of an aspirator, in the hopes of furthering diagnosis as to the presence or absence of pus in the liver. After five punctures had been made and the aspirator laid aside, the straw-coloured fluid issuing from the cannula became tinged with blood, the fluid became more and more sanguineous, until, as far as appearances went, it looked like pure blood. The cannula was withdrawn, the wound healed, and the patient professed himself much relieved, a relief which lasted for a week or two. Tapping the liver by an aspirator is discussed under Liver Abscess.

### Abscess of the Liver, Classification of

There is a tendency, amounting almost to a positive belief, to regard "abscess of the liver" as a definite disease. This clinical fallacy has hampered advance in our knowledge of the subject, and has no doubt proved a detriment to the application of more rational treatment.

1. *Classification.*—Abscesses of the liver are usually differentiated upon an *etiological* or *pathological* basis, and arranged into two great groups, the "tropical abscess of the liver" and "pyæmic abscesses of the liver." Even this attempt at classification has no true foundation; for tropical abscess may be pyæmic in origin, and with the tropical are associated abscesses occurring with, or following upon dysentery, the connection of which with a pyæmic infection of the liver is a matter largely of opinion.

2. Yet another basis of classification, brought forward by the writer, is the *anatomical*. Pus may collect above, within, or below the liver, and the writer has suggested the following terms:—

A. *Suprahepatic abscess* signifies a collection of pus between the layers of the broad (coronary) ligament of the liver, bounded above by the diaphragm, and below by the liver substance where it is destitute of peritoneal covering. The cause of pus collecting in this region is, according to the writer, a lymphangitis of the lymphatic vessels issuing from the upper surface of the liver and passing upwards towards the diaphragm to gain the thoracic duct. The disease follows quickly upon chill, and is independent of dysentery or other intestinal lesions. The pus met with in suprahepatic abscesses is not "liver" pus; it is usually sterile, and the amœba coli appears in it only after it has discharged for some days.

B. *Intrahepatic abscesses* include all abscesses, whatever their origin, occurring within the liver substance.

(1) Pus in the liver may develop, owing to inflammatory changes having their origin *within* the liver itself. Of these, hepatitis ending in suppuration (tropical abscess) is one of the best known; but pus may result from the damage caused to the liver by a blow; a hydatid or other cyst may suppurate; or parasites may be the foci and causes of purulent formations. It is evident also that inflammatory hepatic lesions caused by gall-stones, pylephlebitis, etc., may belong to this group or to the following. (2) Pus in the liver may, however, arise from causes *beyond* the liver, but *within the gathering area of the portal vein.* The suppuration is of a truly pyæmic origin and character, and may have as its origin ulcers (dysenteric, malignant, or simple) anywhere in the alimentary tract from the stomach to the rectum, appendicitis, disease of, or surgical operations upon, any part of the viscera or parts of the abdomen drained by the portal vein. (3) Pus in the liver may result from a general pyæmic state, in which case the infection is conveyed by the hepatic artery. *Pyæmic abscesses of the liver* are usually multiple. They seldom attain a large size, although one may far outstrip the others in dimensions. A cause beyond the liver area is always discoverable either during life or after death. Dysenteric ulceration is a common lesion, in which instance the "tropical" abscess may be merely an example of pyæmic infection. The signs and symptoms of multiple abscesses of the liver developing in the course of a general pyæmia are obscure in the extreme. The local conditions and the train of clinical evidence involved are but of secondary importance to the general state of the patient. There may be local pain, perihepatitis or general peritonitis; and purulent collections, if near the surface, may even be felt through the abdominal wall.

The pathological factor is, no doubt, an embolism of the hepatic radicles of the portal vein. The blood stasis induced leads to degeneration of tissue and the formation of localised suppurations, each of which is surrounded by an area of congested and hyperæmic liver tissue.

Surgical treatment is seldom called for in the pyæmic state; but should any one of the abscesses approach the surface, or give other evidence of its presence, the pus may be

dealt with as in a case of tropical abscess (see p. 511).

C. *Subhepatic Abscess.* — A few cases have been recorded by the writer and others of pus forming between the under surface of the liver and the peritoneum, and pointing in the epigastrium. The liver tissue is exposed but not involved, and the pus seems to be caused by a lymphangitis in the subhepatic region.

TROPICAL ABSCESS OF THE LIVER


NOMENCLATURE . . . . . 502
VARIETIES OF HEPATIC ABSCESS . . 502
INTRAHEPATIC TROPICAL ABSCESS . . 502
*Etiology* . . . . . 502
*Clinical History* . . . . 505
*Local Signs and Symptoms* . . 505
*Effect on other Viscera* . . . 506
*Constitutional Symptoms* . . . 507
*Diagnosis* . . . . . 508
*Prognosis* . . . . . 509
*Mortality* . . . . . 510
*Treatment* . . . . . 510


NOMENCLATURE.—The term tropical abscess may be, and probably is, a misnomer. It can in no sense be defined as a specific ailment; but even if there is such a disease as a specific abscess of the liver, dependent upon tropical environment, it is so obscured by hepatic abscess due to other causes that the exact limitations are impossible to determine. At the present time a "tropical abscess" is considered to be (1) a single collection of pus on the liver, (2) met with in warm climates, and (3) associated with, or caused by, dysentery. This description is in no sense a definition, for every one of the three points mentioned may be and has been contested, and in many instances refuted. However, it is a collection of pus answering to this description which serves as the basis for discussion under the heading Tropical Hepatic Abscess.

VARIETIES OF (TROPICAL) HEPATIC ABSCESS.—The writer has from time to time sought to differentiate between collections of pus met with in and around the liver, and to bestow definite names to the abscesses according to their position, and to some extent also upon their cause and pathology.

A classification into suprahepatic, intrahepatic, and infra- (or sub-) hepatic abscesses is in accordance with anatomical fact; but the writer has insisted that the first two of the group, and it may be all, arise from different causes.

INTRAHEPATIC TROPICAL ABSCESS. — It may seem unnecessary to designate the ordinary form of liver abscess by a qualification such as "intra," and many may dismiss the term with the criticism that an abscess of an organ must be within that organ, and therefore the term "intra" is supererogatory. This is an unanswerable argument; but although theoretically correct, it is far from being practically so, for abscesses are termed hepatic which, whilst occupying the dome of the liver, involve the liver, if at all, only to a slight extent. The latter are to be subsequently described under the heading suprahepatic, and their pathology separately dealt with.

*Etiology.*—It is impossible to make a definite statement as to the cause of a tropical abscess within the liver substance; but there is sufficient clinical and pathological evidence to hand to justify dysentery being considered a potent and all-important factor in the etiology.

*Dysentery* as a cause of liver abscess has the weight of long experience, and the investigations of modern pathology as testimony. It is the endeavour of many observers to attribute all cases of tropical abscess to infection from a dysenteric ulcer; whilst, on the other hand, dysentery as a factor in the etiology of tropical abscess is scarcely recognised by others. This divergence in opinion is to be attributed to the fact that every collection of pus in the hepatic area is usually termed a hepatic abscess without reference as to whether it be of the nature of a suprahepatic or an intrahepatic abscess.

The fault lies in the nomenclature; for, by use of a common term for all hepatic purulent collections, two or more conditions differing in their course and pathology are grouped together unjustifiably. A marked feature in differentiation, it may be stated with confidence, is the part played by dysentery in the etiology.

In suprahepatic abscess dysentery plays, perhaps, no part as an antecedent, and, except as an occasional concomitant, is seldom met with in the history of the case. Intrahepatic abscesses are possibly always subsequent to dysenteric ulceration of the intestine.

Intrahepatic abscess is the result, according to this statement, of infection, pyæmic in nature, from the surface of the eroded and ulcerated bowel.

*The Nature of the Infection.*—When inflammatory lesions ending in suppuration develop anywhere in the gathering area of the portal vein, there is a possibility of purulent collections appearing in the liver. Multiple abscesses from this cause, reasoning *a priori*, would seem the inevitable result, and clinical experience bears out the theoretical suggestion. Were this the result of dysenteric lesions the matter would be simple enough; but it is believed the simple (tropical) abscess is the variety associated with dysenteric changes. Other ulcerations, however, of the bowel, such as those met with in typhoid fever, seldom give rise to suppuration within the liver; and ulcerative changes in the stomach, appendicitis, carcinoma of the bowel, etc., when they infect the liver, do so at a number of foci; and it is considered the peculiarity of the dysenteric gut to give rise to a single abscess. How

far this belief is justified is open to doubt, or at least to discussion.

The infective material can only be conveyed by way of the portal vein, and it is assumed, and in many instances it has been proved, that the etiological factor in liver abscess resulting from dysentery is of the nature of emboli, carried from the surface of the ulcerated bowel to be arrested in the radicles of the divisions of the portal vein within the liver. Dr. Neil Macleod has explained the tendency of dysenteric lesions, in contradistinction to the open surfaces of typhoid or other intestinal ulceration, to set up liver abscess. He contends that in dysentery the purulent material burrows beneath the mucous membrane; that it is apt to be confined in the recesses, and by the undestroyed bands of tissue, and thus retained in contact with open radicles of the intestinal veins. This explanation would seem to merit consideration; yet it cannot in any sense be considered as the complete solution of the phenomena, for it is a fact that dysentery, although even more prevalent amongst natives than amongst Europeans in India and elsewhere, is seldom followed by liver abscesses in the case of the native. Further, it does not in any way solve the question of single and multiple abscesses unless the formation of one or other of these is merely a question of coincidence or accident—a view which is by no means improbable. So puzzling is the question of intestinal ulceration and liver abscesses that more than one observer has sought to explain the association by adducing a chemical agency as the mode of infection of the liver.

It is possible that the dysenteric ulcer is associated with a specific toxic agency—bacterial, chemical, or embolic—which, when it reaches the liver, tends to establish a focus or foci of suppuration, and that the question of singleness or multiplicity is for the most part accidental.

*Abscess of the Liver independently of Dysentery.*—Tropical abscess of the liver is by many considered to be independent of dysenteric lesions in the bowel; and the generality of statistical records, although they show some 50, 60, or even 80 per cent in several computations, in no instance claim that all cases of tropical abscess are to be assigned to dysentery. The opponents of the direct connection between the two phenomena assert that dysentery is an occasional concomitant of liver abscess, but that in a large proportion of cases, as proved by post-mortem records, no dysenteric lesion, old or fresh, is met with. The explanation of this absence of post-mortem evidence is ascribed, by the upholders of the potency of dysentery in the lesion, to imperfect observation. This is a view which cannot be upheld in every instance, and the explanation probably lies in the confusion due to the want of differentiation of liver abscesses. As stated above, suprahepatic abscesses are not caused by, although they may be accompanied by (or provoke), dysentery; but pathological records would seem to point to the conclusion that intrahepatic abscesses are dysenteric in their origin.

The *amœba coli* in relation to dysentery is also a question of dispute and individual opinion. The discovery of so-called amœbic dysentery was, by the believers of the potency for evil of this infusorian, supposed to afford a solution also of liver abscess in connection therewith. Amœbic dysentery as a distinct disease is, however, not established on a secure basis. The very presence of the amœba coli is now known not to bear a pathological significance, for it is a resident in the healthy gut. The normal presence of the amœba in other organs has not been fully investigated; but in many abdominal lesions the amœba finds its way thither, perhaps as a consequence of the pathological change rather than as a factor in the origin of the changes. That this is the case in liver pus has been observed by Dr. Manson, and the writer can confirm his statement. The amœba is not found in the pus flowing from the liver for the first two or three days after operation, but appears with well-nigh regularity on the third or fourth day. Dr. Manson believes that the absence of the amœba in the first liver pus that escapes is due to the fact that the infusorian dies amongst the dead central pus, and is only to be met with when the pus recently formed from the walls of the abscess finds ready escape, or in scrapings from the walls of the abscess cavity itself. The writer is inclined to believe that the amœba plays no part in the changes which lead up to an abscess, and it is only when the abscess is tapped and flowing that the amœba travels towards the region.

It has been suggested that the amœba itself is not a pathological factor in hepatic suppuration, but that it carries in its substance bacterial or other toxic agencies which lead to the establishment of suppurative changes. This is a possible, even a probable, mode of infection; but it is unsubstantiated and speculative, and implies the presence of the amœba in liver abscesses *ab initio*—an assumption which is not corroborated by fact.

*Liver Pus.*—The pus drawn off from a liver abscess is unlike that met with in any other part of the body. The first gush of pus from the centre of an abscess resembles ordinary pus more closely than the subsequent issue, but its appearance from the very first is well-nigh characteristic. A rather sickly, disagreeable, mawkish odour pervades both the initial and subsequent issues of the pus. At first the discharge is of a fairly uniform consistence, dirty white in tinge, and streaked with bile-colouring matters or with blood. As it flows a serosanguinolent appearance is communicated to the pus, the colour becoming more and more sanious.

When allowed to stand in a glass vessel the abscess contents are seen to separate into a maroon-coloured deposit with a supernatant fluid of a lighter hue. In consistence, after the first few hours of drainage, liver pus is peculiarly glairy and gelatinous. It is scarcely absorbed by cotton-wool, gauze, or lint dressings; it tends to escape or lie between the skin and the dressings as a sticky, mucoid mass. The pus as it passes along a syphon drainage-tube, into the length of which a piece of glass tubing has been inserted, is seen to consist of yellowish-white masses of pus, almost the size of a split pea or lentil, floating in a lightly-coloured sero-sanguinolent fluid.

The discharge in a favourable case contains less and less pus, the fluid becomes less red, and in time a biliary green colour is communicated to the fluid, proclaiming the fact that the abscess has healed and that the drainage-tube may be gradually withdrawn.

*Microscopical examination of liver pus* shows it to consist of pus corpuscles, of leucocytes, of large pigmented globular cells, in various stages of perfection or degeneration, according to the stage of the abscess, and as to whether the material under examination is derived from the centre of the cavity or from the walls of the abscess. Red blood corpuscles, hæmatoidin crystals, oil globules, and particles of degenerate liver tissue are fairly constant ingredients of liver pus; and cholesterine and Charcot-Leyden crystals are occasionally present. The presence of micro-organisms opens up the question of the sterility of liver pus. It was remarked above that the amœba coli was found, if at all, on the third day after the commencement of the flow of pus from a liver abscess. The writer has never found the amœba during the early days, and furthermore, has not always found it in the later days.

*Hepatitis and Liver Abscess.*—In warm climates acute (and chronic) hepatitis is more commonly met with than in temperature zones. Under "Hepatitis" this subject was fully discussed, and it would appear that hepatitis may play a part, a significant part, in the etiology of liver abscess. It has long been recognised that amongst Europeans dwelling in the Tropics the liver is the viscus upon which the greatest calls are made. It is too often asserted that excess of food and drink and deficiency of exercise are the explanations of the frequency of hepatitis and liver abscess in European dwellers in tropical as compared with residents in temperate climates. If it is true that Europeans in the Tropics thus indulge, still it remains the fact that persons living in temperate climates who habitually over eat and drink are scarcely affected by either hepatitis or liver abscess. Climate therefore, or some local conditions, must determine the incidence of the hepatic lesions. The native of the Tropics becomes acclimatised—that is, has been taught by experience to suit his habits in eating, drinking, and clothing to his environments. Not so the European immigrant; in none of these does he follow implicitly the teaching of native experience, and he suffers, no doubt, in consequence. The European, more especially in his clothing, in his unwise want of precautions against chills, and in a number of other ways exposes the surface of the body to alternate over-heating and over-cooling, which must call upon some organ to deal with. Experience, and to some degree only physiology, has shown that it is the liver that is mostly taxed, or that at any rate gives out physiologically. The sudden chill after violent exercise, whether it be the marching of soldiers in the sun, or the voluntary exercises of sport, causes a determination of blood to the internal organs, and the liver becomes congested, hyperæmic, or inflamed, according as the recurrences of chill are occasional or continued.

It would appear, however, that there is some determining cause beyond hepatic hyperæmia, for in certain tropical regions liver abscesses are scarcely known, even amongst Europeans. This is especially reported to be the case in the West Indies; but when the matter is inquired into, the region in which liver abscesses are "conspicuous by their absence" is much wider. Within the Tropics the meteorological conditions are very dissimilar. Heat is only one factor of a tropical climate; the cold at nights is seldom considered, yet is it the fluctuations in temperature that test the physical powers. In the equatorial belt—that is, the regions extending to 12° north and south of the equator—night chills are unwonted occurrences. The climate of the equatorial belt is as different from that immediately to the north and south of it, and yet within the Tropics, as is that of London from St. Petersburg or New York. It is the area of equable temperature and almost perpetual calm; winter and summer are mere names; the temperatures of the night and day vary but a few degrees; chill is uncommon therefore, the body being exposed to a fairly equable heat. In the whole of the equatorial belt, liver abscess is almost as rare as in temperate climates, and one is driven to the conclusion that the absence of the sudden changes from day to night temperatures in these regions, and its presence where these conditions prevail, may have a potent influence in determining the ailment.

As with the amœba so with other bacteria—neither streptococci nor staphylococci being demonstrable in liver pus during the first days of flow. It is to the pus of this character that the term sterile has been applied, and the phenomenon is certainly peculiar. The writer is inclined to the view that liver abscesses arising from metastatic or pyæmic infection will be

found to contain bacteria in every case; but that idiopathic or true tropical abscesses, the result of hepatitis, may not show bacteria in their contents until after the pus finds exit from the liver. But the formation of pus independently of bacterial infection would transgress the basis of our pathological knowledge. But liver pus is often wholly unlike pus from other parts of the body; in many instances it scarcely can be classed as pus at all. The discharge more often resembles necrosed tissue, diffluent liver tissue consisting of broken-down liver-cells and tissue floating in a sero-sanguinolent fluid. It is possible that the cutting off of the blood-supply to a localised area of the liver, due to congestion or hyperæmia of the liver itself, may give rise to this necrotic patch, and that bacterial infection may follow upon, instead of preceding, the pathological change. Be this as it may, it is a fact that many observers have substantiated the statement that liver pus issuing from a recently tapped abscess is frequently sterile for the most part.

*Malaria and Hepatic Abscess.*—There is but little to justify the belief that malarial poisoning has any direct influence in causing liver abscess. Although in many instances the two factors coexist, yet are they by no means coterminous in their geographical distribution. Moreover, the changes induced by malaria in the viscera generally are more of a cirrhotic or plastic nature than of a destructive or retrograde character. Malarial pigmentation, the result of blood degeneration by the action of the plasmodium, is not calculated to lead to necrosis of tissues, although it is true the cells in the walls of the hepatic capillaries are so filled with pigment as to obstruct or actually block the lumen of the vessel. Malaria in all probability serves merely to weaken the functional powers of the liver, to thereby render it more susceptible to disease and less fit to resist the influences brought about by chill.

The *clinical history* of an individual case of liver (intrahepatic) abscess may present divergent points of detail and interest, but there are a number of phenomena pretty generally met with when a series of cases are studied. A European, on arrival in the Tropics, generally finds that the continued heat acts as a stimulant, and in mental and physical vigour he is at his best. After it may be twelve months the digestive organs may show symptoms of impairment, and hepatic derangements are apt to give rise to trouble. These may consist of digestive upset merely, or the liver may become actively congested or hyperæmic. Hepatitis, with the usual feverishness and local right hypochondriac discomfort or pain, may ensue, accompanied by an intestinal flux or dysentery. The liver will be found enlarged, tender to percussion and palpation; and the patient will exhibit all the signs and symptoms peculiar to hepatitis (see "Hepatitis"). The end of the attack will be either resolution and recovery, or the inflammation may end in suppuration. In another group of cases the patient may be attacked by dysentery, which, after running the usual course, may end in recovery so far as the dysenteric symptoms are concerned. But he does not regain his strength properly; the appetite is not good; work is a trouble, owing to fatigue and languor; he has sleepless nights, and suffers from headaches, backaches, nausea at times, depression, and irritability of temper. Attacks of fever at irregular intervals, or towards evening, compel him to lie down or seek his bed; and after being asleep for some time he wakes up bathed in perspiration. Sooner or later he becomes conscious of a sense of fulness in the right hypochondrium, associated with occasional twinges of pain which become more and more pronounced and constant. The evidence of hepatic trouble is now so pronounced that the attention of the medical practitioner is drawn to the liver, when the probability of suppuration having taken place will be suggested.

