

## **Wheeler's handbook of medicine / by William R. Jack.**

### **Contributors**

Wheeler, Alexander, -1903.  
Jack, William R. 1866-1927.

### **Publication/Creation**

Edinburgh : E. & S. Livingstone, 1916.

### **Persistent URL**

<https://wellcomecollection.org/works/g69srnfn>

### **License and attribution**

This work has been identified as being free of known restrictions under copyright law, including all related and neighbouring rights and is being made available under the Creative Commons, Public Domain Mark.

You can copy, modify, distribute and perform the work, even for commercial purposes, without asking permission.



Wellcome Collection  
183 Euston Road  
London NW1 2BE UK  
T +44 (0)20 7611 8722  
E [library@wellcomecollection.org](mailto:library@wellcomecollection.org)  
<https://wellcomecollection.org>









22101354315

H. K. LEWIS & CO., LTD.  
105 GOWER STREET,  
LONDON, W.C.





87.2el  
5p

# HANDBOOK OF MEDICINE

*First Edition, February 1894.*

*Second „ June 1903.*

*Third „ May 1908.*

*Fourth „ April 1912.*

*Reprint „ June 1914.*

*Reprint „ February 1915.*

*Fifth „ January 1916.*



WHEELER'S  
HANDBOOK OF MEDICINE

BY

WILLIAM R. JACK

B.Sc., M.D., F.R.F.P.S.G.

PHYSICIAN TO THE GLASGOW ROYAL INFIRMARY  
LECTURER IN CLINICAL MEDICINE IN THE UNIVERSITY, GLASGOW

*FIFTH EDITION*

EDINBURGH  
E. & S. LIVINGSTONE

1916

WELLCOME INSTITUTE LIBRARY	
Coll.	we1M0mec
Call	
No.	WB100
	1916
	W56w



## PREFACE TO THE FIFTH EDITION


SINCE the publication of the fourth edition of this handbook the chief advances in our knowledge of disease are those which have been made possible by the newer methods of investigation of the heart. The section of the volume devoted to cardiac diseases has in consequence undergone considerable enlargement; but though in other sections there are fewer notable additions, every part of the book has been carefully revised. The paragraphs dealing with treatment have in many instances been amplified with a view both to including newer methods and to describing the older ones with somewhat more detail. A slight increase in the size of the work has therefore been inevitable, but by rigid economy of space, it has been found possible to avoid the material alteration which the many internal changes would otherwise have occasioned.

The author desires to express his thanks and those of the publishers to Messrs. Hawksley & Sons for the loan of blocks in illustration of the sphygmomanometer and the polygraph; to Messrs. Baillière, Tindall & Cox for a print showing the position of the cardiac valves; and to Mr. Edward Arnold for an illustration showing the position of the heart and great vessels.

As on former occasions, his cordial thanks are due to the publishers, and especially to Dr. A. Walker, for their helpfulness in the preparation of the volume.

WILLIAM R. JACK.

16 WOODSIDE PLACE,  
GLASGOW, 1916.



Digitized by the Internet Archive  
in 2019 with funding from  
Wellcome Library

<https://archive.org/details/b31363659>



## PREFACE TO THE FOURTH EDITION

THE advances which have taken place in the domain of medicine within the last four years have been so numerous as to call for considerable additions even to so brief a handbook as the present. It would be tedious to enumerate them all, and it must suffice to mention that, with a view to making the volume more complete, a section on Intoxications has been added, and among the Specific Infections many new articles have been devoted to Tropical Diseases, now so extensively studied. The subject of Immunity has also called for notice, and I have very gratefully to acknowledge Professor Robert Muir's kind permission to make use of the admirable article in Muir and Ritchie's *Manual of Bacteriology* in preparing my brief synopsis of its main features. All the other articles have been carefully revised, and many of them almost entirely rewritten. So great indeed have been the changes since 1894, the date of the first edition, that little now remains of Dr. Wheeler's writing save in the sections upon Symptomatology, and even these have been, in the majority of instances, amplified or extensively modified. His original object, however, is still the object of the book, namely, the correlation of symptoms with the facts of anatomy and physiology, in a volume which does not by its size preclude the possibility of its being used as a companion in the clinical study of disease.

My cordial thanks are due to the publishers, and especially to Dr. A. Walker, for their helpfulness in the preparation of the volume.

WILLIAM R. JACK.