*The Local Signs and Symptoms of Hepatic Abscess.*—When an abscess of fair size occupies one lobe of the liver, *percussion* will elicit the fact that there is a general enlargement of one lobe, or that the outline of the liver dulness at one aspect of the organ is altered. The abscess may, however, be so small and so deeply seated that neither by palpation nor by percussion can the liver be made out to exceed the normal limits. On the other hand, when the abscess is large, one lobe may be palpably increased in size, or an increase in dulness in one direction is evident. The direction in which the dulness exists will depend on the direction the abscess takes; for it may advance towards the upper or under aspects of the liver, or it may bulge towards the abdominal wall or laterally towards the right wall of the chest. The abscess, should it advance towards the anterior wall of the abdomen, may be *felt* as a uniform, rounded tumour, smooth in outline, and yielding a sense of tension or of fluctuation, according as the abscess is fairly deep in the liver tissues or as it occupies the surface. The abscess may bulge to such an extent that its presence is obvious to *inspection*, and a distinct prominence is discernible somewhere in the epigastrium or in the right hypochondrium. If easily palpated, it may be possible to recognise that the parts around the abscess are hard and indurated owing to inflammatory thickening of the liver. As the abscess approaches the abdominal wall *peritoneal friction* will be heard. At first the friction sounds correspond to the apex of the tumour only, but as the abscess increases in size and still further encroaches upon the abdominal wall, the area of friction will widen in a circular manner. With the widened area, however, it will be found that in the central part friction sounds disappear, due

either to adhesions having taken place or to an effusion of lymph between the opposite peritoneal surfaces. If the abscess is allowed to advance without operation the friction area gradually extends, and the central non-frictional patch increases *pari passu* until it attains a diameter of several inches. This observation is of great practical importance, for it indicates the exact locality where the abscess may be opened or tapped with safety. The writer has availed himself of this observation in all operations for hepatic abscesses which develop forwards; and he has been able to prove, by post-mortem observations, that it is safe to cut into or to push a trocar and cannula into the central non-frictional area without fear of peritoneal extravasation even when no adhesions had formed, but when merely flocculent and unorganised lymph existed.

A liver abscess, however, of considerable size may be present deep in the parenchyma of the liver, and give rise to no evidence of a tumour, nor of perihepatitis, nor any other sign of local hepatic trouble whatever. In such cases the constitutional symptoms may alone serve to suggest the presence of pus in the liver.

*Pain.*—In the early stages, when suppuration develops after hepatitis, the pain peculiar to hepatic inflammation is to be expected; but as the abscess develops the hepatitis subsides, and pain may practically completely disappear. There may be a couple of pints of pus present in the liver, and the patient suffer no pain therefrom. This disappearance of pain often leads the doctor astray, and the patient, being free of the pain (and it may be the feverishness), refuses to be operated upon. This is one of the commonest sources of fallacy in the treatment of liver abscess, and the possibility of the occurrence should never be lost sight of.

Pain, however, may never have formed any part in the initial symptoms. In a liver abscess arising some months subsequent to an attack of dysentery, the suppuration, if deeply seated, may proceed independently of hepatitis, and without pain. The pain when present may be acute, or it may amount to discomfort merely, the variation in severity depending upon the part of the liver which has suppurated. The nearer the pus is to the surface of the organ the greater will the pain be, and *vice versâ*. The parenchyma of the liver is scarcely, if at all, susceptible to pain—a fact which is well illustrated when aspirating the liver without an anæsthetic. When the needle of the aspirator reaches the liver tissue in such circumstances, the organ can be traversed in various directions without eliciting any degree of pain; and it is only when the cutaneous structures or when the diaphragm is punctured that acute pain is complained of. It is perihepatitis, and not hepatitis, which causes pain in the region of the liver.

It is plain, therefore, that pain may usher in hepatic suppuration, may then completely sub side, only to appear again when the abscess advances to the surface of the liver, causing perihepatitis; on the other hand, pain may not play a part in the original onset of the disease, and may be deferred until the surroundings of the liver are affected.

Pain when present may amount to mere discomfort, felt mostly when the patient turns upon the left side — the increased weight of the liver causing dragging pains in the right hypochondrium. The heart's action also is impeded by the same posture. When more acute, the pain may give rise to a stabbing feeling in the intercostal spaces overlying the liver. The right shoulder may be, and very frequently is, the seat of a constant aching and irritating pain. The situation of the shoulder pain varies with the part of the environment of the liver involved; it is chiefly when the diaphragm, and consequently the phrenic nerves, are irritated, that the pain is felt in the shoulder. The explanation of the pain is found in the connection between the radicles of the phrenic nerve in the neck and the descending branches of the superficial cervical plexus. When the liver is merely congested or "heavy" with pus, the sensation felt in the shoulder is usually of a dragging nature and may be mistaken for "rheumatic" pains. The vertebral border of the scapula is then the part complained of; but when subdiaphragmatic perihepatitis develops, the pain is often of a burning character, and situated just within the angle formed between the clavicle and the acromion process of the scapula. The patient may be able to exactly localise the pain, and can place the finger on the precise spot; but occasionally the pain is diffuse, and its limits are conveyed by the patient placing the whole hand on the top of the shoulder.

The presence or absence of pain in the shoulder is no aid to diagnosis of the presence or absence of pus in the liver. It merely indicates a perihepatitis; but the cause of the lesion may be other than a purulent collection.

*The Effect upon other Viscera.*—An abscess in the liver may involve neighbouring viscera and structures. The particular issues affected depend upon the course the pus follows. When the pus burrows towards the dome of the liver the diaphragm and the structures adjacent on its thoracic aspect will be implicated. *Pleurisy* is common, and the friction sounds of the inflamed pleura will obscure the friction sounds due to perihepatitis. The two layers of the basal pleura will be cemented by lymph, and the adjacent surface of the lung sympathises in the inflammatory change. The *lung* at its base becomes congested or pneumonic. These changes are due in part to the arrest in thoracic movement over the seat of the inflammation and pain. Abdominal breathing gives place

to thoracic, and the lower ribs are maintained in as immobile a state as possible. The consequence is that the blood in the lower part of the right lung is apt to sag, and the adhesion to the diaphragm and the inflammatory changes which affect the base of the lung all combine to favour consolidation. A right basal congestion is a common clinical experience in medical practice in the Tropics; it may merely indicate a stagnation of blood in the region of the lung immediately over a congested or hyperæmic liver, or it may point to a real inflammatory state of the lung due to hepatitis or perihepatitis, and the subphrenic formation of pus.

When the *stomach* is encroached upon, gastric irritation will be in evidence, with vomiting, nausea, and inability to retain nourishment. The *colon*, although a hepatic abscess frequently finds its way thither, seldom shows marked evidence of physiological change. Just before perforation in the colon takes place constipation usually obtains, and then distension of the coecum and ascending colon from gas. The opposite condition, however, may prevail, and an irritating flux characterise the action of the bowel. The *gall-bladder* is seldom encroached upon, and it is rare for jaundice, due to implication of the biliary ducts, to be met with. A hepatic abscess advancing downwards may press upon the main bile duct, or the bile passages may, owing to duodenal catarrh, become obstructed, and so cause jaundice. The spleen in liver abscess, or in the hepatitis which usually precedes it, may be said to be always small. The reason for this is to be found in the fact that the portal circulation is in no way interfered with by a tropical abscess in the liver. When the spleen is enlarged in inflammatory liver lesions it points to a pylephlebitis as the cause of the disorder. For the same reason *ascites* and congestion of the veins below the diaphragm are pronounced by their rarity in inflammatory affections and suppuration of the liver itself.

The right rectus muscle of the abdominal wall is usually maintained in a rigid condition. The muscle assumes this position as part of the general effort to ensure freedom from movement in the neighbourhood of the inflamed and painful organ. When examination of the liver by percussion or palpation is attempted the rectus muscle is thrown into a still more firm state of spasm, and may be felt as hard as a board in its attempts to guard the inflamed organ beneath. In this direction also the involuntary protective impulse is so marked that the patient inclines the whole trunk towards the right side, and should he assume the erect position the body is seen to incline to the right and forwards.

*Constitutional Symptoms.*—The presence of a liver abscess may come to be recognised only by a gradual process of exclusion and by no pronounced initial symptoms. At other times a definite period of commencement is ascertainable and can be readily determined. When suppuration follows upon a hepatitis a *rigor* usually ushers in the development of pus; but when the abscess arises subsequently to dysentery the period of invasion is more indefinite, and a collection of pus may be present unknown to patient or doctor.

*Sweating at night* is a marked symptom in suppurative hepatitis, and as the illness develops the sweating becomes profuse, appearing on every occasion on which the patient goes to sleep during the daytime; and the patient may have to get up, especially in pyæmic abscesses of the liver, not once, but two or three times in the night, to change his garments owing to their being saturated with sweat. At all times, except during the actual period during which the rigor threatens or prevails, the skin is moist and clammy.

The *temperature* of the body in liver abscess does not always run a typical course. As a rule, after pus has been developed, and has been present in the liver for some time, a morning drop in temperature and an evening exacerbation, with or without a rigor, is the rule. The matinal temperature may fall below the normal considerably, 96° F. or even lower being registered; the evening temperature may rise to 100° F. only, or it may reach 101° to 104° F.

When the abscess develops gradually and insidiously, as is the case in most intrahepatic abscesses, following upon old dysenteric troubles, the initial rise of temperature is very gradually developed; but when the abscess is acute, following upon acute hepatitis, or when the abscess is suprahepatic, the presence of pus is usually ushered in by a temperature ranging from 103° to 105° F. This high register may be maintained for several days, but afterwards the temperature fluctuates and the accessions of fever are irregular.

In all cases, however, when the pus has accumulated in quantity and hectic is not present, the tendency is for the temperature to fall to about the normal. Many cases of established liver abscess, in which a pint or two of pus is present, are attended by no abnormality of temperature, the thermometer showing a normal temperature morning and evening.

When hectic follows upon operation, the usual accession of temperature as the day advances becomes pronounced.

*The Alimentary Canal.*—The *tongue* in the earlier stages of hepatic suppuration remains wonderfully clean. It is usually somewhat reddened, and swollen; the furrows and rugæ are preternaturally prominent, and at the back a velvety coat of a yellowish tinge persists. It is in no sense the tongue of "biliousness," and it is remarkable how small a part "bilious"

symptoms play in hepatic abscess. As the disease advances the tongue becomes dry and abnormally red, and even bare; aphthæ and sordes in the teeth are a late sign, and usually indicate a fatal issue. *Vomiting* in the early part of the disease is a common symptom; but, as the general hepatitis or congestion subsides and the abscess develops, vomiting is not a marked feature. Should, however, the abscess advance towards and affect the walls of the stomach or duodenum, vomiting in an aggravated form will recur and continue until the pus finds its way into either viscus. Dysentery, constipation, or actual diarrhœa may prevail throughout or alternate during the course of a liver abscess. When the abscess is more or less "latent," constipation is the rule; when hepatic congestion obtains, diarrhœa will probably ensue; but should hepatitis occur in conjunction with or develop at any period of the course of a liver abscess, dysentery (blood and mucus) may result—the condition of the bowel depending altogether upon the state of the liver. An intestinal flux with blood and mucus may or may not be dignified by the name of dysentery. We are apt to regard the presence of blood and mucus in the stools as indications of a specific disease—namely, dysentery; but it is possible that mere portal congestion, with changes in the radicles of the portal vein or the intestines, may cause a flux of blood and mucus of a temporary nature and of a non-specific character. Certain it is that, with the presence of pus in the liver, a flux of the kind, and lasting for a few days only, is a common feature. Dysentery as a specific ailment runs a definite course; but an intestinal flux, resembling dysenteric motions, may come and go, during the presence of pus in the liver, in a somewhat erratic fashion totally unlike true dysentery.

*The Urine.*—Urates and uric acid are well-nigh invariably deposited when the urine cools. The urine is high-coloured or dark, and usually somewhat scanty when an abscess is developing; but when the pus gives rise to no feverish symptoms the urine becomes fairly normal. The quantity of urea varies; one day it is in excess, the following day it may be normal in quantity or considerably diminished. As a rule, when the patient's temperature is up, the urea is in excess—that is to say, whilst the hepatic parenchyma is irritated and its function exalted the urea is in excess; the opposite condition, however, naturally obtains when a large portion of the liver is rendered functionless by the presence of pus.

Albumin in the urine is by no means a common feature in liver abscess, but occasionally traces of albumin are found.

*The Effect upon the Liver itself.*—When one part of the liver is rendered functionless or destroyed, the remaining portion becomes hypertrophied, and takes up the work of the diseased portion. The right lobe of the liver may be reduced to a mere shell, yet may the functional activity of the liver continue, and in course of time return to full working order. The writer has brought this fact prominently forward on several occasions. As an instance of the compensatory possibilities of the healthy, in regard to the diseased portion of the liver, the following example will suffice:—At a post-mortem examination of a Chinese prisoner who committed suicide in the gaol in Hong-Kong, the writer found the right half of the liver reduced to a small mass of fibrous tissue. A closer examination showed that the fibrous mass was continuous with a tissue of like nature, which extended upwards through the diaphragm and into the right lung. In other words, that a right liver abscess had discharged upwards through the lung, that the pus had been expectorated, and that the whole tract had healed. On weighing what remained of this liver, it was found that it scaled within an ounce or two of the normal weight of the liver. The left lobe, the lobulus spigellii, the lobulus quadratus, and the lobulus caudatus were enormously hypertrophied—all the parts, in fact, to which the left branches of the portal vein and hepatic artery are distributed. By subsequent observations, by experimental injections, etc., the writer has been able to prove that there are two *sides* to the liver, each equal in bulk, and each half presided over by an equal-sized branch of the portal vein, hepatic artery, and hepatic duct. In fact there is a right and left liver in juxtaposition, equal in weight, but having separate vascular and biliary circulation. Disease of one side of the liver need not, and very seldom does, involve the other; and, moreover, when one side is destroyed the other may take up its functions. There is nothing unique in this, for we have the example of one kidney executing the work of both when one is removed. A liver, therefore, in which disease is confined to one side may come in time to fulfil the function of the whole; and given time to develop, the compensatory power is so marked that a person may recover completely with only half a liver left.

*Diagnosis.*—It is plain from the account of the signs and symptoms of liver abscess that there is no one feature which is permanent. There may be no pain, cough, temperature, or hepatic enlargement to indicate that a collection of pus—it may be of a fair size—occupies the hepatic area. On the other hand, all these may be present and yet the signs and symptoms may be due to other causes than an abscess. *Hydatid* of the liver, in countries where hydatids prevail, is the most likely ailment to be mistaken for liver abscess. There are typically several distinguishing features: pain is rare in hydatids, common in liver abscess; increase of temperature is the rule in abscess,

the exception in hydatids; a history of hepatitis, dysentery, and hectic are frequent concomitants of abscess; hydatids, on the other hand, unless when they proceed to suppuration, show none of these. But an abscess may be *latent* and, if large, may give rise to the presence of a painless tumour with fluctuation, without any other local or constitutional disturbance. On the other hand, nothing is more common than for a tropical resident to show signs of failing health without any definite diagnosable lesion. Loss of weight and appetite, want of mental and physical energy, evanescent attacks of fever, occasional intestinal disturbances may persist, and yet it is impossible to ascribe the signs and symptoms to any particular organ. More often than not "climate" is assigned as the cause, and the patient is advised to go home—that is, to return to Europe. During the voyage home, especially when colder latitudes are reached, or after being at home a year or more, symptoms of liver abscess develop which in all probability was present but "latent" before the patient left the Tropics. Most of the single liver abscesses met with, in Britain at all events, occur in persons who have returned from the Tropics—not necessarily recently, for a liver abscess may appear years after leaving warm climates. That the abscess was in the liver before leaving the Tropics is, of course, not positively known; but the writer has had experience of patients whom he has sent home with the indefinite symptoms above recorded and in whom a liver abscess developed after reaching England, and the conclusion that undiagnosed pus existed in the liver before the patient left the Tropics would seem feasible and probable.

Of the conditions and ailments likely to be mistaken for liver abscess are—pylephlebitis, pyelitis of the right kidney, pneumonia of the right base, hepatitis, hyperæmia, congestion or enlargement of the liver due to cardiac or pulmonary lesions, gall-stones or inflammation of the gall-bladder, subphrenic abscess. These can only be eliminated by careful clinical observation and a study of the history in each case. Malaria in its symptoms resembles in some measure those due to pus in the liver, and here again clinical records are of the utmost value. In liver abscess the spleen is seldom enlarged; there may be no malarial parasites in the blood; the maximum rise in temperature takes place towards evening only; quinine has no specific effect on the course of the fever. Hepatic enlargement in malaria is general; in hepatic abscess there is, when the abscess approaches the surface, a distinct tumour. Perihepatitis, the result of malaria, is usually general, and not localised as in the case of pus reaching the liver boundaries.

Enlargement of the liver in leucocythæmia or pernicious anæmia may suggest pus, but the general symptoms and an examination of the blood will settle the diagnosis well-nigh conclusively.

Of other tumours, such as gummata, those of a malignant nature, etc., clinical acumen must determine the differential points at issue. In almost all cases where liver abscess is suspected, explanatory puncture of the liver is justified and should be undertaken at an early period of the illness. Should leucocythæmia or anæmia be present, however, puncture of liver is unadvisable; but in this case, as in malaria, an examination of the blood from the finger will aid, if it does not altogether decide, the diagnosis.

*Prognosis.*—Recovery from a liver abscess depends upon the cause of the disease, upon the course the pus pursues, upon the stage at which the disease is brought under treatment, and upon the nature of the operation undertaken.

When there is more than one abscess, and when the disease is associated with (general) pyæmia, the prognosis is highly unfavourable. Should, on the other hand, the abscess be single and be of the nature of a "tropical" abscess, instead of a pyæmic infection, the patient with or without operation has a chance—it may be a fair or even a good chance—of recovery.

When the abscess points at the *anterior wall of the abdomen*, and is there opened, the prognosis is favourable. When the abscess bursts in the *bowel*, recovery may also be hoped for.

Good results may also follow the *bursting of the abscess into a bronchus*, the pus finding exit by way of the expectoration. This result, although considered a favourable termination by those who still believe in non-interference, cannot be regarded as highly favourable. The abscess may be of such a size that the pus cannot be sufficiently quickly expectorated, and the air-passages of both lungs may be swamped with the quantity of pus, causing an infective pneumonia with speedily fatal results. On the other hand, the consequent expectoration, owing to the pus rising in the bronchi, may so harass the patient that all attempts at rest or sleep are impossible; and the patient, already enfeebled by the illness, may succumb in a day or two from exhaustion. Should, however, the initial rupture of the abscess be lived through, there is no guarantee that recovery will result. In such cases the rule is that when the abscess cavity is relieved of its contents a cessation of fever, cough, etc., takes place, only to be renewed when the cavity refills. Emptying and refilling may alternate for many months or for years before the cavity heals; and the patient at any time may develop hectic and succumb to the prolonged constitutional strain. At first the intervals in the attacks may be but a week or two, but afterwards two or three or more months may intervene. Every time the cavity

fills the patient becomes feverish, melancholic, loses appetite and weight, is unable to pursue his daily occupation and is confined to his bed or to the house. The result may be recovery; but, on the other hand, a fatal issue is not uncommon Moreover, once the abscess has been allowed to burst into the lung, the chances of localising the pus in the liver by aspiration are greatly diminished, or may be rendered altogether impossible owing to the narrowed limits of the sac of the abscess in the liver. Such a result, therefore, should never be waited for. To allow a liver abscess to burst through the lung is unjustifiable; it means either want of acumen in diagnosis, or a hesitation in operation which is not consistent with rational treatment.

Rupture of an abscess of the liver into the *pleura* causes symptoms of empyema, and unless speedily operated upon will certainly tend to a fatal result.

Rupture of a hepatic abscess into the *stomach* brings on vomiting of liver pus, and a cessation of the urgent symptoms arising from the retained pus; but the prognosis is most unfavourable.

When liver pus finds exit into the *pericardium*, the *peritoneal* cavity, or the *inferior vena cava*, a speedily fatal case can only be looked for.

Pus from the liver may burrow downwards, and after involving the right kidney, may find exit in the ureter; or it may pass behind the kidney and point in the loin. The former generally ends fatally, but the latter may result in recovery.

Of all the modes of spontaneous termination, that by way of the lung is the most common. Render has analysed the results of 563 cases of liver abscess and finds that in 159 cases —that is, in 28 per cent — spontaneous rupture occurred, and the path taken by the pus in the 159 cases is recorded by Render as follows:—Ruptured into the lung in 59 cases; into the peritoneum, 39; into the pleura, 31; into the stomach and duodenum, 8; into the colon, 6; into the loin, 6; into the bile ducts, 4; into the inferior vena cava, 3; into the kidney, 2; and into the pericardium, 1. The order here cited fairly well represents the scale of danger in spontaneous rupture generally. It will be observed that 90 of the cases burst upwards into either the lung or pleura, proving the frequency with which the abscess is situated towards the back or dome of the liver. The course of the pus is no doubt determined by the course of the lymphatics, and as the majority of the lymphatics of the liver find exit upwards between the layers of the coronary ligament, the path of the pus is no doubt thus determined.

*Early operation* is an important factor in the recovery from liver abscess. Delay can only lead to loss of strength, destruction of liver tissue, and involvement, or, it may be, the perforation of some neighbouring viscus. There is no justification for delay, and the postponement of the operation for even a single day may lead to unfavourable results.

*Mortality.*—Statistics of the results of liver abscess up to the last twenty years have little bearing upon present-day results. Operations are undertaken now at a much earlier stage in the disease; clinical symptoms have a clearer interpretation; the aspirator as an aid to diagnosis has revolutionised our treatment; Listerism has minimised the dangers of laparotomy; and a liver abscess is no longer viewed as the rare disease it was once held to be, nor dreaded by the surgeon as a well-nigh incurable ailment. Figures which once indicated a mortality of 80 per cent from liver abscesses have fallen to between 20 and 30 per cent, and there is no doubt that with earlier operations the mortality will be still further reduced.

Treatment. — *Medicinal* treatment in suppuration in the liver is confined to allaying the effects of such concurrent ailments as hepatitis, intestinal flux, dysentery, and fever. These are to be treated with the usual remedies specially applicable when no abscess in the liver is present; but as each and all may be dependent upon or caused by the presence of pus in the liver, the means of treatment and drugs at our disposal may fail to accomplish the marked effects usually attributed to them. Poultices or counter-irritation of the liver, and drugs, may serve to mitigate the local and general effects of hepatitis. Dysentery may be treated by Epsom salts or by ipecacuanha, and fever may be controlled by some one of the available methods. None of these applications or drugs can, however, affect the abscess, which, when once formed, exists as a pathological product, incapable of treatment by medicinal agencies.

*Expectant Treatment.*—Liver abscesses have been found on post-mortem examination to have become absorbed. In the substance of the liver, what could only have been the sites of pus have been very occasionally met with in bodies in which no clue to pus being present in the liver was afforded during life. The pus evidently became absorbed, and a fibrous or a cretaceous remnant may be all that betrays the fact that pus once was present.

Expectant treatment, however, bears a broader significance. The belief existed, if it does not yet exist, that it was better in the interests of the patient not to operate, but to let the pus pursue its bent towards the lung, intestine, or whatever organ or tissue it selects to traverse. This principle of treatment was, no doubt, advocated in view of the unsatisfactory results which obtained from the older form of operation. Partly also it was due, no doubt, to hesitation on the part of medical practitioners isolated in out-of-the-way places to undertake abdominal

operations single-handed. There is now, however, no justification for the expectant treatment. With the aspirator and a simple trocar and cannula any medical man can operate by himself and at once, and there is no reason for delay. The expectant treatment, therefore, cannot be too strongly condemned; it is wrong in principle, and dangerous in the extreme to the patient. Delay even is indefensible. The moment a liver abscess is suspected, no time ought to be lost in exploring the liver and draining the abscess when found.

*Exploring the Liver for Pus.*—There are two rules to be followed when an abscess in the liver is suspected: (1) explore the liver at once by the needle of an aspirator; (2) be prepared to operate forthwith if pus is found. There need be no hesitancy in regard to probing the liver by a hollow needle in search of pus. If pus is not found, the congestion, hyperæmia, or inflammatory state of the liver will be relieved by the punctures. The pricking of the liver capsule in several places may of itself lessen the pain and tension, and the abstraction of blood, either immediately by the needle of the aspirator, or by the subsequent escape of blood into the peritoneal cavity from the punctures in the liver (see above), will do good. When operating in such cases, and when the blood flows fairly freely through the aspirating needle, the writer invariably allows the blood to flow to the extent of 5 to 8 ounces before withdrawing the needle and inserting it elsewhere. This procedure is usually attended by satisfactory results, even when no pus is found, the temperature falling almost immediately, the pain subsiding, and any cough, local distress, or intestinal flux, which had existed previously, disappearing.

*The Site of Puncture.*—The needle of the aspirator has to be inserted at the point where, by percussion, abnormal dulness is elicited. It matters not whether the dulness or the tumour is made out in the front, the right side, or in the scapular line of the chest; or whether it is towards the epigastrium that the abscess is tending, and the abdominal wall has to be punctured. When the chest wall has to be traversed, the inexperienced, in such cases, hesitate for anatomical reasons to puncture beyond a couple of inches above the lower border of the rib cartilages. The pleura descends to within three fingers' breadth of the rib cartilages, and it is this anatomical feature which engenders the hesitancy to puncture higher. There need be no such dread; the fact that the pleura is traversed by the needle or by a trocar and cannula is no deterrent, and all danger from such a source is to be discounted. The danger of puncturing the lung itself is also of but anatomical interest. The right lung normally comes as low as a line drawn from the sternum, at the spot where the sixth cartillage joins it, backwards along the right side to the spine of the tenth dorsal vertebra. This limit in the nipple line crosses the sixth rib; in the posterior axillary line it crosses the eighth rib; and in the scapular line the tenth rib. It may be unwise to puncture above these levels; but when the liver is swollen, or when a collection of pus has pushed up the right lung, there is but little danger of the lung being wounded. The writer has frequently punctured the lung during an exploration of the liver, as testified by a blood-tinged sputum subsequently, but no untoward symptom has ever resulted. It is needless, therefore, to confine exploratory punctures to subpleural or even subpulmonary limits, as no damage can be done; by adhering too closely to these normal anatomical limits an abscess of the dome of the liver may be altogether missed.

*The Depth of the Puncture.*—How deeply is it safe to insert a needle or a trocar and cannula into the liver? The chief danger to be contemplated is puncture of the inferior vena cava or the trunk of the portal vein before it gains entrance to the liver substance. The writer has experimentally tested the point, but the chances of exact measurement are few. So far this has been proved—namely, that in a body of 32 inches in circumference, and in the region of the liver, the inferior vena cava is 4 inches from the surface of the chest, anywhere in the hepatic region in a horizontal line drawn round the right side of the chest from the mid-line in front to the angles of the ribs. The measurements were made in the frozen section of the body. When chest measurements are greater or less than 32 inches, the distance, of course, proportionably diminishes or increases; but it may be taken as a broad rule that no needle or trocar should be introduced to a greater depth into the liver in the direction of the vertebral column, horizontally from the surface, than 4 inches. Should this rule be observed, the dangers of hæmorrhage which have been recorded as having occurred after hepatic exploratory puncture would probably be annulled. The portal vein, should it be punctured at the gate of the liver before it breaks up in the liver substance, would probably result in fatal hæmorrhage. This accident it is difficult to guard against by anatomical measurements, but care exercised during operation that the needle-point is traversing resisting tissues will go far towards preventing so untoward a result. The writer has had the needle of the aspirator and the trocar and cannula with which he operates marked in inches, so that the depth of the puncture by either instrument is exactly known.

*Before* exploratory puncture of liver, steps should be taken to render the skin and instrument aseptic. *After* operation, should pus not be found, it is necessary only to wipe the puncture wounds clean, and cover them over with a film of blue wool and a coating of collodion.

A binder round the body from the level of the nipples to the umbilicus, pulled tight and securely pinned, should be applied and kept in place for several days.

An *anæsthetic* should always be administered when exploratory puncture is undertaken unless there are pulmonary or cardiac conditions forbidding it.

*Operation by* (1) *the Trocar and Cannula.*—The treatment of liver abscesses by trocar and cannula and subsequent drainage has many advocates and many detractors; these, however, will be discussed later. The principle and practice followed by the writer in the treatment of liver abscess is that initiated and practised by Dr. Patrick Manson. In the hands of many practitioners, also, the writer has seen excellent results from the same line of treatment; and there can be little doubt that on the principle taught by Dr. Manson liver abscesses will in future be treated. The instrument is a trocar with a bayonet point fitting into a cannula some 4 or 5 inches long. The steps of the operation are as follows:—When pus has been found by exploratory puncture with the needle of the aspirator, the pus should not be drawn off in any quantity, but merely in quantity sufficient to establish the fact that pus is present. The reason for this is evident; for were the abscess to be emptied it would be impossible to tell when the cannula was introduced — whether it was in the cavity of the abscess or not—owing to the absence of the flow of pus. The depth to which the needle entered and the direction it followed should be carefully noted, so as to serve as a guide to the course along which the trocar is to be introduced. The movements of the needle after it is introduced into the liver aid in the diagnosis of whether or not the point is an abscess cavity. When the needle is embedded in the liver tissue, the part of the needle external to the chest or abdominal wall will move up and down with the respirations; but should the needle lie in a cavity, a more pronounced swing or pendulum-like movement (Manson) is communicated. Unless, however, this is pronounced, the observation is of little value. When the pendulum-like movement is very limited it may serve to indicate that adhesions have taken place between the liver and its surroundings; but when it is entirely absent, then the needle, even if pus is drawn off, is not in the liver at all. When the needle of the aspirator is withdrawn, along the same track introduce the trocar and cannula in the direction indicated by the needle. When the depth at which pus was found by the needle is reached withdraw the trocar, and pus, if the abscess cavity is reached, will flow from the cannula. Do not allow much pus to flow, only sufficient to indicate its presence, when the thumb is to be placed over the opening of the cannula.

Dr. Manson's description of his apparatus and his mode of using it is as follows:—

The necessary apparatus consists of large trocar and cannula, 4 to 5 inches long, by $\frac{3}{8}$-inch in diameter; a steel stilette, at least 14 inches in length; two metal buttons, $\frac{1}{4}$-inch at their greatest diameter, with long ($\frac{1}{2}$-inch), hollow, roughened necks into which the ends of the stilette fit loosely; 6 inches of $\frac{1}{2}$-inch stout drainage-tubing. While the ends of the drainage-tubing are held and well stretched by an assistant, they are firmly lashed to the stem of the buttons, over the ends of the shorter of which, for additional security, the tubing is also tied. Two large holes, to provide for free drainage, are then cut close to one end of the drainage-tube. The tube is then mounted on the stilette by inserting one end of the latter through one of the drainage-holes, and lodging it in the hollow neck of the distal button, and thereafter so stretching the rubber that the other end of the stilette can be inserted into the neck of the other button. When thus stretched, the drainage-tube should be capable of passing easily through the cannula. The apparatus being thus prepared, and rendered thoroughly aseptic by soaking in carbolic lotion, and the position and depth of the abscess having been carefully ascertained by means of the aspirator and noted, the aspirator is withdrawn, and an incision about an inch in length made with a scalpel through the skin at the site of the puncture. The trocar and cannula are then thrust into the abscess and the trocar is withdrawn. After allowing a small quantity of pus to escape, so as to relieve any tension that may be present in the abscess sac, the stretched drainage-tube, perforated end first, is slipped into the cannula and carried to the back of the abscess. Holding the stretched drainage-tube firmly, and maintaining it carefully in contact with the back of the abscess with one hand, the cannula is withdrawn with the other. Still grasping the drainage-tube firmly, the button on the free end of the apparatus is slipped off the stilette, the end of which is made to perforate the drainage-tube close to the button. This it readily does, and the drainage-tube is allowed slowly to resile towards the fixed end, still held in contact with the back of the abscess. When the drainage-tube has completely contracted the stilette is withdrawn.

The writer has adopted Dr. Manson's principle of treatment, and also his apparatus, but with a few modifications of the latter. It will be observed that the tube employed by Dr. Manson is closed at the end, and that as the tube lies in the abscess cavity the deeper $\frac{2}{3}$rds of an inch is blocked and valueless as a drain. The writer introduces a drainage-tube open at the end, and stretched merely upon a metal rod with a blunt hook at one side so as to catch and stretch the tube. This allows of an open end to the

drainage-tube—a most important point; as if the tube has only just entered the abscess wall, or if the abscess is small, and when contraction during healing takes place, there is a probability that the pus is not removed, but lies around the end of the tube, and must be in some quantity before reaching the apertures at the side.

Another important point is the question of drainage. The writer used invariably, whilst practising in the Tropics, to follow the plan of draining off the pus through a long tube, conducted from the patient's side over the side of the bed into a basin full of carbolised water placed on a low stool by the side of the bed. In this way a syphon action was continuously at work furthering the flow of pus. Following the practice of surgeons in Britain, the writer left off the syphon drainage only to repent it, and has now returned to the use of the syphon drainage as above described. Liver pus is so gelatinous and tenacious that no known dressing will absorb it, and it gathers between the skin and dressing; the exit is thus hampered by the dressing and by its own consistency. The syphon drainage obviates this and promotes a continuous flow.

The drainage-tube ought to be used until pus ceases to flow, and the fluid coming away is bile-stained merely. This may be weeks after the operation, when a smaller tube is inserted and the length gradually shortened. The presence of a tube in the side causes no inconvenience. A patient of the writer's went through the China-Japan campaign in 1894-95 with a drainage-tube in his side which had been introduced after an operation for a liver abscess; and another, the captain of a sailing vessel, started on his long voyage from China to New York with a tube in his side, getting rid of it only after being seven weeks at sea. Dr. Neil MacLeod, of Shanghai, has still further modified the apparatus for liver abscess operation by using an oval silver flexible drainage-tube, which possesses the advantage of not kinking. Dr. MacLeod has also introduced a trocar and cannula which can be introduced upon a guiding rod, by which means failure to reach the pus, after the needle of the aspirator has proved pus to be present, is well-nigh impossible. (See *Journal of Tropical Medicine*, Nov. 15, 1900.)

(2) *Operation by Incisions.*—A liver abscess can be reached by way of the abdomen (laparotomy) or by way of the chest wall. Both of these operations have been so severely condemned by those who have had experience of operations for liver abscess that it seems unnecessary to continue to mention them. Dr. Manson, Colonel Kenneth MacLeod of Netley, and Dr. Neil MacLeod of Shanghai, have all spoken and written so strongly on the subject that there seems no justification for regarding cutting operations seriously. The writer is also thoroughly in accord with these authorities.

*When the tumour formed by the liver abscess is felt in the epigastrium or anywhere in the wall of the abdomen* the abscess may be reached by cutting down upon it directly. The preliminary steps are those necessary for an ordinary laparotomy. An incision is made, some 4 inches long, over the most prominent part of the tumour.

(*a*) When the peritoneum is reached and opened the pus may at once flow from the wound. This is due to close apposition or adhesions between the liver and abdominal wall, and is what may be termed the "old method" of dealing with liver abscess when the disease was allowed to develop (if the patient lived as long) until the abscess bulged in the epigastrium or the right hypochondrium. With our modern methods of treatment such a state of development should never be allowed to obtain; as for one case in which a favourable issue ensues, there are many which succumb to some one of the many possible contingencies consequent upon allowing the abscess to run an unchecked course.

(*b*) When the peritoneum is opened and the liver is seen at the bottom of the wound, and the outline of an abscess can be traced, one of two measures can be taken. (1) Should the abscess appear as likely to burst soon, the surrounding parts of the wound should be packed with gauze or sponges, the whole pressed down upon the liver, and the abscess incised. (2) Should the abscess seem less likely to burst immediately, the wound may be packed with gauze, dressed, and bandaged, with the idea of allowing adhesions to form between the liver and parietes. Every day the wound should be inspected, and if the abscess threatens to point very decidedly it should be opened; but if the indications are not urgent the wound may be kept open for three or four more days before the abscess is cut into. It is doubtful whether twenty-four hours is not long enough, unless the abscess is very large. With a small abscess there is little danger of the liver retracting with even the slight adhesions formed after twenty-four hours; but if the abscess is enormous, the liver may recede and leave a gap by which the pus may reach the peritoneal cavity. But even with a large abscess equable pressure on the abdominal wall will probably suffice to prevent the retraction of the liver, and, after a few days, barriers of lymph will develop and obviate danger of extravasation.

(*c*) When the peritoneum is opened, the liver exposed, and no abscess is to be seen, or the presence of pus indicated in any way, the needle of an aspirator is to be introduced into the liver in search of pus. When it is found, one of two measures may be undertaken. (1) The wound may be packed around its circumference with sponges or gauze, the abscess incised, emptied, the margins of wound stitched to the margins of the abdominal wound, and a large drainage-

tube inserted. (2) Before opening the abscess some surgeons recommend that the liver be secured to the parietal peritoneum by a double row of sutures. By an outer row of silk interrupted (overlapping) stitches the liver is fixed to the parietal peritoneum about 1 inch from the margin of the cut peritoneum; by an inner row of continuous catgut sutures the margin of the parietal peritoneum is sewn to the liver surface—leaving a surface of almost 1½ inch in circumference exposed. Mr. Godlee, who advocates this method of suture, recommends that the posterior sheath of the rectus be included with the peritoneum in the sutures when the incision is made through the mass of the rectus muscle.

The difficulty found by all surgeons is to get sufficient traction upon these sutures without the liver substance tearing through. This is especially the case when the liver around the abscess is in a state of inflammation. It should be remembered, however, that but slight traction on the suture is necessary, and that what appears a lax suture will be sufficient to establish speedy effusion and close juxtaposition of the peritoneal surfaces.

*Operation of Incision by way of the Thorax.*—When the abscess is situated at what is called the back of the liver, it must be approached by way of the chest wall. When the pus is bulging at an intercostal space it may be sufficient to incise the abscess freely and insert as large a drainage-tube as can pass between the ribs without being compressed. Most surgeons, however, recommend the removal of an inch or two of a rib so as to give more room. When the pus is deeply-seated, and the cavity of the right pleura has to be traversed, it is considered necessary to stitch the parietal and the diaphragmatic pleuræ together; then to puncture the abscess through the diaphragm, or, if the peritoneum is opened and yet no pus found, to stitch the peritoneal surfaces together and then to the margins of the pleuræ and parietal parts before opening the abscess. This is an operation which few would care to undertake, and after these preliminary steps are taken there is the uncertainty of finding the abscess.

*Operation by Incision and Trocar and Cannula compared.*—The advocates of operation by incisions—that is, by a laparotomy or by a thoracic wound—declare that only by these means can one see what one is doing. In bold terms they state that they wish to look "their enemy in the face," and that the operation by puncture is a half-hearted step, and undertaken only by timorous surgeons. So far is their pronouncement carried that many declare it is unsurgical and unwise to use even the needle of an aspirator whereby to explore for pus in the liver as a preliminary.

On the other hand, those who use the trocar and cannula claim that there is no (or but little) danger exploring the liver by an aspirator needle; that with the trocar and cannula and the introduction of a large drainage-tube through the cannula the abscess cavity is reached easily, and that the drainage is ample; and further, that the patient is not submitted to the dangers of laparotomy or to wound of pleura, diaphragm, or lung.