43 LANSDOWNE CRESCENT,  
GLASGOW, 1912.





# CONTENTS

	PAGE
I. FEVER . . . . .	1
Physiology of Fever . . . . .	4
Types of Fever . . . . .	5
The Typhoid State . . . . .	8
Delirium . . . . .	9
General Treatment of Fever . . . . .	10
II. IMMUNITY . . . . .	12
Theories of Immunity . . . . .	15
Vaccine Treatment . . . . .	17
III. SPECIFIC INFECTIOUS DISEASES . . . . .	19
Enteric or Typhoid Fever . . . . .	19
Typhus Fever . . . . .	26
Relapsing Fever . . . . .	28
Variola or Small-pox . . . . .	30
Vaccinia . . . . .	34
Varicella or Chicken-pox . . . . .	35
Scarlatina or Scarlet Fever . . . . .	36
Morbilli or Measles . . . . .	40
Rubella or German Measles . . . . .	42
Pertussis or Whooping-cough . . . . .	43
Mumps . . . . .	46
Glandular Fever . . . . .	47
Influenza . . . . .	47
Dengue . . . . .	49
Cerebro-spinal Fever . . . . .	50
Posterior Basic Meningitis . . . . .	53
Diphtheria . . . . .	54
Croup . . . . .	59
Erysipelas . . . . .	60
Septic Diseases . . . . .	62
Rheumatic Fever . . . . .	64
Plague . . . . .	67
Cholera . . . . .	70
Dysentery . . . . .	73
Yellow Fever . . . . .	77
Mediterranean Fever (Malta Fever) . . . . .	80
Malarial Fever . . . . .	81
Actinomycosis . . . . .	87
Anthrax . . . . .	89
Glanders . . . . .	92
Foot-and-Mouth Disease . . . . .	93
Hydrophobia . . . . .	94
Tetanus . . . . .	96

	PAGE
Gonorrhœal Infection . . . . .	98
Syphilis . . . . .	99
Yaws . . . . .	108
Trypanosomiasis . . . . .	108
Leishmaniasis (Kala-azar) . . . . .	110
Tuberculosis . . . . .	111
Leprosy . . . . .	115
IV. DISORDERS OF NUTRITION . . . . .	118
Gout . . . . .	118
Rickets . . . . .	123
Beri-beri . . . . .	126
Chronic Rheumatism . . . . .	128
Muscular Rheumatism . . . . .	129
Rheumatoid Arthritis . . . . .	129
Diabetes Mellitus . . . . .	132
Diabetes Insipidus . . . . .	139
V. THE INTOXICATIONS . . . . .	141
Alcoholism . . . . .	141
Morphinism and Cocainism . . . . .	143
Lead Poisoning . . . . .	144
Arsenical Poisoning . . . . .	146
Food Poisoning . . . . .	147
Sunstroke . . . . .	149
VI. DISEASES OF THE ALIMENTARY TRACT . . . . .	151
1. The Mouth . . . . .	151
Stomatitis . . . . .	151
Acute Tonsillitis . . . . .	153
Chronic Tonsillitis . . . . .	155
2. The Œsophagus . . . . .	155
Œsophagitis . . . . .	155
Stricture of the Œsophagus . . . . .	156
Tumours of the Œsophagus . . . . .	157
3. The Stomach . . . . .	157
Examination of the Stomach Contents . . . . .	157
Acute Gastritis . . . . .	160
Chronic Gastritis . . . . .	161
Gastric and Duodenal Ulcer . . . . .	162
Cancer of the Stomach . . . . .	168
Hæmatemesis . . . . .	171
Dilatation of the Stomach . . . . .	173
Hypertrophic Stenosis of the Pylorus . . . . .	175
Nervous Dyspepsia . . . . .	175
4. The Intestines . . . . .	178
Enteritis . . . . .	178
Appendicitis . . . . .	183
Intestinal Obstruction . . . . .	186
Tumours of the Intestines . . . . .	189
Diarrhœa . . . . .	190
Constipation . . . . .	191