The writer has practised both methods, and unhesitatingly declares in favour of the trocar and cannula. His reasons for so doing are:—(1) That the operation is one of much less severity. (2) That there is less likelihood of the pus finding its way into the peritoneal or pleural cavities after puncture by trocar and cannula than after incisions. The writer has never found pus gain access to either the peritoneum or pleura after puncture, but has seen it frequently do so after incisions. (3) Suturing the liver to the abdominal wall is only occasionally (if ever) possible so as to secure a continuous channel. (4) Suturing the pleural layers and the diaphragm, etc., is usually only a surgical farce—no completeness in the closing of the wounds and complete apposition of the parts being possible owing to the mobility of the organs and the nature of the tissues being dealt with. (5) When the liver is exposed by a laparotomy the surgeon is in no better position as regards the localisation of a deep-seated liver abscess. The liver has still to be punctured by a searching needle, and with more probability of the blood or pus, which follows withdrawal of the needle, gaining access to the peritoneal cavity than when the parts are in naturally maintained apposition. Colonel Macleod, Netley, related an instance of this at the section of Tropical Disease at Ipswich, 1900. A surgeon had cut down by a laparotomy on a liver believed to be the seat of an abscess. The liver, however, when exposed, appeared normal and the operation was abandoned; yet, in a few days afterwards, a quantity of liver pus was passed by the rectum. Had such a case been explored by a needle, in all probability the abscess would have been discovered, and a trocar and cannula would have allowed its escape by a safer channel. (6) The ease with which a liver abscess is reached and treated by a trocar and cannula encourages a medical practitioner, who may be single-handed at the operating table, to treat a liver abscess by this means, who would be deterred practising a laparotomy or a thoracic incision. Liver abscesses occur in the tropics usually, and the surgeon may be called upon to operate away from hospital wards and hospital appliances. Operations by incisions under these circumstances can only be done at great risk to the patient, the operator being alone and with perhaps a native hospital attendant or ward coolie assisting. Therefore, anything that can simplify operations for liver abscess is to be

strongly commended, as early operation is everything, and the despatch of a patient with a liver abscess by train, native cart, or other rude conveyance to the nearest large hospital, where laparotomy may be done with fair prospect of success, may mean rupture of the abscess into the peritoneum or elsewhere with fatal results. Independently, however, of appliances, of assistance, of nursing, etc., the writer agrees with the verdict on this subject come to by all those present at the section of Tropical Diseases at Ipswich, and they represented some of the best of our tropical practitioners: the operation by incision is unnecessary in its severity, and is attended by less favourable results than operation by trocar and cannula.

PYLEPHLEBITIS—PORTAL THROMBOSIS

ADHESIVE THROMBOSIS . . . . 515
SUPPURATIVE THROMBOSIS . . . 518
*Pathological Considerations* . . 518
*Signs and Symptoms* . . . 519
*Diagnosis and Prognosis* . . . 519
*Treatment* . . . . . 519

A. ADHESIVE THROMBOSIS OF THE PORTAL VEIN.—Obstruction of the portal vein may be caused by coagula developing in the capillary distribution of the portal vein within the liver, in the trunk of the vein or its main branches, or in the radicles of the portal vein anywhere in the gathering area of the vessel.

1. *Coagula of Blood commencing in the Capillaries of the Portal Vein within the Liver.*—This is by far the most common starting-point of portal obstruction resulting in adhesive phlebitis. The coagula having formed in the portal capillaries within the liver extend backwards to the small venous trunks, and ultimately may completely or partially block the main trunk of the portal vein itself. The causes of the primary coagula may be—

(*a*) Deficient cardiac power, due to thrombosis of the pulmonary artery or other cardiac lesion, to pulmonary disease or other causes of blood stasis in the circulation generally or the portal circulation in particular. The obstruction in the portal vein may develop a short time before death, or it may appear only as the patient is dying. The causes of obstruction of this nature are usually associated with lesions of a fatal nature affecting the thoracic viscera, so that the signs and symptoms induced are but of classic interest clinically. The coagula in such cases will be found of a reddish-black colour, evidently of recent formation, completely filling the lumen of the vein, but readily detachable from the wall. The abdominal viscera will be found deeply congested, with frequent ecchymoses, and with a quantity of serous effusion tinged red in the peritoneal cavity in cases of some standing.

(*b*) Cirrhosis of the liver is the commonest cause of adhesive portal thrombosis. The contractile nature of the fibrous deposit in the liver in cirrhosis leads to obstruction and exclusion of the portal capillaries and the consequent formation of coagula within their channels. The coagula once formed tend to increase by deposit and accretions upon their surfaces until the venous branches and ultimately the main trunk of the portal vein itself are blocked. The coagula in cirrhosis may be and generally are slowly deposited, as may be gathered by a study of the thrombosis post-mortem. The earlier-formed coagula are found frequently laminated in structure indicative of gradual deposit, or to consist of brownish masses firm in consistence and even organised into an embryonic connective tissue. Associated with the older thrombi are more recent coagula which may extend into the lumen of the larger veins as semi-solid coagula. The whole thrombus becomes adherent to the vein wall, and the inner aspect of the vein loses its gloss and appears corrugated and injected. The whole thickness of the vein becomes thickened and swollen. The connective tissue around the vein is involved in the inflammatory change, so much so that it becomes adherent to the circumferential structures. The obstruction may be even so gradually developed that the portal vein or its main tributaries become varicose or dilated.

(*c*) Cancerous deposit in the liver may give rise to a form of adhesive portal thrombosis; but the affection, in contradistinction to that which occurs in cirrhosis, is usually of a limited character, and the thrombus is a mere coagulation usually, although the cancerous material may even invade the lumen of the vein itself.

(*d*) Inflammatory affections of the liver, more especially those that end in suppuration, may give rise to thrombosis of the portal capillaries. The thrombi formed in this connection are liable to break down or become foci for further purulent deposit. Thrombosis from liver abscess is not, however, a common occurrence.

2. *Thrombosis* arising from lesions affecting the *trunk* of the vein or its main tributaries occasionally occurs.

(*a*) Dilated bile ducts in the lesser omentum may cause chronic inflammatory thickening in the surrounding tissues, to such an extent that the trunk of the portal vein may be incorporated in the fibrous mass and become thereby compressed and constricted. The altered nutrition of the vein wall leads also to alteration and thickening of its several coats, rendering them indurated or even semi-cartilaginous in consistence. In consequence of these changes the calibre is narrowed, laminated coagula are formed within its lumen, and a dilated and even varicose condition may obtain throughout the radicles and large

trunks of the splenic and superior mesenteric veins.

(*b*) Morbid growths, of a cancerous character mostly, may impinge upon the wall of the portal vein more especially near the gate of the liver and determine the formation of a thrombus in the trunk of the vein; but the large trunks which converge to form the portal vein may each and all be involved in malignant growths according as the cancer has its seat in the pylorus, the pancreas, or the mesentery. The lymphatic glands on the front of the vertebral column may be the seat of cancerous or tubercular deposit, and one of the branches of the portal vein may become involved in the tumour, with the result that the vein traversing the mass becomes thrombosed. Not only may this thrombus occupy the branch, but, from the smaller vessels, the coagulum may extend to the main trunk.

(*c*) Inflammatory thickenings having their origin in gastric or pyloric ulcers or in pancreatic inflammation may encroach upon and involve the portal vein, leading to the deposit of coagula and the formation of a completely obstructing thrombus.

(*d*) Chronic peritonitis may cause constriction of the portal vein near the gate of the liver or of the mesenteric vein, etc., with a resulting thrombosis.

3. *Thrombosis commencing in the Peripheral Radicles of the Portal Vein.*—In any one of the several chylopoietic viscera, inflammatory or other lesions may cause the formation of thrombi which extend towards the main channels or the trunk of the portal vein, or emboli may become detached and block the terminal capillaries of the portal vein within the liver itself, leading to thrombosis. It is but seldom that adhesive thrombosis results from lesions of this nature, the suppurative form being the more common; yet it is not unknown clinically—coagula becoming detached from the neighbourhood of ulcers and cancerous growth in the intestines and other viscera and buried in the liver.

*Signs and Symptoms.*—Obstruction of the portal vein, due to adhesive thrombosis, be the origin what it may, presents many signs and symptoms in common. The affection is characterised by a rather chronic manifestation of symptoms when compared with the more acute course of thrombosis of a suppurative character. The disease does not arise primarily, but as a superimposed lesion in some constitutional or local disease. Although disease of the thoracic viscera may play a part in the etiology, it is in the abdominal viscera that the genesis of the ailment is to be looked for. During the course of cirrhosis or cancer of the liver, of ulceration of either a cancerous or inflammatory nature in other abdominal viscera, of chronic peritonitis, etc., obstruction of the portal vein may arise. The onset of the disease is usually sudden, or, at all events, the manifestation of the results of a gradual thrombosis declares itself suddenly.

*Ascites* is one of the most constant developments in portal thrombosis. It is natural it should be so, and it is also natural that the ascitic fluid should accumulate rapidly. In a few days the abdomen may be enormously distended with fluid, and the rapidity of its accumulation is well shown by the way in which the abdomen fills after paracentesis has been performed. The only cases in which ascites may occupy a secondary place is when relief to the congested portal vein is obtained by hæmorrhage into either the stomach or the intestine. Occasionally the ascites is gradually developed, implying an incomplete blocking of the venous channel, or from the fact that only one of the main tributaries of the portal vein is involved.

*Disorders of the alimentary canal*, due to adhesive portal thrombosis, are universal. In the stomach, vomiting, dyspepsia, and hæmatemesis are usual concomitants, the loss of blood being at times alarming. An intestinal flux, of the nature of watery diarrhœa, or accompanied by some hæmorrhage from the bowel, is a predominant feature; the hæmorrhage, however, may be severe, amounting at times to the passage of a quantity of almost pure blood from the bowel. Piles, although an early feature of the disease, cease to give trouble usually when either ascites or an intestinal flux, simple or hæmorrhagic, takes place.

The *spleen* swells to many times its normal bulk, a fact which may be only discernible when the ascitic fluid has been drawn off. Should, however, the portal vein be relieved by either hæmatemesis or hæmorrhage from the bowel, the spleen may be simultaneously relieved and assume normal proportions. Should, on the other hand, the spleen be bound down by previous perisplenitis, or its parenchyma altered by chronic disease, its enlargement may be arrested.

The *liver* may show few signs of alteration, apart from the original disease of which it may have been the seat. The size seldom exceeds the normal, and, as a rule, it is diminished in size owing to the prevalent cirrhosis. Should one branch or one division of a branch of the portal vein as it breaks up in the liver become alone the seat of thrombosis, an atrophy of the part of the liver to which it is distributed ensues. Post-mortem lesions of this nature are occasionally met with, although no evidence of hepatic derangement has been observed during life. The liver in the part cut off from portal blood shrivels, the hepatic cells liquefy, and the residue becomes absorbed, and a mass of cicatricial fibrous tissue takes their place. As, however, it is now well known that when one part of the liver is cut off from nutrition

the remainder hypertrophies and takes upon itself extra functions, it is easily understood how an even extensive atrophy of part of the liver may be attended by no clinical evidence.

The *general* condition of the patient when portal thrombosis is established is one of decline: the physical powers fade and the digestive functions fail; the urine is small in quantity and of a high specific gravity; œdema of the feet, an anæmic appearance, and, finally, a cachectic state supervene as the inevitable consequence approaches.

*Compensatory Channels of Circulation.*—Dilatation of the superficial abdominal veins is often a marked feature of adhesive portal thrombosis. The apparent venous enlargement is, however, only an indication of extensive and more deep-seated efforts of the same nature to establish a collateral circulation so as to relieve the portal system. On the extent to which this is developed depends to some extent the length of life possible for the patient, and it also has some influence in modifying the signs and symptoms, such as ascites, hæmorrhage, etc., caused by the thrombosis.

The chief anastomoses between the portal and systemic systems are as follows:—

VEINS

| *Portal System.* | *General or Systemic.* |
|---|---|
| 1. Coronary veins of stomach anastomose with | œsophageal. |
| 2. Coronary veins of stomach anastomose with | phrenic. |
| 3. Coronary veins of stomach anastomose with | left renal. |
| 4. Vasa brevia of stomach anastomose with | left phrenic. |
| 5. Gastro-epiploica of stomach anastomose with | left renal. |
| 6. Superior mesenteric veins anastomose with | left renal. |
| 7. Hæmorrhoidal veins anastomose with | pudic. |
| 8. Veins in liver adhesions anastomose with | phrenic. |
| 9. Veins in liver adhesions anastomose with | veins in abdominal wall. |
| 10. Veins in falciform ligament anastomose with | phrenic. |
| 11. Veins in falciform ligament anastomose with | veins in abdominal wall. |

It is by way of the veins in the falciform ligament (10 and 11) of the liver that the anastomosis which gives rise to the superficial abdominal veins becoming enlarged takes place. In almost every case of hepatic cirrhosis these veins will be found to be enlarged; but as the distension is usually confined to the deeper veins, the condition is apt to be overlooked, and it is only when the obstruction in the portal vein is pronounced and prolonged that the superficial veins become apparent. The veins in the falciform ligament pass from the wall of the abdomen opposite the attachment of the falciform ligament and from the under surface of the diaphragm to the liver; the former, according to Sappey, reaching the under surface and joining the left branch of the portal vein; the latter gaining the dome of the liver. The current of blood in these veins is normally towards the liver, but when the portal vein is obstructed the current of blood is reversed and passes between the layers of the falciform ligament to gain the deep epigastric (superior and inferior) veins in the substance of the rectus muscle, and in these the blood is conducted upwards to the internal mammary or downwards into the external iliac veins.

The portal vein, as the result of its hepatic capillaries becoming partially obstructed, may become distended or varicose. Portal, in *Maladies du Foie*, describes a portal vein enlarged to the dimensions of the small intestines. Uniform distension is, however, much more common than varicosity, owing no doubt to the absence of valves in the veins of the portal system, in consequence of which true varicosity cannot take place. The causes which induce thrombosis are for the most part similar to those which induce a dilatation of the portal vein, but localised dilatations due to patchy peritonitic thickening are more common than is localised thrombosis.

The wall of the portal vein may be itself the seat of chronic phlebitic changes and of calcification of a more or less extensive character. These may each induce an adhesive portal thrombosis independently of hepatic or other diseases.

*Diagnosis.*—Although there is no single sign or symptom which definitely pronounces the portal vein to be obstructed, a fairly positive opinion may be arrived at when the signs and symptoms are taken collectively. Ascites rapidly developing and rapidly recurring after the abdomen is tapped, hæmatemesis or hæmorrhage from the bowel, the splenic enlargement, etc., all point to a possible thrombosis, although these are all but part and parcel of the usually adopted indications of ordinary advanced cirrhosis. It is, however, well-nigh certain that when these developments occur during the course of a cirrhosis of the liver they are due to a supervening thrombosis of the portal vein.

*Prognosis.*—A fatal issue is to be looked for when the portal vein is thrombosed. Collateral circulation, precarious at best, may prolong life for a time; but with the liver and its functions practically withdrawn from the economy, recovery is impossible. It is only in cases due to mechanical obstructions, such as those caused by dilated bile ducts due to gall-stones, or to

constriction by peritoneal thickenings, to pressure of enlarged glands, etc., or in any condition in which, by surgical measures, the cause of the obstruction could be removed, that there is any hope of recovery. Relief by surgical operation is seldom possible, and the necessity for surgical measures are seldom sufficiently pronounced to justify their adoption.

*Treatment.*—Relief by paracentesis is well-nigh the only treatment which is justifiable. Withdrawal of the ascitic fluid should only be done, however, when the breathing or circulation is deeply embarrassed. When hæmorrhage is severe from either the stomach or bowel, astringents may be employed. Diarrhœa when excessive may require treatment. To attempt to get rid of the ascitic fluid, or to increase the quantity of urine by drastic purgatives or by diuretics, are measures which are calculated to do more harm than good, as the patient's strength will not allow of severe purging, and the low blood-pressure prevents the kidney assisting much in the excretion of the body fluids.

Surgical treatment with the idea of removing obstructions to the trunk of the portal vein, although in some cases rational, is in practice a negligible quantity.

B. Suppurative Thrombosis of the Portal Vein.—The description given of the possible seats of the pathological factors concerned in the formation of adhesive thrombi in the portal vein applies equally in the suppurative form of the disease.

The disease finds origin in some inflammatory lesion in which pus or ulceration has played a part, and from which, as a focus, the thrombosis may extend, or at which coagula may be formed which journey onwards to the liver and there get caught, forming emboli. The nature of the thrombus or coagulum is such that, either by bacterial changes or by altered nutrition, or both combined, the thrombosis is of a diffluent character, unorganised and ready to break down.

*Pathological Considerations.*—It is inexpedient here to discuss the pathology of phlebitis in general, so therefore the question of whether the thrombus is the result of an endophlebitis or of a periphlebitis will be set aside. It is possible both endo- and peri-phlebitis play their part in the disease, the latter resulting from inflammatory lesions around the peripheral venous radicles, and the detached coagula determining an endophlebitis in other parts of the system. Periphlebitis, at all events, is a commonly-met-with condition; the presence of pus along the path of the connective tissue, surrounding the venous trunks with a coterminous thrombosis in the lumen of the vein, being not infrequently found.

The coats of the vein are swollen, congested, and infiltrated. The inner wall loses its endothelial lining, and is injected, dull red or yellowish in appearance, the clot adhering to it in flocculent masses. The muscular coat seems paralysed, refusing to contract when the vein is cut across. The outer coat and sheath are infiltrated with sero-sanguinolent fluid or with pus, and the whole trunk seems swollen and enlarged. The thrombus is almost invariably soft and friable, reddish in parts, almost black in others, and with fibrinous coagula diffused throughout. The thrombus may lie loosely within the lumem of the tube, adherent only at irregular points along the vein wall. The thrombus usually undergoes a fatty degeneration and liquefaction, and the parts of the thrombus so affected have all the appearance of pus. Microscopic examination of these apparently purulent masses shows the cellular elements to consist of nucleated cells and of others undergoing fatty metamorphosis.

1. Suppurative portal thrombosis *commencing within the liver* itself is no doubt a factor in the formation of liver abscess consequent upon suppurative hepatitis, but pathological data are wanting. At the gate of the liver, however, a subhepatic abscess has been found associated with portal thrombosis; but which was the primary lesion in affections of this nature is unsettled. Portal phlebitis is a rarer lesion in liver abscess than is hepatic phlebitis. This is no doubt accounted for by the fact that the portal vein is supported by a sheath of connective tissue even in its branches in the liver, whereas the hepatic veins in the liver are more of the nature of channels in the liver substance having a wall of but microscopic dimensions.

2. Suppurative thrombosis may arise in consequence of affection of the *trunk of the portal vein* or of its main tributaries. Wounds of the trunk or branches of the superior mesenteric vein may lead to thrombosis. Resection of the gut, operations for strangulated hernia, etc., may determine the lesion; and Lambron, in the *Archiv. gén. et méd.*, 1842, relates a case of a thrombus caused in this vein by the transfixion of a fish-bone.

Gall-stones in the ductus communis choledochus, however, attended by inflammation and suppuration, may induce portal thrombosis. Suppurating mesenteric glands occasionally press upon and affect the vein, causing thrombi, which may extend for some distance from the seat of the lesion. Although there are many instances of the trunk of the veins being blocked by thrombi and surrounded by pus, it is usually impossible to ascertain which is the cause and which the effect of the condition; but it has been demonstrated clearly that pus may surround a venous channel and yet no thrombus be found within the lumen of the vessel.

3. By far the most common cause of suppurative phlebitis is inflammatory and purulent changes in connection with the peripheral

radicles of the portal vessels. Ulcerations and cancerous changes in the stomach, in the pylorus, in the small and large intestine, and in the rectum, have each and all been followed by thrombus, purulent periphlebitis of the portal vein, and abscesses of the liver. One of the most common lesions in which suppurative thrombi form is in connection with appendicitis, and it is one of the sequelæ of that troublesome affection which most frequently causes death. Suppurative thrombosis of the mesenteric veins was found by the writer in all cases of malignant dysentery he had the opportunity of examining. The coagulation commences in the venous radicles issuing from the affected part of the gut, and advances upwards towards the main trunk, and as far as where the splenic and superior mesenteric veins unite to form the portal. In such cases the gut becomes black, the mucous surface sloughs in huge patches; but whether the phlebitis is consequent upon the so-called dysentery, or *vice versâ*, is a point not yet determined. It is doubtful whether malignant dysentery is in any sense dysentery or a specific disease; it is more likely a conditional and intestinal flux consequent upon primary phlebitis.

*Trismus Nascentium.*—The trismus of newly-born infants is but a local sign of a general blood disease, the primary starting-point being thrombosis of the umbilical vein, due to septic infection. The writer had experience of this in Hong-Kong, where, owing to the frequency of trismus nascentium amongst the infants of the Chinese poor, half the total death-rate was caused by it.

*Foreign bodies* in the alimentary canal, such as penetrating fish-bones, etc., may serve as the starting-point of phlebitis and thrombosis. Abscesses in the spleen are liable to be followed by thrombosis of the splenic and portal veins, the peculiar anatomical conditions of the splenic pulp and its relations to the vein favouring thrombosis.

*Any old cicatrix or adhesion* in the abdominal cavity may, after years of quiescence, lead to phlebitis of the veins in its tissue and subsequent suppuration and venous thrombosis. Such a case occurred in the practice of the writer, where, at the post-mortem performed by Dr. William Hunter, a gastric ulcer, which had healed twenty-five years previously, and acquired adhesion to the pancreas, became irritated in consequence of fish-poisoning, with the result that the veins in the cicatrix became inflamed, the splenic vein and the portal vein up to the gate of the liver were enveloped in pus, and a loose thrombus occupied the channels of both the splenic and portal veins.

*Signs and Symptoms.*—When some one of the several affections mentioned above as provocative of suppurative portal thrombosis occurs, the onset of the lesion in the vein gives rise to the following symptoms. The pain of the affection changes or extends its seat. If the cause is an appendicitis the seat of pain is no longer confined to the right iliac fossa, but extends upwards to the right hypochondrium, or to the umbilicus; and as peritonitis develops, the pain becomes general over the abdomen. In the same way, if the spleen is the seat of trouble, the pain extends across the stomach and gives rise to "cramp in the stomach"; and in similar fashion for other affections.

A *rigor* is an early indication of an extending portal phlebitis, followed by the usual profuse sweating. Rigors may occur not only at the commencement of the illness, but may recur at irregular intervals afterwards.

*Jaundice* sometimes to a marked degree supervenes, and the *liver* becomes permanently enlarged and may be painful on percussion and palpation. The *spleen* enlarges if the splenic vein or the trunk of the portal vein becomes thrombosed. A thin watery *diarrhœa* is the rule, but constipation occasionally obtains. When blood escapes by the bowel in quantity the splenic and hepatic tenderness and enlargement may disappear. *Fever* is always present, and usually assumes an intermittent type; towards the end it usually assumes a hectic character. Pyæmia, with rigors, sweating, and deposit of pus in the joints, in the viscera, or beneath the skin, may result. The general symptoms are feverishness, loss of appetite, loss of flesh, emaciation, and rapid decline in strength. A typhoid state usually supervenes as death approaches.

*Diagnosis.*—It is by the aggregation of the signs and symptoms described above, supervening upon some local inflammatory lesion within the abdominal cavity, by which the presence of suppurative portal thrombosis may be inferred to be present. No single symptom is constant; the pain may be obscured; the rigors and sweating may be ascribable to malaria or general pyæmia; or the presence of dysentery, hepatitis, or splenitis may obscure the diagnosis.

*Prognosis.*—The suppurative phlebitis extends to the trunk of the portal vein. Death is inevitable.

*Treatment.*—The relief of pain and distress and the support of the strength are the only measures to be adopted in the treatment of this fatal malady.

LIVER PARASITES

PARASITES . . . . . 519
*Distomum hepaticum* . . . 520
,, *lanceolatum* . . . 520
,, *sinense* . . . 520
,, *conjunctum* . . . 520
,, *hæmatobium* . . . 520
*Pentastomum constrictum* . . . 520
*Ascaris lumbricoides* . . . . 520
*Tœnia echinococcus* (*Hydatid Disease*) 521

Excluding hydatids, already described, several parasites, all belonging to the distomata variety

of the family *Trematoda*, are met with in the liver or bile passages. Their presence is, however, by no means common; in fact, a liver parasite may be justly considered a rare affection. Still more rare is it for the parasites to be located in the liver itself; the almost invariable habitat is the biliary passages. In the portal vein, also, parasites of this group have been found. Liver parasites belong to the VERMES; class, *Platodes*; family, *Trematoda*, or flukes; variety, *Distoma*.

1. *Distomum hepaticum.* — In its fully developed state the *Distomum hepaticum* is found as a smooth, oval, faintly-yellow, flat worm, measuring 28 mm. by 12 mm. in its widest part. The head protrudes as a short beak from the broader end of the parasite, and presents on its ventral surface a sucker. Immediately behind the sucker is the genital pore, and behind that again a second sucker. The genital pore leads to the uterus, which lies convoluted like a ball of wool above and behind the second sucker. The intestine is branched. The eggs are oval in outline, measuring 0·13 mm. by 0·08 mm. They are of a brown colour; the shell or citaderm consists of two fairly well-marked layers, and at the broader end of the egg is a small lid or operculum.

2. *Distomum lanceolatum.* — This is, as its name implies, a lance-shaped entozoon, measuring 9 mm. by 3·5 mm. The parasite is narrow in front, but widens out gradually until near the posterior extremity. The genital pore is, in the lanceolate species, as in the hepaticum, between two ventral placed discs, but the vaginal tract is much longer, the uterus being situated farther back in the widest part of the body. The intestine is more complex than in the hepaticum, being divided up so as to form many sacs and canals. The eggs are spherical, measuring ·04 mm. by ·03 mm., and within them the mature ova may be found.

The *Distomum hepaticum* and *Distomum lanceolatum* are parasites of the biliary passages of the ruminantia, and more particularly of sheep. Sheep "rot" is attributed to the pathological processes set up by this parasite; but although the parasites are so prevalent and destructive in the ruminants, they are seldom met with in the human being, and even when they occur may give but few indications of their presence.

The following parasites are met with for the most part in tropical countries, but one of them, the *Distomum sinense*, has a wide distribution in India, Annam, China, Japan, Corea, and in Mauritius.

3. *Distomum sinense.* — A lanceolate-shaped worm, narrowed in front, but widening behind until near the posterior extremity. It measures 22 mm. by 7 mm. in its widest part. The surface is smooth, and in appearance the parasite is of a fleshy colour and almost transparent. The ova are gourd-shaped, measuring 32 $\mu$ by 16 $\mu$, of a dark appearance, with a distinct operculum at the broader end. The embryo is a ciliated organism.

4. *Distomum conjunctum* is characterised by the surface of the body being covered by fine spines. The parasite is wide posteriorly, 2·5 mm., but tapers towards the head; in length it measures from 9 to 12 mm. The ova are operculated, shaped like a Florence flask, measuring 34 $\mu$ by 19 $\mu$. So far, this parasite has only been found in a few natives of India.

*Distomum hæmatobium.*—The *Bilharzia hæmatobia* is met with in the branches of the portal vein within the liver, and its eggs may be found embedded in the liver tissues. Although of classical interest, the presence of the bilharzia in the liver or portal vein is attended by no known clinical or pathological features. The clinical manifestations of this parasite are almost solely confined to the urinary passages, and it is in connection with these organs that a detailed account of this interesting parasite is to be found.

Besides the distoma other parasites are met with.

5. *Pentastomum constrictum.* — In the liver and lungs of African negroes cysts have been found to contain a large whitish-coloured parasite, measuring 1 to 1½ inch in length and ¼ inch in breadth. The parasite is ringed, rounded, terminates abruptly behind, but anteriorly exhibits hooks on either side of the oval apertures arranged in two pairs; and on the dorsum of the rings, which in a fully-developed *Pentastomum constrictum* number twenty-three, are fine spinous-looking processes.

6. *Ascaris lumbricoides* find their way into the biliary passages and may even become embedded in the liver. They may be met with singly or in considerable numbers. The entrance of so large a worm into the biliary passages from the intestine is rather difficult to account for, and it has been asserted by some that it is only when the bile ducts are dilated by biliary calculi, by hydatids, or by other causes that the round worm can gain entrance. This contention, however, is refuted by the fact that the worm has been found in the bile ducts even of children.

*The clinical and pathological* effects of the presence of parasites in the biliary passage vary with the parasite and with the numbers which infest the region. We have but few clinical records of the signs and symptoms associated with fluke worms in the liver. Pain in the hepatic region, continuing it may be many months; diarrhœa of long standing; vomiting and occasional hæmatemesis; jaundice, slight or pronounced, may be present; convulsions, especially in children, and delirium in old persons, syncope or coma, hysterical attacks, sudden accessions of fever, abdominal distension, and peritonitis are amongst the more prominent

features of a condition which, unless the parasite itself or the eggs are met with in the vomit or in the stools, is well-nigh impossible to assign to the proper cause.

The changes induced in the biliary passages vary. Occasionally the ducts are dilated, paunched, inflamed, and even purulent. The *Distomum sinense* and the changes it causes in the biliary passages have been most exactly recorded. Baelz has recorded that in certain Japanese villages 20 per cent of the inhabitants suffered from this parasite. In these persons the distomum was found in the gall-bladder, the hepatic and bile ducts in great numbers; the passages become dilated and paunched, and in the paunches the parasites thrive in swarms. The liver and spleen are recorded by Baelz to be enlarged, and the mucous surfaces of the gut in a state of chronic catarrh. In the liver the parts in contact with the main branches of the hepatic ducts atrophy; anæmia, anasarca, and a cachectic state supervene, but it may be only after years of gradual decline.

The *Pentastomum constrictum* causes, according to Aitken, cysts in the liver within which the parasite lies coiled up. When the cysts occupy the surface of the liver a local perihepatitis may be set up, and it is possible to feel the cysts as nodules upon the liver surface.

*Treatment.*—When the ova of any of the liver parasites appear in the stool or in the fæces attempts may be made by anthelmintics to dislodge the parasite. In the case of the *Ascaris lumbricoides* this may be effected by santonine, but no specific means is known whereby the liver-flukes may be dislodged.

### Hydatids of the Liver

The hydatids met with in the human liver differ in no essential point from those met with elsewhere in the body. In England and Wales during ten years (1871-1880) 436 deaths were returned as due to hydatid disease, representing 1 death out of 11,876 deaths during that period. In British India, from 1870 to 1879, 8 cases occurred in soldiers belonging to the European army, whilst in the native army no cases of hydatids were met with. In the Punjab, 4 cases of hydatids were diagnosed in natives in nineteen years' observation at Lahore, out of a total of 25,579 in-patients. In Hong-Kong the writer never met with a case of hydatids in Chinese during nine years' hospital experience, and in Southern China generally the disease is unknown. It is by the dog as the primary host and sheep as intermediary hosts that the life cycle of the echinococcus is chiefly maintained, and in Southern China there are no sheep.

*Site.*—Hydatids are much more frequently met with in the right lobe of the liver than in the left. There has been no explanation offered of this seeming idiosyncrasy, but, as pointed out by the writer, it may be accounted for by the mistaken notion that the right lobe is five or six times larger than the left.

*Signs and Symptoms.*—The physical signs of a hydatid hepatic tumour wholly depend on its size and situation; when the tumour is small it may give rise to no symptoms or signs, but when its dimensions are considerable the local indications will vary according as the tumour is within the liver, upon the upper aspect of the liver, or attached to its under surface, *i.e.* according as the hydatid is *supra*hepatic, *intra*hepatic, or *sub*hepatic. The intrahepatic type, as being the most common, will be taken as the basis for description. An hydatid tumour of the liver may remain latent, that is to say, the tumour even when of fair size may occupy the liver without giving rise to either pain or inconvenience. The detection, post-mortem, of hydatids previously unsuspected is one of the chief characteristics of the disease.

The presence of a hydatid tumour may be made manifest by the patient accidentally noticing an enlargement in the hepatic region, by local discomfort or ascites, jaundice or biliary colic may develop, and during examination of the liver the physician may recognise an hydatid tumour as the origin of any of these conditions. Marked pain is an uncommon symptom. The usual complaint is that of a feeling of weight or dragging extending from the right hypochondrium upwards towards the shoulder, or there may be some difficulty in breathing or cardiac distress. Escape of the contents of the hydatid into a bile duct or the peritoneal cavity causes characteristic attacks of pain peculiar to each.

*Clinical Characters of the Tumour.*—The hydatid when of moderate dimensions is usually globular, but when it attains large proportions the outline will be modified by the resistance met with as it encroaches on neighbouring viscera and structures. The surface of the tumour is smooth and rounded in outline; very rarely is the tumour of lobulated character. On palpation there is a sensation of elasticity; fluctuation is to be felt when the tumour has thinned out the liver tissue and approached the wall of the abdomen. The hydatid thrill, fremitus, or vibration is but seldom present, and is not conclusive proof that the tumour is hydatid in origin; but as hydatids are by far the most common hepatic tumours of cystic character, the thrill when it can be felt is of some value in diagnosis.

*Constitutional symptoms* set up by the presence of even a very large uncomplicated hydatid tumour are seldom in evidence. As a rule there is no interference with the general health, the temperature is not altered, and except for pressure on neighbouring organs there are no signs or symptoms indicative of the ailment beyond the local evidences. If the pressure is upwards, an irritating cough may

be complained of; if the stomach is encroached on, nausea and vomiting may be present; and cardiac distress may result from direct or indirect pressure on the heart. When the tumour progresses downward into the abdominal cavity it may attain a large size, may well-nigh fill the abdomen, and compression of the veins may induce ascites and œdema of the lower limbs. Venous obstruction and its results are, however, uncommon sequelæ of even large hepatic hydatid tumours. Only when the splenic vein is compressed is the spleen likely to be swollen. The ureter may be compressed and the renal vein may likewise be narrowed, and the urine may in consequence become albuminous. Jaundice may arise from pressure upon the obstruction of the ductus communis choledochus, or by the escape of echinococci into the bile ducts; jaundice is occasionally due to catarrh of the biliary passages.

*Course and Termination of the Disease.*—I. *Rupture of the sac* is by far the most common termination when the disease is allowed to run its course without treatment. The rupture may occur spontaneously, but more often it is consequent upon injury of severe muscular effort. The directions in which the tumour ruptures have been summarised by Neisser as the result of 131 observations:—

| | | |
|---|---|---|
| Rupture into the lung and pleural cavity, | 47 | cases. |
| ,, ,, stomach and intestines, | 46 | ,, |
| ,, ,, peritoneal cavity, . | 16 | ,, |
| ,, through the abdominal wall, . | 15 | ,, |
| ,, into the urinary passages, . | 7 | ,, |

Rupture into the *Bronchial Tubes and Pleural Cavity.*—Naturally, it is into the right side of the chest that the contents of the cyst find exit. When the cyst perforates the diaphragm, previous to the formation of pleuritic adhesions, a fatal pleurisy is the usual result; but when the hydatid has set up adhesive inflammation in the pleura the cystic contents find their way into the pulmonary tissue, and, pressing towards the point of least resistance, may gain the channel of a bronchus and be got rid of in the sputum. This mode of exit may be followed by speedy and complete recovery, or the long-standing suppuration induced leads to exhaustion, induration of the lung, general wasting, and may end fatally or in recovery after several years of profuse discharge. In such cases the sputum contains hooklets, and it may be scolices, liver pus, shreds of membrane, occasionally stained by bile. Sudden death may follow rupture into the lung; the bronchial tubes of both sides may be blocked by the profusion of the discharge into the air passages, and suffocation ensue. Long-standing suppuration may lead to consolidation or gangrene of one or both lungs. Thus exit by way of the bronchial passages is an undesirable result to wait for, and a surgical operation, when once the hydatid has commenced to discharge through the lung, is not attended by promising results.

Rupture into the *Intestine.*—The cystic contents do not all escape in one stool, but trickle into the intestinal canal rather slowly. Rupture into the intestine does not necessarily imply that the patient recovers. Death may result from a general peritonitis, or from hepatic abscess, infection from the intestine reaching the cavity of the emptied cyst. Rupture into the *stomach* allows of the escape of the hydatid contents by vomiting. Rupture into the *peritoneal cavity* is usually followed by death, which follows in a few hours from shock, or may be deferred for a few days, during which severe peritonitis takes place. Should the hydatid suppurate, fatal peritonitis may be set up without rupture. Rupture through the *abdominal walls*: the point of exit is usually the right upper quarter of the abdomen. Rupture into the *urinary passages*: there is no direct proof that such an event has ever occurred. Rupture into the *biliary ducts* may be followed by a spontaneous cure of the disease, or by severe inflammation of the sac and suppuration. The contents of the tumour may pass along the common bile duct to the duodenum, or the calibre of the bile duct proves insufficient to transmit the hydatids, and blocking of the channel gives rise to jaundice. The passage of a small cyst down the common bile duct is frequently attended by symptoms of biliary colic. It is possible that the spontaneous cure of unobserved hydatid of the liver takes place more often by this channel than by any other. Rupture into the *pericardium*: a fatal result has been the invariable consequence. Rupture into the *inferior vena cava* or *portal vein*: the former leads to sudden death from pulmonary embolism, the latter to scattered abscesses in the liver. *Multiple channels of exit*: not infrequently the hydatid contents may escape from the liver by two or more channels simultaneously.

II. *Suppuration* of the sac may be "spontaneous," may be the result of surgical treatment by puncture, etc., or may arise from bile gaining access to the cavity of the cyst. A blow may determine suppuration, or it may follow rupture of the sac into a neighbouring viscus. The subsequent history of a hydatid cyst after suppuration is identical with that of a liver abscess (*vide* Liver Abscess).

III. Other fatal modes of termination may be due to (1) pressure on neighbouring organs, (2) exhaustion consequent upon any one of the above conditions, (3) implication of the liver to so great an extent that it becomes totally unfitted to perform its function and carry on the work of the economy.

IV. Spontaneous Cure.—The mother cyst slowly contracts and shrivels, and, along with the contents, becomes replaced by a putty-like

mass, which when once seen can never be forgotten. Although putty-like in consistence in parts, it is of a whitish colour, pearly white towards the interior, a duller shade pervading in the peripheral portions; it resembles a mass of wet kaolin or china clay of jelly-like consistence. Plates of calcareous material may be found in the laminated mass, and pieces of the wall of the mother cyst may be found here and there.

*Echinococcus multilocularis* is described in vol. iv. pp. 261-264.

*Diagnosis.*—By a process of exclusion other ailments can be eliminated, and by drawing off and examining the contents a positive conclusion may be arrived at. Hydatids in the *suprahepatic* region of the liver have to be diagnosed from suprahepatic abscesses, from pleuritic effusion, or from hydatids of the base of the right lung. *Intrahepatic* hydatid growths may be mistaken for intrahepatic abscess or for malignant tumours. In fatty, amyloid, and syphilitic enlargements of the liver, and in hypertrophic cirrhosis, the history will serve to eliminate any possibility of confusion. Hydatids in the *subhepatic* region have to be diagnosed from subhepatic abscess, enlarged gall-bladder, hydronephrosis, renal or ovarian cysts, aneurysm, and phantom tumour.

*Treatment.*—Although hydatids of the liver may disappear by "spontaneous cure," may find exit by several channels, and the patient recover, no medical practitioner is justified in waiting for such a result. The modern treatment consists in aspiration, incision, suture of the sac to the margins of a parietal incision and clearing out the contents, or complete extirpation of the tumour.

*Aspiration* of the contents of the hydatid can be used for both diagnostic and curative purposes, and puncture by the fine needle of an aspirator is attended by infinitesimal danger. The fluid removed thereby may serve to establish the character of the tumour, and when a small quantity of the fluid is withdrawn the tumour may shrink and the whole cyst disappear. The dangers of tapping a hydatid are:—(1) Escape of the contents into the peritoneal cavity. That the slow and it may be intermittent escape of a small quantity of hydatid fluid into the peritoneum causes untoward symptoms is improbable, and treatment by aspiration alone may still find a place in surgery. (2) Suppuration may be induced in the hydatid; this is a real danger. The removal of the fluid leaves behind a dead mass within the liver, which may at any moment become septic.

*Manson's Method* of treating Liver Abscess applied to Hydatids.—The operation is fully described under Liver Abscess, and the method recommends itself.

*Laparotomy and Removal of the Contents of Cyst Walls.*—In 1871, Lindemann improved upon Volkmann's method by stitching the margins of the wound in the liver to the wound in the abdominal wall, and all subsequent methods of operation have been mere modifications of Lindemann's principle. At the present time the plan recommended by Godlee is most in vogue.

*Operation.*—A vertical incision three or four inches in length is made over or just above the most prominent part of the tumour, and it is carried down to the peritoneum. All bleeding is arrested, and the peritoneum is then incised for almost one inch and the finger introduced. The presence of adhesions is felt for. Where there are no adhesions the escape of hydatid contents into the peritoneal cavity can be guarded against in one of three ways:—The opening of the cyst deferred for a few days or until sufficiently firm and extensive adhesions are apparent. Should suppuration have occurred the cyst must be incised at once, and every surgeon prefers to open the tumour, whatever its state, at one sitting. (2) By packing the peritoneal surface with sterilised sponges or gauze so thoroughly that all passage into the peritoneum is blocked. The cyst may then be incised sufficiently to allow the contents to escape. When the first rush of fluid has subsided a small sterile sponge with string attached is passed into the cavity of the sac. It there expands, and on pulling the string the sponge blocks the exit of fluid until the margins of the wound in the liver or adventitious capsule are stitched to the wound in the abdominal wall. The wound in the cyst wall is hereafter enlarged, and the finger, or a sponge on a holder, may be introduced to loosen adhering brood capsules and daughter cysts. (3) When practicable, the liver may be stitched to the parietal peritoneal wound by a double row of sutures after the method recommended by Mr. Godlee for liver abscess. The further treatment varies; in the majority of instances the mother cyst is removed as well as the contents, and a large drainage-tube introduced. Bond of Leicester, in 1891, introduced the plan of removing the parasite, mopping out the cavity left, stitching (or leaving open) the wound in the liver, and sewing up the wound in the abdominal wall after the manner of an ovariotomy wound. Bond met with excellent results, and his method is likely to be followed.

The results of operations on Lindemann's principle show the percentage of recoveries to be about 90 per cent.

**Liver of Sulphur.** — Sulphurated potash, consisting of potassium thiosulphate, potassium trisulphide, and some potassium carbonate.

**Lividity.**—The state of being discoloured or livid; livedo or livor; as in such expressions as cadaveric lividity, the bluish discoloration

of the skin of a corpse, due to gravitation of the blood to the most dependent parts. *See* MEDICINE, FORENSIC (*Signs of Death, Post-mortem Lividity*); POST-MORTEM METHODS (*External Examination, Post-mortem Lividity*).

**"Living Skeletons."**—The name given to individuals of an extreme degree of thinness, who are not unfrequently exhibited at shows; thus, Claude Ambroise Seurat was shown in London in 1826 as the "Anatomie Vivante" or "Living Skeleton," and Calvin Edson, a native of the United States, was exhibited in 1830. There is marked atrophy of the subcutaneous fat and, to some extent, also of the muscular system.

**Lixiviation.**—The formation of a lye (Lat. *lixivia*), a lye being a solution formed by the extraction with water of the ashes of burnt substances; it is a process employed in pharmacy. Thus, in the making of sodium carbonate, sodium sulphate is roasted with limestone and coal, and a mass called *black ash*, consisting of sodium carbonate and calcium sulphide, results; from this mass the carbonate is obtained by washing or lixiviation, the sulphide of calcium being left behind.

**Llandrindod.** *See* BALNEOLOGY (*Great Britain*); MINERAL WATERS (*Muriated Sulphated Waters*).

**Llandudno.** *See* THERAPEUTICS, HEALTH RESORTS (*English, Wales*).

**Llangammarch.** *See* BALNEOLOGY (*Great Britain*).

**Llanwrtyd.** *See* BALNEOLOGY (*Great Britain*); MINERAL WATERS (*Muriated Saline*).

**Lobar.**—Belonging or referring to a lobe, as distinguished from *lobular*, which means referring to a lobule, *e.g.* lobar pneumonia, in which the inflammatory process involves a lobe or even a whole lung, croupous or fibrinous pneumonia.

**Lobe or Lobus.**—A segment of an organ, usually rounded in shape, and separated from neighbouring parts by grooves or fissures, *e.g.* the lobes of the brain, of the liver, of the lung, of the prostate, and of the thyroid gland.

**Lobelia.**—Lobelia is composed of the dried flowering herb, *Lobelia inflata*, the constituents of which are the alkaloid, *lobeline* or *lobelina*, *lobelic acid*, and *inflatine*; the official preparation is the ethereal tincture (*Tinctura Lobeliæ Ætherea*), the dose of which is 5 to 15 m.; and the drug is now used chiefly for asthma, for it has a depressing effect on the heart which limits its range of applicability.

**Lobeline.**—The volatile, fluid alkaloid of *Lobelia inflata* (*q.v.*), with an undetermined chemical constitution. *See* ALKALOIDS.

**Lobengulism.**—A morbid condition in which there is loss of sexual power, associated with a marked deposition of subcutaneous fat; it may affect both sexes, causing enlargement of the mammary glands in men and a form of spurious hermaphroditism, and cessation of the menstruation in women; lipomatosis universalis asexualis.

**Lobstein's Cancer.**—Retroperitoneal sarcoma; it is with difficulty differentiated from tumour of the kidney.

**Lobstein's Disease.**—A form of fragilitas ossium or osteopsathyrosis. *See* FRAGILITAS OSSIUM.

**Lobster-claw Deformity.**—A defect of the digits in which two only are left (*bidactyly*), usually the thumb or great toe and the little finger or little toe, giving the appearance of two claws.

**Lobular.**—Relating to a lobule, *e.g. lobular pneumonia* or bronchopneumonia as distinguished from lobar pneumonia or croupous pneumonia.

**Lobule or Lobulus.**—A segment or subdivision of a lobe.

**Local Anæsthesia.**—The production of anæsthesia in a limited or localised part of the body, without the loss of general consciousness. *See* ANÆSTHETICS (*Local Anœsthesia*).

**Localisation.**—The process by which a particular physiological or pathological action is found to originate or be situated in any particular part, especially of the nervous system. *See* BRAIN, PHYSIOLOGY OF (*Functions of the Cerebral Cortex*); PHYSIOLOGY, NERVOUS SYSTEM (*Localisation of Functions*).

**Locarno.** *See* THERAPEUTICS, HEALTH RESORTS (*Italian Lakes*).

**Lochia.**—The discharges from the genital tract (from uterus to vulva) which follow the birth of the child and last about two weeks (Gr. λοχεύω, I bring to the birth); during the first few days they are named *lochia rubra*, thereafter *lochia serosa*, and finally *lochia alba*, according to the colour changes which take place in them. *See* PUERPERIUM, PHYSIOLOGY OF (*Lochia*); PUERPERIUM, PATHOLOGY OF (*Puerperal Infection, Symptomatology*).

**Lochio- or Locho-.**—In compound words *lochio-* or *locho-* means relating to the lochia, *e.g. lochiometra*, retention of the lochial discharges in the uterus; *lochiopyra*, puerperal fever; *lochiorrhagia*, an excessive flow of the lochia; *lochometritis*, puerperal metritis; and *lochoperitonitis*, puerperal peritonitis.

**Lock-Finger.** — An affection of the fingers, supposed to be due to a fibroma in the metacarpo-phalangeal joint, by which they are fixed in the attitude of flexion.

**Lockhart-Clarke's Column.**—A column of nerve-cells lying on the mesial aspect of the posterior horn of the spinal cord. *See* PHYSIOLOGY, NERVOUS SYSTEM (*Spinal Cord, Structure*).

**Locking of Heads.**—A complication of twin labours, due to the hitching of the chin of one twin (being born by the breech) over that of the other twin (coming by the head), or to the hitching of the one occiput under the other. *See* LABOUR IN MULTIPLE PREGNANCY (*Interlocked Twins*).

**Lockjaw.** *See* TETANUS.

**Lock-Spasm.**—A firm contraction of the fingers upon any body, such as a pen, *e.g.* in writer's cramp. *See* NEUROSES, OCCUPATION (*Writing*).

**Lockwood's Ligament.**—The suspensory ligament of the eyeball.

**Lockwood's Method.** *See* HERNIA (*Radical Cure, Femoral Variety*).

**Loco Deflector.**—The bend put upon a soil-pipe at the foot of the house, so that the flow of water into the larger drain may not be checked; it is of importance in the proper drainage of a house.

**Loco Disease.**—A disease of cattle and horses due to their eating the plants known as *loco weed* (poisonous leguminous plants), and characterised by staggering gait, emaciation, loss of appetite, etc.

**Locomotor Ataxia.** *See* TABES DORSALIS; *see also* BALNEOLOGY (*Balneo-Therapeutics in Diseases of the Nervous System*); BRAIN CYSTS (*Cystic Degeneration in Locomotor Ataxia*); DEFORMITIES (*Cerebral and Spinal Paralysis, Locomotor Ataxy*); FRAGILITAS OSSIUM; JOINTS, DISEASES OF (*Associated with Lesions of the Nervous System*); OSTEO-ARTHROPATHIES (*Osteopathies*); PURPURA (*Symptomatic, Nervous*); ULCERS AND ULCERATION (*Perforating Ulcer of the Foot*).

**Loculus.**—A small space, *e.g.* of a gland or cystic tumour.

**Locus.**—A place; a name given in Anatomy to various spots in such organs as the brain, *e.g.* the *locus cinereus* in the fourth ventricle, the *locus perforatus* (*anticus* or *posticus*) at the base of the brain, etc. The term *locus minoris resistentiæ* is given to that part of the body which from some cause is enfeebled and less able to resist the onslaughts of disease.

**Lodging-Houses.** *See also* VENTILATION AND WARMING, etc.—A lodging-house in Sanitary Law is defined as "a house or part of a house let in lodgings or occupied by members of more than one family"; the hygiene of such dwellings is regulated and enforced by the Public Health Act of 1875, by the Public Health Acts Amendment Act of 1890, by model by-laws and memorandum of the Local Government Board, and by the Public Health (Scotland) Act of 1897. Common lodging-houses must be registered; they must be open to the inspection at any time of the Local Authority. A space of not less than 300 cubic feet for each adult living therein must be provided; the walls and ceiling must be limewashed twice a year (in April and October), and the occurrence of a case of infectious disease must be immediately reported to the Medical Officer of Health.

**Loebisch's Formula.**—If the last two figures of the specific gravity of the urine be multiplied by 2·2, the product indicates the number of grammes of solids in 1000 cc. of urine.

**Loeche-les-Bains.** *See* BALNEOLOGY (*Switzerland, Indifferent Thermal Waters*); MINERAL WATERS (*Thermal*).

**Loeffler's Bacillus.** *See* DIPHTHERIA (*Morbid Anatomy and Pathology*); POST-MORTEM METHODS (*Bacteriological Investigations*); TEETH (*General Bacteriology of Mouth*). *Loeffleria* is the name which has been given to a disease in which Loeffler's bacillus is present but in which diphtheritic symptoms are absent.

**Loeffler's Blood-Serum Mixture.** — A culture-medium, consisting of neutral meat-infusion bouillon containing 1 per cent. of glucose and three parts of blood-serum.

**Lochlein's Diameter.**—The length of a line drawn from the centre of the subpubic ligament to the antero-superior angle of the great sacro-sciatic foramen.

**Loewenthal's Reaction.** — The name given to the agglutination of spirochæte obermeieri by the blood-serum of a person suffering from relapsing fever. *See* RELAPSING FEVER.

**Loganin.** —A glucoside got from the seeds and fruit of *Strychnos nux vomica*; it has the formula $C_{25}H_{34}O_{14}$, and when boiled with dilute sulphuric acid yields *glucose* and *loganetin*.

**Logo-.** — In compound words *logo-* (Gr. λογος, a word) signifies relating to speech or words; thus *logamnesia* is word-deafness, *logagnosia* is word-blindness, *logoneurosis* is a neurotic affection associated with defect in speech, *logoplegia* is the loss of power of articulating words, and *logorrhœa* is morbid loquacity.

**Logomania or Logomonomania.** — Insanity associated with great talkativeness or loquacity.

**Logwood.** *See* HÆMATOXYLI LIGNUM.

**Loimology.**—The science of the study of infectious maladies (Gr. λοιμός, pestilence).

**Loin.**—The part of the body situated (on both sides) between the false ribs and the iliac bone. *See* LUMBAR REGION.

**Loka.**—A watering-place in Sweden where there are mud-baths (*see* BALNEOLOGY, *Sweden*); also an arrow-poison, probably of the nature of nux vomica.

**Lolium Temulentum.** — Darnel seeds, which, if present in bread, may cause poisonous symptoms (vertigo, delirium, and other nervous symptoms).

**Longevity.** *See* VITAL STATISTICS (*Determination of Longevity*); SENILE INSANITY (*Dementia, the "fatal psychical heritage of Longevity"*).

**"Longings."**—The disordered appetite often associated with pregnancy in which the patient desires to eat unusual or unsuitable things; pika.

**Longissimus.** — A name given to various muscles characterised by their great length, *e.g.* the longissimus dorsi, and the longissimus cervicis (or trachelo-mastoid) muscles.

**Longus.** — A name applied to many muscles of the body to distinguish them from neighbouring or similarly acting muscles of less extent, *e.g.* the supinator radii longus, the palmaris longus, and the flexor longus pollicis, etc.

**Loose Bodies.** *See* ANKLE-JOINT, REGION OF, DISEASES (*Loose Bodies*); BURSÆ, INJURIES AND DISEASES OF (*Tuberculous Bursitis*); JOINTS, DISEASES OF (*Loose Bodies*); KNEE-JOINT, DISEASES OF (*Tuberculous Affections, Diagnosis*).

**Loose Shoulders.**—The name given to an abnormal mobility of the scapulæ seen in certain muscular dystrophies, *e.g.* in Erb's progressive muscular dystrophy.

**Lopez Root.**—The dried root-bark of *Toddalia aculeata*; it is used in India and the Colonies, and is described 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.); it is a vegetable bitter, and is used for the same purposes as other bitters (to increase the appetite, aid the digestion, etc.).

**Lopho-.**—In compound words *lopho-* (Gr. λόφος, a ridge or tuft) means tuft-like, *e.g.* in the group of bacteria called *lophotrichous* (having cilia in tufts) and in *lophocomous* (having the hair in tufts).

**Lordosis.**—Spinal curvature in which the convexity is directed forwards; it is usually an exaggeration of the normal lumbar curve. *See* CRETINISM (*In Adult Age, Description*); DEFORMITIES (*Congenital Dislocation of the Hip, Diagnosis*); LABOUR, PRECIPITATE AND PROLONGED (*Pelvic Deformities, Kyphotic Pelvis*); PARALYSIS (*Spastic, Clinical Aspect*); SPINE, SURGICAL AFFECTIONS OF (*Lordosis*).

**Lorenz Operation.** — A method ("bloodless") of treating congenital dislocation of the hip, in which the dislocation is forcibly reduced and the head of the femur is maintained in contact with the ilium till a deep enough acetabulum is formed to keep it permanently in position. *See* DEFORMITIES (*Congenital Dislocation of the Hip, Reduction*).

**Loreta's Operation.**—The forcible dilatation of the pyloric outlet of the stomach (by fingers or dilating instruments); pylorodiosis. *See* STOMACH, SURGICAL (*Operations on the Stomach*).

**Loretine.** — An antiseptic substance ($C_9H_6IO_4SN$), a derivative of quinoline; it is employed in aqueous solution (1 in 1000) or as a dusting powder.

**Los Angeles.** *See* THERAPEUTICS, HEALTH RESORTS (*American*).

**Losophan.**—A proprietary preparation (triiodometacresol, $C_7H_5I_3O$), used in the treatment of skin diseases; it contains 80 per cent of iodine.

**Lota Vulgaris.** *See* SNAKE-BITES AND POISONOUS FISHES (*Fish*).

**Lotio.**—A lotion or wash, for external application; there are two official *lotiones*, the lotio hydrargyri flava and the lotio hydrargyri nigra.

**Lotio Rubra.**—Red lotion; a solution of the sulphate of zinc coloured with red compound tincture of lavender, acting as an astringent wash in cases of ulceration, etc.

**Lotio Spiritus.**—A wash or lotion used in cases of bruise or sprain, and consisting of 4 parts of rectified spirit to 1 of water.

**Louis's Law.**—The statement that the lungs always contain tubercles if there is tuberculosis of any other part of the body.

**Louping Ill.**—A peculiar disease met with in animals (sheep), which is characterised by springing or leaping movements during progression; it is said to be due to a bacillus.

**Louvres.**—In the ventilation of rooms with windows, fixed or movable louvres of glass may be placed in the panes of the upper sash so as to allow the air to enter.

**Lowcock's Filter.**—A bacteriological filter consisting of sand and gravel, fitted with air-pipes, used in the disposal of sewage.

**Lower Extremity.** *See* DEFORMITIES (*Lower Extremity*); FRACTURES (*Special, Lower Extremities*).

**Lower Jaw.** *See* MOUTH, DISEASES AND INJURIES OF THE JAW (*Mandible*).

**Lower, Tubercle of.**—A small projection on the wall of the right auricle of the heart between the openings of the superior and inferior venæ cavæ.

**Lower Uterine Segment.** *See* LABOUR, PHYSIOLOGY OF (*Factors of Labour, Primary Powers*); LABOUR, INJURIES TO THE GENERATIVE ORGANS (*Rupture of Uterus*); PREGNANCY, HÆMORRHAGE (*Placenta Prævia or Unavoidable Hæmorrhage*).

**Low Fever.** *See* TROPICS, UNCLASSED FEVERS OF THE ("*Low*" *Fever*).

**Lozenges.**—Solid medicinal preparations made of gum acacia and various flavouring substances (black-currant paste, rose water, or tincture of tolu), containing a drug such as tannic acid, benzoic acid, etc.; they melt slowly in the mouth when sucked. *See* PRESCRIBING.

**Lubarsch's Crystals.** — Peculiar crystals found in the epithelial cells of the testicle after death.

**Lucan.** *See* BALNEOLOGY (*Great Britain, Ireland*).

**Lucas' Sign.**—Distension of the abdomen in rickets.

**Lucca.** *See* BALNEOLOGY (*Italy*); MINERAL WATERS (*Calcareous*).

**Lucid Interval.**—A temporary return to mental health seen in certain cases of insanity and delirium.

**Lucifer Match-Making.** *See* TRADES, DANGEROUS (*Phosphorus Poisoning*).

**Lucilia Hominivora.** *See* NOSE, FOREIGN BODIES (*Parasites of the Nose*).

**Lucilia Macellaria.** — The screw worm, an insect whose larvæ may be found in nose, vagina, and in wounds. *See* MYIASIS; NOSE, FOREIGN BODIES (*Parasites*); etc.

**Lucina.**—The goddess (Juno or Diana) who, in the Roman Mythology, presided over childbirth; *floreat Lucina* is a toast sometimes given at convivial meetings where obstetricians are in the majority.

**Ludwig's Angina.** *See* ANGINA LUDOVICI; NECK, REGION OF (*Inflammatory Affections*); PHARYNX, ACUTE PHARYNGITIS; SUPPURATION (*Angina Ludovici*); TONGUE.

**Ludwig-Salkowski's Method.**—A quantitative test for uric acid and its salts. *See* URIC ACID.

**Ludwig's Theory.**—A theory of the mechanism of renal secretion. *See* KIDNEY, PHYSIOLOGY OF (*Mechanism of Renal Secretion*).

**Lueck's Reaction.**—A test for hippuric acid, depending upon the intense odour of nitro-benzol.

**Lues.**—A plague, pestilence, or epidemic, now used in a more restricted sense and usually with a qualifying adjective; thus *lues venerea* or *lues celtica* is syphilis, *lues congenita* is congenital syphilis, *lues divina* is epilepsy, and *lues inguinaria* or *lues pestifera* is the plague.

**Lugano.** *See* THERAPEUTICS, HEALTH RESORTS (*Italian Lakes*).

## Lumbago.

INTRODUCTORY . . . . . 527
ANATOMICAL AND PHYSIOLOGICAL . . 527
CLINICAL . . . . . 528
THERAPEUTIC . . . . . 529

*See also* ABDOMINAL ANEURYSM (*Diagnosis, Differential*); GOUT (*Irregular Gout, Muscular and Connective Tissues*); HYDROPATHY (*Rheumatism and Gout*).

LUMBAGO is usually regarded as a manifestation of rheumatism or gout. It is, however, too much the practice to pronounce as rheumatic or gouty any chronic pain of which the nature is obscure or to which no other specific character is assigned. So loosely is the term applied, that it is more than probable that a variety of ailments, with no character in common save that of pain, have been classed under it, and much both of false experience and faulty practice has resulted thereby. However difficult it may be to classify the different conditions which may induce lumbago, it is probably better to leave their place in classification unassigned, than, by ranging them under a definite head of rheumatism, to beget confusion where clearness and precision are essential both for diagnosis and treatment. It is advisable to refer briefly to some anatomical and physiological facts which must ever be kept in the foreground as constituting the central point in the line of investigation.

*Anatomy and Physiology.*—We are here dealing with the most dependent part of the trunk —that part of the body mainly concerned with the maintenance of the upright posture. For

the due performance of this function we have a series of specially well-developed vertebræ and intervertebral discs, clothed by soft tissues to a degree befitting the importance of function subserved. From without inwards the more important soft structures are: (1) the latissimus dorsi, serratus posticus inferior, and internal oblique lying in the posterior layer of lumbar fascia; (2) the erector spinæ, within its own layer of fascia; (3) the quadratus lumborum, also similarly enclosed; (4) the psoas muscle, lying in front of the anterior layer of lumbar fascia, with the lumbar plexus lying in its substance. Particular attention should be paid to the vessels, five in number, which run out behind the quadratus into the space between the transversalis and internal oblique muscles. Capillaries and veins correspond. The lymphatic system is of even greater importance. For an account of the vessels and glands concerned the reader is referred to the section on "Lymphatic System" (*vide* vol. vi.). The difficulties attendant on the investigation of the state of the lymphatic vessels and glands in this region have, I fear, led us to neglect thinking of them as factors of much importance. It is more than probable that, if we only had opportunities of investigating the state of the glands in and about the lumbar area, much light would be thrown on the etiology of the local morbid conditions. The nerves involved are derived from the lumbar plexus, the anterior divisions being intimately associated with the lumbar ganglia of the sympathetic system.

The pain of lumbago must be connected with one or other or a combination of the following: muscles, skin, connective tissue, vessels including capillaries and lymphatics, and nerves. Each and all of them must be considered so as to have an adequate conception of the condition, and before leaving this part of the subject it is well to add:—

1. The *dependent* nature of this region, with its relatively large amount of soft tissues, predisposes to defective drainage, so to speak, of the part.

2. The extent of capillary area in association with it is very considerable.

3. The significance of failing elasticity of adult and later life, with its associated limitation of movement, is of special importance in this region of the body.

*Clinical.* — Lumbago in its simplest form consists of a pain in the small of the back, with diminution in movement, anteriorly and laterally, the pain being aggravated by any movement which brings into play the forementioned muscles. There is no *apparent* constitutional disturbance. The onset of pain may take place after exposure to cold or damp, but frequently no exciting cause can be assigned. The duration is a few days or more; in some instances the pain may remain in a subacute or chronic stage for months. A more adequate account of the disease is found in the description of a regular acute attack of lumbago. The following notes are made from the case of a young medical man under the writer's care, and is the most striking example of the disease that has come under his observation. It illustrates better than any general description the severity and importance of the condition:—

When about sixteen years of age (now aged thirty-two) patient had his first attack of lumbago, which laid him up for at least several days. Had been much exposed to wet and cold the previous night. Since that time he has had similar attacks, of greater or less severity, up till the present time, on an average about twice a year. The last attack was in January 1900, and was a rather severe one. Its description also applies to other attacks with very slight modifications. For two or three days before the attack patient felt a stiffness in the lumbar muscles. From past experience this was an indication to "walk warily." When bending over the basin in the act of washing his face in the morning, and when in the act of assuming the erect attitude, he was suddenly seized with a very severe pain in the upper lumbar region. The pain was described as "terrible," and of a sharp, stabbing character. He had to seize hold of the back of a chair, and had to be assisted back to bed. He felt as if one of the lumbar vertebræ had been suddenly removed, and that, if he attempted to stand without supporting himself with his hands on the back of a chair, he would have "broken in two." When in bed and at rest the pain persisted, though of a dull aching character; when any movement was attempted, the severe lancinating pain recurred. On coughing, but especially on sneezing, the pain was excruciating. A dull, aching pain was felt continuously for first five or six days. After the first day or two the pain began to move downwards, and at the end of a week it was most severe in the sacral and right gluteal region; a few days later it was felt in the back of the right thigh. (There was considerable pain caused by the movements of respiration during this attack, but this had never been experienced before—possibly due to implication of the sheath of the quadratus lumborum muscle or some of the abdominal muscles.) In every previous attack the pain gradually moved downwards, and was usually last felt in the back of the right thigh. This was not in the least affected by pressure over the course of the sciatic nerve, and the pain was not increased at all by the ordinary tests for sciatica. During the whole period in which patient was confined to bed the general health was quite good, the appetite was unimpaired. He got out of bed on the tenth day, and at the end of a fortnight was able to resume work, but an aching stiffness and weakness continued for

another week. On several occasions patient has experienced severe pain in the abdominal, pectoral, and neck muscles of a nature similar to the above, but less in degree. The hereditary predisposition in this case was very pronounced. The father and mother have both suffered from lumbago as far back as patient can remember, and a brother had a very severe attack when æt. 18.

This case was a little exceptional in respect of the absence, so far as noted, of definite constitutional disturbance. Some derangement of appetite, furred tongue, and febrile urine are very common; and it is probable that if the state of the bowels was very closely inquired into both previous to and during an attack, some evidence would be forthcoming of constipation and increased fœtor of evacuations, pointing to a source of toxic poisoning. It is one of the striking features of this disease, that it may be present under circumstances apparently least favourable to its development. Thus it is not infrequently met with after active exercise, such as a round of golf, especially in subjects of a sedentary occupation. (To prevent any misconception as to the writer's views on the value of that exercise, he might here state that in such a case the best plan of treatment is to recommend another round, followed by a hot bath.) Again it is frequently seen in middle-aged and elderly gentlemen in the autumn, after the summer holiday; in this respect it resembles some other conditions.

*Etiology.*—What is the etiology of this condition? In the absence of definite pathological data to go upon we can only theorise. The views commonly expressed are as follows:—

(*a*) Inflammatory changes in the muscles or intermuscular connective tissue.

(*b*) Neuralgiac affections of the nerve terminations in the muscles.

(*c*) Actual rupture of muscle-fibres at the seat of attachment to the iliac crest.

It is unnecessary to discuss these views in detail. Personally I am inclined to the view that a thrombosis of the arterioles and capillaries best explains many of the subacute and chronic cases; it probably also plays a part in the more acute cases. The cause of this is to be sought in the action of some toxic substance which, from reasons already noted, accumulates in that region to an extent sufficient to induce pronounced vascular and probably also muscular and nervous changes. The source of this toxic substance is partly in the alimentary canal and partly in the tissues themselves. The disease is, in short, in most cases merely a local expression of a toxæmia. It is probable that if the blood was carefully examined before, during, and after a paroxysm, much important evidence in favour of this view would be obtained; the well-known facts of its occurrence after exposure to cold, fatigue, and the like, are also in its favour.

*Differential Diagnosis.*—Very little need be said under this head. Renal conditions must be differentiated; and this, in doubtful cases, can only be done after a systematic examination of centrifuged urine. We must also bear in mind that this region is the favourite site for the initial pain of some infectious disease—*e.g.* influenza, smallpox, etc. (This point is of special interest in view of the theory suggested as to the toxic nature of this condition.) In women, coxalgia may have to be eliminated before any conclusion be arrived at. As a rule, however, the diagnosis is perfectly apparent.

*Treatment.*—Whether that view be correct or not, there is no doubt that it constitutes the safest and best guide to treatment. The main principles of treatment are:—

1. Promote depletion by (*a*) a brisk purgative, and (*b*) by diaphoresis, preferably by a hot bath, Turkish bath, or the like. The degree of this depends on the type of the individual. Sthenic patients require more powerful measures; in asthenic cases these measures may be advantageously combined with judicious stimulation.

2. Put the muscles out of action by adopting the recumbent posture; soothe the part by the application of heat. The manner of heat is immaterial.

3. The food and drink should be restricted in amount, and of the simplest quality; water-drinking should be encouraged.

4. When the services of a really skilled masseur can be obtained, they are invaluable even in the acute stages. The judicious use of *effleurage*, etc., in combination with heat will usually effect a wonderful relief, even to apparent cure.

In the hands of some authorities various drugs have been found useful, especially salicylate of soda, phenacetin, guaiacum, pot. iodide, etc.; but the writer's experience leads him to lay very little stress on drug treatment of this condition. Similarly, external applications other than those mentioned are, he believes, comparatively inert. Chronic cases should be treated along lines similar to those already laid down. Seek to restore the impeded circulation in the capillaries, and renew the secretory and excretory processes of which they are the agents. Balneological measures are of the first importance, both as prophylactic and curative remedies.

To those who cannot readily obtain the requisite amount of active exercise and fresh air desirable, it is advisable to recommend the daily performance of two or three studied muscular movements—movements calculated to bring into play those muscles whose function, through non-use, is being abused. These movements are best done in the morning, and should be performed slowly, and extend over at least ten to fifteen minutes.

**Lumbar.**—Relating to the lumbar region (*lumbo-* in compound words, such as *lumbo-sacral*).

**Lumbar Abscess.** *See* ABDOMINAL ABSCESS; SPINE, SURGICAL AFFECTIONS OF.

**Lumbar Anæsthesia.** — Spinal Anæsthesia; analgesia produced by the injection of a solution of a drug (cocaine, novocaine, stovaine, etc., usually combined with adrenalin) into the spinal arachnoid in the lumbar region; it has been extensively employed for operations on the abdomen, pelvis, and lower limbs, and has been used to relieve the pains of labour.

**Lumbar Colotomy.** *See* COLOTOMY (*Lumbar*).

**Lumbar Glands.** *See* LYMPHATIC SYSTEM, PHYSIOLOGY AND PATHOLOGY (*Abdomen*); UTERUS, MALIGNANT TUMOURS OF (*Infection of Glands*).

**Lumbar Hernia.** *See* HERNIA (*Lumbar*).

**Lumbar Nephrotomy.** *See* KIDNEY, SURGICAL AFFECTIONS OF (*Operative Procedures*).

**Lumbar Plexus.** *See* SPINE, SURGICAL AFFECTIONS OF (*Lumbar Plexus*).

**Lumbar Puncture.** *See* SPINE, SURGICAL AFFECTIONS (*Lumbar Puncture*); *see also* HYDROCEPHALUS (*Surgical Treatment, Lumbar Puncture*); MENINGITIS, EPIDEMIC CEREBRO-SPINAL (*Diagnosis*).

# INDEX


| | PAGE |
|---|---|
| Inulin | 1 |
| Inunction | 1 |
| Invagination | 1 |
| Invalid Feeding | 1 |
| Invasion | 32 |
| Inversion | 32 |
| Invertase | 32 |
| Invertins | 32 |
| Involucrum | 32 |
| Involution | 32 |
| Iodalbin | 32 |
| Iodic Acid Test | 32 |
| Iodides | 32 |
| Iodine | 32 |
| Iodipin | 34 |
| Iodism | 34 |
| Iodo- | 34 |
| Iododerma | 34 |
| Iodoform | 34 |
| Iodoformin | 35 |
| Iodoformism | 35 |
| Iodol | 35 |
| Iodophilia | 35 |
| Iodothyrin | 35 |
| Ionic Action | 35 |
| Iothion | 35 |
| Ipecacuanha | 35 |
| Ipomœa | 35 |
| Iridalgia | 35 |
| Iridectomy | 35 |
| Irideremia | 35 |
| Iridin | 35 |
| Irido-Choroiditis | 36 |
| Iridocinesis | 36 |
| Irido-Cyclitis | 36 |
| Iridodesis | 36 |
| Iridodialysis | 36 |
| Iridodonesis | 36 |
| Iridoparesis | 36 |
| Iridoplegia | 36 |
| Iridoptosis | 36 |
| Iridotomy | 36 |
| Iris and Ciliary Body | 36 |
| Irish Moss | 47 |
| Irisin | 47 |
| Iritis | 48 |
| Iron | 48 |
| Irreducible Hernia | 48 |
| Irrespirable Gases | 48 |
| Irrigation | 48 |
| Irritability | 48 |
| Irritable | 48 |
| Irritants | 48 |
| Isaconitina | 48 |
| Isadelphia | 48 |
| Isapiol | 48 |
| Isatropyl-Cocaine | 48 |
| Ischæmia | 48 |
| Ischæmic Paralysis | 48 |
| Ischia | 48 |
| Ischiadelphia | 48 |
| Ischiagra | 48 |
| Ischialgia | 48 |
| Ischidrosis | 48 |
| Ischio- | 48 |
| Ischiocele | 48 |
| Ischiomelus | 48 |
| Ischiopagus | 48 |
| Ischium | 48 |
| Ischl | 48 |
| Ischochymia | 49 |
| Ischolochia | 49 |
| Ischuria | 49 |
| Isinglass | 49 |
| Isle of Wight | 49 |
| Iso- | 49 |
| Isobars | 49 |
| Isobe | 49 |
| Isochronous | 49 |
| Isoform | 49 |
| Isogens | 49 |
| Isolation | 49 |
| Isomer and Isomeric | 49 |
| Isometric Method | 49 |
| Isometropia | 49 |
| Isopral | 49 |
| Isotachiol | 49 |
| Isotherms | 49 |
| Isotonic | 49 |
| Isotropy | 49 |
| Ispaghula | 49 |
| Issue | 49 |
| Isthmus | 49 |
| Italy | 50 |
| Itch | 50 |
| Itch, Cuban | 50 |
| Itch, Dhobie | 50 |
| Itching | 50 |
| Iter | 50 |
| Itrol | 50 |
| Ixodes | 50 |
| Ixodiasis | 50 |
| Izal | 50 |
| Jaborandi | 50 |
| Jacket, Strait | 50 |
| Jacket, Plaster | 50 |
| Jacksonian Epilepsy | 50 |
| Jacobson's Nerve | 51 |
| Jacquemier's Test | 51 |
| Jactation or Jactitation | 51 |
| Jaeger's Test-Types | 51 |
| Jaffe's Test | 51 |
| Jail Fever | 51 |
| Jalap | 51 |
| James's Powder | 51 |
| Janet's Method | 51 |
| Janiceps or Janus | 51 |
| Japan | 51 |
| Jarisch's Ointment | 51 |
| Jasminum | 51 |
| Jaundice | 51 |
| Jaw-Clonus | 61 |
| Jaws | 61 |
| Jecorin | 62 |
| Jecur Uterinum | 62 |
| Jejuno- | 62 |
| Jejunostomy | 62 |
| Jellies | 62 |
| Jelly-Fish | 62 |
| Jelly of Wharton | 62 |
| Jenner, Edward | 62 |
| Jensen's Pump | 62 |
| Jensen's Tumour | 62 |
| Jequiritin | 62 |
| Jequiritol | 62 |
| Jequirity Seeds | 62 |
| Jerk | 62 |

| | PAGE |
|---|---|
| Jeyes' Fluid | 62 |
| Jigger | 62 |
| Jiu-Jitsu | 62 |
| Johannisbad | 62 |
| Johimbine | 62 |
| Joints, Diseases of | 62 |
| Jores' Fluid | 86 |
| Jothion | 86 |
| Joule's Law | 86 |
| Jugular | 86 |
| Juices | 86 |
| Jujubes | 86 |
| Julep | 86 |
| Jumpers | 86 |
| June Cold | 86 |
| Juniper | 86 |
| Junker's Apparatus | 86 |
| Jurisprudence, Medical | 86 |
| Jury Mast | 86 |
| Justo-major Pelvis | 86 |
| Justo-minor Pelvis | 86 |
| Juvenile General Paralysis | 86 |
| Kahler's Disease | 87 |
| Kairine | 87 |
| Kakke | 87 |
| Kala-azar | 87 |
| Kaladana | 87 |
| Kalium | 87 |
| Kalmuck Idiocy | 87 |
| Kalomelol | 87 |
| Kamala | 87 |
| Kamloops | 87 |
| Kangaroo Tendon | 87 |
| Kaolinum | 87 |
| Kaposi's Disease | 87 |
| Kaputine | 87 |
| Kariyat | 87 |
| Karlsbad | 87 |
| Karyochrome Cells | 87 |
| Karyokinesis | 87 |
| Karyomitosis | 87 |
| Katabolism | 87 |
| Katadicrotism | 87 |
| Kataphoresis | 87 |
| Katatonia | 87 |
| Katharsion | 87 |
| Kauri | 87 |
| Kava | 87 |
| Kawine | 87 |
| Kedgeree | 87 |
| Kefir | 87 |
| Kehrer's Operation | 87 |
| Kelene | 88 |
| Keloid | 88 |
| Kemmern | 88 |
| Kentucky | 88 |
| Kephir | 88 |
| Keratectasia | 88 |
| Keratectomy | 88 |
| Keratin | 88 |
| Keratinisation | 88 |
| Keratitis | 88 |
| Keratocele | 88 |
| Keratoconus | 88 |
| Keratodermia | 88 |
| Keratoglobus | 88 |
| Keratohyalin | 88 |
| Keratolysis | 88 |
| Keratoma | 88 |
| Keratomalacia | 88 |
| Keratonosus | 88 |
| Keratonyxis | 88 |
| Keratorrhexis | 88 |
| Keratoscopy | 88 |
| Keratosis | 88 |
| Kerion | 88 |
| Kernig's Sign | 88 |
| Ketones | 89 |
| Khosam | 89 |
| Kidinga Pepo | 89 |
| Kidney, Physiology of | 89 |
| Kidney, Surgical Affections of | 93 |
| Movable and Floating | 93 |
| Injuries of | 96 |
| Traumatic Nephritis | 98 |
| Perinephritis and Perinephritic Abscess | 98 |
| Renal Fistula | 100 |
| Pyelitis | 101 |
| Suppurative Pyelonephritis | 103 |
| Cysts of the Kidney | 103 |
| Stone in the Kidney | 104 |
| Renal Tuberculosis | 109 |
| Hydatid Cysts | 115 |
| Hydronephrosis | 116 |
| Pyonephrosis | 117 |
| Kidney Tumours | 119 |
| Actinomycosis | 120 |
| Ureter | 121 |
| Stone in the Ureter | 121 |
| Operative Procedures | 123 |
| Operations on the Ureter | 125 |
| Kiestein | 126 |
| Kiln-burning | 126 |
| Kinæsthesis | 126 |
| Kinæsthesiometer | 126 |
| Kincough | 126 |
| Kindergarten Teaching | 126 |
| Kinesioneurosis | 126 |
| King's Evil | 126 |
| Kinks | 126 |
| Kino | 126 |
| Kino, Bengal | 126 |
| Kinship | 126 |
| Kirkilissa | 126 |
| Kirrhonosis | 126 |
| Kissingen | 126 |
| Kjeldahl's Method | 126 |
| Klebs-Löffler Bacillus | 126 |
| Klein's Bacillus | 126 |
| Kleptomania | 127 |
| Klikuschi | 127 |
| Klopemania | 127 |
| Klopsophobia | 127 |
| Knacker | 127 |
| Knee-Jerk | 127 |
| Knee-Joint, Diseases of | 127 |
| Knee-Joint, Injuries of | 142 |
| Kneippism | 152 |
| Knock-Knee | 153 |
| Knots | 153 |
| Koch Treatment | 153 |
| Kocher's Method | 153 |
| Kocher's Operation | 153 |
| Koenig's Rods | 153 |
| Koilonychia | 153 |
| Kola Nut | 153 |
| Kolpo | 153 |
| Kombé | 153 |
| Kopftetanus | 153 |
| Koplik's Spots | 153 |
| Korsakoff's Syndrome | 153 |
| Kötschenowa | 153 |
| Koumiss | 153 |
| Kousso | 153 |
| Kra-Kra | 153 |
| Krameriæ Radix | 153 |
| Kraske's Operation | 153 |
| Kraurosis | 153 |
| Krause, Glands of | 153 |
| Kreatin | 153 |
| Kreatinin | 153 |
| Kresamine | 153 |
| Kresol | 153 |
| Kreuznach | 153 |
| Krishaber's Disease | 154 |
| Krohne and Sesemann's Apparatus | 154 |
| Kromskop | 154 |
| Krypton | 154 |
| Kubel's Method | 154 |
| Kubisagari | 154 |
| Kudowa | 154 |
| Kusatsu | 154 |
| Kutubuth | 154 |
| Kyestine | 154 |
| Kyphosis | 154 |
| Kythmos | 154 |
| Labia | 154 |
| Labidometer | 154 |
| Labio- | 154 |
| Labis | 154 |
| Labitome | 154 |
| Labium | 154 |
| Labour | 154 |
| Physiological Section | 154 |
| Physiology of Labour | 154 |
| Stages and Duration | 163 |
| Diagnosis and Mechanism | 188 |

| | PAGE |
|---|---|
| Labour—Physiological Section—*contd.* | |
| Management | 189 |
| Labour in Multiple Pregnancy | 204 |
| Pathological Section | 207 |
| Precipitate and Prolonged Labour | 208 |
| Faults in the Passenger | 239 |
| Accidental Complications affecting Child | 246 |
| Retention of Placenta | 252 |
| Post-partum Hæmorrhage | 256 |
| Injuries during Labour | 264 |
| Operations, Obstetrical | 276 |
| Induction of Premature Labour | 276 |
| Forceps | 281 |
| Vectis | 287 |
| Version | 287 |
| Embryotomy | 290 |
| Cleidotomy | 295 |
| Symphysiotomy | 297 |
| Cæsarean Section | 299 |
| La Bourboule | 305 |
| Labridæ | 305 |
| Labrum | 305 |
| Labyrinth | 305 |
| Labyrinthitis | 305 |
| Lac | 305 |
| Lace-Maker's Disease | 305 |
| Lacerated Wounds | 305 |
| Lacerations | 305 |
| Lachrymal | 305 |
| Lacing-Liver | 305 |
| Laconicum | 306 |
| Lacrimal Apparatus, Diseases of | 306 |
| Lacrimation or Lachrymation | 312 |
| Lacs | 312 |
| Lactagol | 312 |
| Lactalbumin | 312 |
| Lactams | 312 |
| Lactase | 312 |
| Lactate | 312 |
| Lactation | 312 |
| Lactational Insanity | 312 |
| Lacteals | 312 |
| Lactic Acid | 312 |
| Lactifuge | 312 |
| Lactometer | 313 |
| Lactophenin | 313 |
| Lactophosphates | 313 |
| Lactoscope | 313 |
| Lactose | 313 |
| Lactoserum | 313 |
| Lactoserve | 313 |
| Lactosomatose | 313 |
| Lactosuria | 313 |
| Lactuca | 313 |
| Lacuna | 313 |
| Læhme | 313 |
| Lævulose | 313 |
| Lævulosuria | 313 |
| Lagneia or Lagnesis | 313 |
| Lagophthalmos | 313 |
| Lagostoma | 313 |
| La Grippe | 313 |
| Lahs' Theory | 313 |
| Laking of Blood | 313 |
| Lalling | 313 |
| Laloo | 313 |
| Lalopathy | 313 |
| Laloplegia | 313 |
| Lamalou | 313 |
| Lambdacism | 313 |
| Lambdoidal | 313 |
| Lamblia Intestinalis | 314 |
| Lamella | 314 |
| Lamellæ | 314 |
| Lameness, Intermittent | 314 |
| Lamina | 314 |
| Laminaria Tents | 314 |
| Laminectomy | 314 |
| Lancet | 314 |
| Lancinating | 314 |
| Landeck | 314 |
| Landouzy-Déjerine's Myopathy | 314 |
| Landry's Paralysis | 314 |
| Langenbeck's Operation | 314 |
| Langerhans' Cells or Islets | 314 |
| Langhans' Layer | 314 |
| Language | 314 |
| Lanoform | 314 |
| Lanoline | 314 |
| Lanugo | 314 |
| Laparo- | 314 |
| Laparocele | 314 |
| Laparoelytrotomy | 314 |
| Laparomyomectomy | 314 |
| Laparonephrectomy | 314 |
| Laparotomy | 314 |
| Lapis | 315 |
| Lappa | 315 |
| Lard | 315 |
| Lardacein | 315 |
| Lardaceous Degeneration | 315 |
| Largin | 319 |
| Larva | 319 |
| Larval or Larvated | 319 |
| Larva Migrans | 319 |
| Laryngeal Crises | 319 |
| Laryngeal Injections | 319 |
| Laryngeal Obstruction | 319 |
| Laryngeal Paralysis | 319 |
| Laryngeal Phthisis | 319 |
| Laryngeal Spasm | 319 |
| Laryngectomy | 319 |
| Laryngismus Stridulus | 320 |
| Laryngitis | 320 |
| Laryngo-Fissure | 320 |
| Laryngoscopy | 320 |
| Larynx | 320 |
| Examination | 320 |
| Acute and Chronic Inflammations | 324 |
| Acute Laryngitis | 324 |
| Chronic Laryngitis | 327 |
| Hypertrophic Laryngitis | 331 |
| Membranous Laryngitis, Non-diphtheritic | 331 |
| Œdematous Laryngitis | 332 |
| Chronic Subglottic Laryngitis | 333 |
| Nodular Laryngitis | 334 |
| Laryngitis Sicca | 335 |
| Acute Laryngitis in Children | 336 |
| Injuries | 336 |
| Dislocations | 337 |
| Foreign Bodies | 337 |
| Hæmorrhage | 339 |
| Congenital Glottic Stenosis | 339 |
| Pachydermia Laryngis | 340 |
| Chronic Infective Diseases | 341 |
| Laryngeal Phthisis | 342 |
| Lupus | 345 |
| Syphilis | 345 |
| Scleroma | 346 |
| Leprosy | 347 |
| Neoplasms | 347 |
| Benign or Simple | 347 |
| Malignant | 351 |
| Neuroses | 362 |
| Sensory | 363 |
| Motor | 364 |
| Hypertrophy of Lingual Tonsil | 369 |
| Affections of the Cartilages | 369 |
| Perichondritis | 369 |
| Anchylosis of Crico-arytenoid Joint | 371 |
| Stenosis of the Larynx | 372 |
| Congenital Laryngeal Stridor | 372 |
| Laryngismus Stridulus | 373 |
| Lasanon | 375 |
| Lasègue's Law | 375 |
| Lasègue's Sign | 375 |
| Laserol | 375 |
| Lassar's Paste | 375 |
| Lassitude | 375 |
| Latah | 375 |

| | PAGE |
|---|---|
| Latent | 375 |
| Laterad | 375 |
| Lateral Chain Theory | 375 |
| Lateral Curvature | 375 |
| Lateral Deviation | 375 |
| Lateral Fillet | 375 |
| Lateral Operation | 375 |
| Lateral Sclerosis | 376 |
| Lateral Sinus | 376 |
| Lateral Ventricles | 376 |
| Late Rickets | 376 |
| Lateritious Deposit | 376 |
| Latero- | 376 |
| Latex | 376 |
| Lathyrism | 376 |
| Lathyrus | 376 |
| Latissimus Dorsi | 376 |
| Latitude | 376 |
| Latrines | 376 |
| Laudable | 376 |
| Laudanum | 376 |
| Laughing-Gas | 376 |
| Laugier's Hernia | 376 |
| Laumonier's Ganglion | 376 |
| Laundries | 376 |
| Laurocerasi Folia | 376 |
| Laurvik | 376 |
| Lavage | 376 |
| Lavatories | 376 |
| Lavender | 377 |
| Laveran's Bodies | 377 |
| Lavey | 377 |
| Law | 377 |
| Laxative | 377 |
| "Laxoin" | 377 |
| Layman | 377 |
| Lazaretto | 377 |
| Lead | 377 |
| Lead Colic | 378 |
| Lead Palsy | 378 |
| Lead Poisoning | 378 |
| Leamington | 378 |
| Leaping Ague | 378 |
| Leash Ulcer | 378 |
| Leather Dressing | 378 |
| Leber's Atrophy | 378 |
| Lecanopagus | 378 |
| Lecithin | 378 |
| Lecitogen | 378 |
| Ledesma | 378 |
| Leeches | 378 |
| Leeks | 378 |
| Leg | 378 |
| Legal Medicine | 379 |
| Legal's Disease | 379 |
| Legitimacy | 379 |
| Legumens | 379 |
| Leguminosæ | 379 |
| Lehman's Sign | 379 |
| Leio- | 379 |
| Leiodermia | 379 |
| Leiomyoma | 379 |
| Leishman-Body | 379 |
| Leiter's Coil | 379 |
| Lembert Suture | 379 |
| Lemniscus | 379 |
| Lemon | 379 |
| Lemonade | 379 |
| Lenigallol | 379 |
| Lenirobin | 379 |
| Lenitive | 379 |
| Lenk | 379 |
| Lennhoff's Sign | 379 |
| Lens (Crystalline) | 379 |
| Lenses | 381 |
| Lenticonus | 381 |
| Lenticular | 381 |
| Lentigo | 381 |
| Lentils | 381 |
| Lentin | 381 |
| Leontiasis Hystrix | 381 |
| Leontiasis Ossea | 381 |
| Lepanto | 382 |
| Lepcophtheirus | 382 |
| Leper | 382 |
| Lepido- | 382 |
| Le Pita | 382 |
| Lepra | 382 |
| Leprelcosis | 382 |
| Leprology | 382 |
| Leprosy | 382 |
| Leptismus | 390 |
| Lepto- | 391 |
| Leptocephaly | 391 |
| Leptochrous | 391 |
| Leptomeningitis | 391 |
| Leptomicrognathus | 391 |
| Leptomitus Lacteus | 391 |
| Leptophonia | 391 |
| Leptoprosopous | 391 |
| Leptorrhine | 391 |
| Leptothrix | 391 |
| Leptus Autumnalis | 391 |
| Le Ragle | 391 |
| Lerema | 391 |
| Leresis | 391 |
| Les Avants | 391 |
| Leschenoma | 391 |
| Lesion | 391 |
| Lesser's Triangle | 391 |
| Leste | 391 |
| Lethal | 391 |
| Lethargy | 391 |
| Letheomania | 391 |
| Letheon, Compound | 391 |
| Lethin | 391 |
| Lethrinus Mambo | 391 |
| Lettuce | 391 |
| Leucæmia | 391 |
| Leucæthiopia | 391 |
| Leucine | 391 |
| Leucinuria | 392 |
| Leucoblasts | 392 |
| Leucocidine | 392 |
| Leucocytes | 392 |
| Leucocythæmia | 392 |
| Leucocytosis | 400 |
| Leucoderma | 405 |
| Leucokeratosis Mucosæ Oris | 405 |
| Leucoma | 405 |
| Leucomaines | 405 |
| Leucomelanoderma | 406 |
| Leucomoria | 406 |
| Leucomyelitis | 406 |
| Leuconecrosis | 406 |
| Leuconychia | 406 |
| Leucopathia | 406 |
| Leucopenia | 406 |
| Leucophlegmasia | 406 |
| Leucorrhœa | 406 |
| Leucotrichia | 406 |
| Leukæmia | 406 |
| Leukanæmia | 406 |
| Leukokeratosis | 406 |
| Leukoplakia | 406 |
| Levatores | 406 |
| Levico | 406 |
| Levigation | 406 |
| Levinstein's Method | 406 |
| Levulose | 406 |
| Levurine | 406 |
| Leyden Jar | 406 |
| Leyden's Ataxia | 406 |
| Leyden's Crystals | 406 |
| Leysin | 406 |
| L.G.B. | 406 |
| Lice | 406 |
| Lichen | 406 |
| Lichenification | 411 |
| Lichens | 411 |
| Lidja | 411 |
| Lie | 411 |
| Lieberkühn's Follicles | 411 |
| Liebermann's Test | 411 |
| Liebig's Extract | 411 |
| Lienculus | 411 |
| Lienitis | 411 |
| Lienocele | 411 |
| Lienomalacia | 411 |
| Lientery | 411 |
| Liernur System | 411 |
| Life, Change of | 411 |
| Life Insurance | 411 |
| Ligaments | 421 |
| Ligar's Line | 421 |
| Ligation | 421 |
| Ligature | 421 |
| Light | 421 |
| Lightening | 421 |
| Lighting | 421 |
| Lightning | 421 |
| Lightning-Pains | 422 |
| Lignin | 422 |
| Lignosulphin | 422 |
| Lignum | 422 |

| | PAGE |
|---|---|
| Ligula | 422 |
| Lilienfeld's Theory | 422 |
| Lily of the Valley | 422 |
| Liman Cure | 422 |
| Limatura Ferri | 422 |
| Limb | 422 |
| Limbus | 422 |
| Lime | 422 |
| Lime Fruit | 422 |
| Lime Process | 422 |
| Lime Test | 422 |
| Limiting Membrane | 422 |
| Limnœa Truncatulata | 422 |
| Limonis Cortex | 422 |
| Limosis | 422 |
| Limotherapy | 422 |
| Linadin | 422 |
| Linalool | 422 |
| Linamarin | 422 |
| Linctus | 422 |
| Linctus Opiatus | 422 |
| Linea | 422 |
| Linen | 422 |
| Lingering Labour | 423 |
| Ling's System | 423 |
| Lingua | 423 |
| Lingua Frænata | 423 |
| Lingual | 423 |
| Linguatula | 423 |
| Lingula | 423 |
| Linimentum | 423 |
| Linoleum | 423 |
| Linseed or Linum | 423 |
| Liouville's Icterus | 423 |
| Lip | 423 |
| Lipacidæmia | 423 |
| Lipaciduria | 423 |
| Lipæmia | 423 |
| Lipanin | 423 |
| Liparocele | 423 |
| Liparomphalus | 423 |
| Lipase | 423 |
| Lipliawsky's Test | 423 |
| Lipo- | 424 |
| Lipochromes | 424 |
| Lipoma | 424 |
| Lipomatosis | 424 |
| Lipomphalocele | 424 |
| Lipomyoma | 424 |
| Lipomyxoma | 424 |
| Lipothymia | 424 |
| Lipotrichia | 424 |
| Lippitude | 424 |
| Lippspringe | 424 |
| Lip-Tie | 424 |
| Lipuria | 424 |
| Liquefaction-Necrosis | 424 |
| Liqueurs | 424 |
| Liquid | 424 |
| Liquor | 424 |
| Liquor Amnii | 424 |
| Liquor Antisepticus | 424 |
| Liquor Ammoniæ Fortis | 424 |
| Liquor Anodynus | 424 |
| Liquor Arsenicalis | 424 |
| Liquor Arsenii et Hydrargyri Iodidi | 424 |
| Liquor Calcis | 424 |
| Liquor Carbonis Detergens | 424 |
| Liquor Concentratus | 425 |
| Liquor Epispasticus | 425 |
| Liquor Folliculi | 425 |
| Liquor Graafianus | 425 |
| Liquor Hydrargyri Perchloridi | 425 |
| Liquor Hydrogenii Peroxidi | 425 |
| Liquor Morgagni | 425 |
| Liquor Opii Sedativus | 425 |
| Liquor Pancreatis | 425 |
| Liquor Picis Carbonis | 425 |
| Liquor Sanguinis | 425 |
| Liquorice | 425 |
| Liriodendron Procerum | 425 |
| Lisdoonvarna | 425 |
| Lisfranc's Amputation | 425 |
| Lisfranc's Tubercle | 425 |
| Lissauer's Tract | 425 |
| Lissencephaly | 425 |
| Listerine | 425 |
| Listerism | 425 |
| Lister's Ointment | 425 |
| Lithæmia | 425 |
| Lithagogue | 425 |
| Litharge | 425 |
| Lithates | 425 |
| Lithia | 425 |
| Lithiasis | 425 |
| Lithic Acid | 425 |
| Lithio-Piperazine | 425 |
| Lithium | 426 |
| Litho- | 426 |
| Lithoclast | 426 |
| Lithodialysis | 426 |
| Lithogenesis | 426 |
| Lithokelyphopædion | 426 |
| Litholapaxy | 426 |
| Lithonephrotomy | 426 |
| Lithontriptic | 426 |
| Lithopædion | 426 |
| Lithophone | 426 |
| Lithotomy | 426 |
| Lithotomy Position | 426 |
| Lithotripsy | 426 |
| Lithotrite | 426 |
| Lithotrity | 426 |
| Lithuria | 426 |
| Litmus Paper | 426 |
| Litten's Sign | 426 |
| Littlehampton | 426 |
| Little's Disease | 426 |
| Littré's Colotomy | 427 |
| Littré's Glands | 427 |
| Littré's Hernia | 427 |
| Litzmann's Obliquity | 427 |
| Livedo Annulare | 427 |
| Liver | 427 |
| Physiology of | 427 |
| Diseases of | 431 |
| Anatomical Abnormalities | 431 |
| Hepatoptosis | 435 |
| Functional Disease | 437 |
| Diseases of Hepatic Artery | 440 |
| Diseases of Hepatic Veins | 441 |
| Diseases of Lymphatic Vessels | 441 |
| Chronic Venous Engorgement | 442 |
| Biliary Cirrhosis | 445 |
| Portal Cirrhosis | 451 |
| Hepatic Cirrhosis in Early Life | 462 |
| Degenerations and Infiltrations | 462 |
| Innocent Tumours | 466 |
| Cysts | 468 |
| Infective Granulomata | 469 |
| Malignant Disease | 478 |
| Icterus | 483 |
| Weil's Disease | 487 |
| Tropical Diseases of | 488 |
| Perihepatitis | 488 |
| Acute Hepatitis | 494 |
| Derangements of Circulation | 496 |
| Suppuration | 501 |
| Parasites | 519 |
| Liver of Sulphur | 523 |
| Lividity | 523 |
| "Living Skeletons" | 524 |
| Lixiviation | 524 |
| Llandrindod | 524 |
| Llandudno | 524 |
| Llangammarch | 524 |
| Llanwrtyd | 524 |
| Lobar | 524 |
| Lobe or Lobus | 524 |
| Lobelia | 524 |
| Lobeline | 524 |
| Lobengulism | 524 |
| Lobstein's Cancer | 524 |
| Lobstein's Disease | 524 |
| Lobster-claw Deformity | 524 |
| Lobular | 524 |
| Lobule or Lobulus | 524 |
| Local Anæsthesia | 524 |
| Localisation | 524 |
| Locarno | 524 |
| Lochia | 524 |
| Lochio- or Locho- | 524 |
| Lock-Finger | 525 |

| | PAGE |
|---|---|
| Lockhart-Clarke's Column | 525 |
| Locking of Heads | 525 |
| Lockjaw | 525 |
| Lock-Spasm | 525 |
| Lockwood's Ligament | 525 |
| Lockwood's Method | 525 |
| Loco Deflector | 525 |
| Loco Disease | 525 |
| Locomotor Ataxia | 525 |
| Loculus | 525 |
| Locus | 525 |
| Lodging-Houses | 525 |
| Loebisch's Formula | 525 |
| Loeche-les-Bains | 525 |
| Loeffler's Bacillus | 525 |
| Loeffler's Blood-Serum Mixture | 525 |
| Loehlein's Diameter | 525 |
| Loewenthal's Reaction | 525 |
| Loganin | 525 |
| Logo- | 525 |
| Logomania or Logomonomania | 526 |
| Logwood | 526 |
| Loimology | 526 |
| Loin | 526 |
| Loka | 526 |
| Lolium Temulentum | 526 |
| Longevity | 526 |
| "Longings" | 526 |
| Longissimus | 526 |
| Longus | 526 |
| Loose Bodies | 526 |
| Loose Shoulders | 526 |
| Lopez Root | 526 |
| Lopho- | 526 |
| Lordosis | 526 |
| Lorenz Operation | 526 |
| Loreta's Operation | 526 |
| Loretine | 526 |
| Los Angeles | 526 |
| Losophan | 526 |
| Lota Vulgaris | 526 |
| Lotio | 526 |
| Lotio Rubra | 526 |
| Lotio Spiritus | 526 |
| Louis's Law | 526 |
| Louping Ill | 526 |
| Louvres | 527 |
| Lowcock's Filter | 527 |
| Lower Extremity | 527 |
| Lower Jaw | 527 |
| Lower, Tubercle of | 527 |
| Lower Uterine Segment | 527 |
| Low Fever | 527 |
| Lozenges | 527 |
| Lubarsch's Crystals | 527 |
| Lucan | 527 |
| Lucas' Sign | 527 |
| Lucca | 527 |
| Lucid Interval | 527 |
| Lucifer Match-Making | 527 |
| Lucilia Hominivora | 527 |
| Lucilia Macellaria | 527 |
| Lucina | 527 |
| Ludwig's Angina | 527 |
| Ludwig-Salkowski's Method | 527 |
| Ludwig's Theory | 527 |
| Lueck's Reaction | 527 |
| Lues | 527 |
| Lugano | 527 |
| Lumbago | 527 |
| Lumbar | 530 |
| Lumbar Abscess | 530 |
| Lumbar Anæsthesia | 530 |
| Lumbar Colotomy | 530 |
| Lumbar Glands | 530 |
| Lumbar Hernia | 530 |
| Lumbar Nephrotomy | 530 |
| Lumbar Plexus | 530 |
| Lumbar Puncture | 530 |


END OF VOL. V.

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