# CONTENTS

xi

	PAGE
VII. DISEASES DUE TO INTERNAL PARASITES . . . . .	194
Cestoda . . . . .	194
Tænia Solium . . . . .	196
Tænia Mediocanellata . . . . .	197
Bothriocephalus Latus . . . . .	198
Tænia Echinococcus . . . . .	199
Nematoda . . . . .	201
Trichina Spiralis . . . . .	202
Ascaris Lumbricoides . . . . .	204
Oxyuris Vermicularis . . . . .	205
Ankylostomum Duodenale . . . . .	205
Filaria Sanguinis Hominis . . . . .	206
Trematoda . . . . .	208
Bilharzia Hæmatobia . . . . .	208
VIII. DISEASES OF THE LIVER . . . . .	210
Functions of the Liver . . . . .	210
Jaundice . . . . .	213
Icterus Neonatorum . . . . .	216
Acute Yellow Atrophy . . . . .	216
Abscesses in the Liver . . . . .	217
Cirrhosis of the Liver . . . . .	220
Syphilis of the Liver . . . . .	224
Cancer of the Liver . . . . .	225
Hydatid Disease . . . . .	227
Amyloid or Waxy Liver . . . . .	228
Fatty Liver . . . . .	229
Passive Congestion of the Liver . . . . .	229
IX. DISEASES OF THE GALL-BLADDER AND BILE-DUCTS . . . . .	230
Cholecystitis . . . . .	230
Cholangitis . . . . .	230
Tumours . . . . .	231
Gall-stones . . . . .	231
X. DISEASES OF THE PANCREAS . . . . .	234
XI. DISEASES OF THE PERITONEUM . . . . .	236
Ascites . . . . .	236
Acute Peritonitis . . . . .	239
Chronic Peritonitis . . . . .	241
XII. DISEASES OF THE CIRCULATORY SYSTEM . . . . .	244
1. The Heart . . . . .	244
Physical Examination . . . . .	245
Pericarditis . . . . .	251
Acute Endocarditis . . . . .	254
Chronic Endocarditis . . . . .	258
Effects of Chronic Valvular Disease . . . . .	259
Failure of Compensation . . . . .	261
Hypertrophy of the Heart . . . . .	263
Dilatation of the Heart . . . . .	265
Valvular Lesions of the Heart . . . . .	266
Diseases of the Myocardium . . . . .	276



	PAGE
Angina Pectoris . . . . .	279
Disorders of the Cardiac Rhythm . . . . .	281
2. The Arteries . . . . .	286
Arterio-sclerosis . . . . .	286
Aneurysm . . . . .	287
Aneurysm of the Thoracic Aorta . . . . .	289
Aneurysm of the Abdominal Aorta . . . . .	294
 XIII. DISEASES OF THE BLOOD AND DUCTLESS GLANDS . . . . .	 295
1. The Blood . . . . .	295
Examination of the Blood . . . . .	295
Anæmia . . . . .	297
Symptomatic Anæmia . . . . .	298
Chlorosis . . . . .	298
Pernicious Anæmia . . . . .	300
Leukæmia . . . . .	303
Lymphadenoma . . . . .	305
Status Lymphaticus . . . . .	307
Scurvy . . . . .	308
Hæmophilia . . . . .	309
Paroxysmal Hæmoglobinuria . . . . .	311
Purpura . . . . .	311
2. The Thyroid Gland . . . . .	313
Myxœdema . . . . .	313
Goitre . . . . .	316
Exophthalmic Goitre . . . . .	317
3. The Suprarenal Glands . . . . .	319
Addison's Disease . . . . .	319
4. The Spleen . . . . .	321
Movable Spleen . . . . .	321
Splenic Anæmia . . . . .	322
Splenic Tumour . . . . .	323
5. The Pituitary Body . . . . .	323
Acromegaly . . . . .	323
 XIV. DISEASES OF THE RESPIRATORY ORGANS . . . . .	 325
Examination of the Respiratory System . . . . .	326
1. The Larynx . . . . .	332
Acute Laryngitis . . . . .	332
Œdema of the Glottis . . . . .	333
Chronic Laryngitis . . . . .	334
Laryngeal Tuberculosis . . . . .	334
Laryngeal Syphilis . . . . .	335
Laryngeal Tumours . . . . .	335
Laryngismus Stridulus . . . . .	335
Laryngeal Paralysis . . . . .	337
2. The Bronchi . . . . .	338
Acute Bronchitis . . . . .	338
Chronic Bronchitis . . . . .	340
Bronchiectasis . . . . .	342
Asthma . . . . .	343
3. The Lungs . . . . .	345
Emphysema . . . . .	345



# CONTENTS

xiii

	PAGE
Collapse of the Lung . . . . .	347
Œdema of the Lungs . . . . .	348
Congestion of the Lungs . . . . .	349
Hæmoptysis and Infarction . . . . .	350
Lobar Pneumonia . . . . .	351
Bronchopneumonia . . . . .	356
Interstitial Pneumonia . . . . .	358
Phthisis . . . . .	359
Gangrene and Abscess of the Lung . . . . .	368
Tumours of the Lung . . . . .	369
4. The Pleura . . . . .	369
Pleurisy and Empyema . . . . .	369
Pneumothorax . . . . .	376
Hydrothorax . . . . .	378
5. The Mediastinum . . . . .	378
Mediastinal Tumours . . . . .	378
 XV. DISEASES OF THE KIDNEYS . . . . .	380
Examination of the Urine . . . . .	380
Albuminuria . . . . .	389
Hæmaturia . . . . .	390
Uræmia . . . . .	392
Acute Bright's Disease . . . . .	394
Chronic Bright's Disease . . . . .	397
Lardaceous (Amyloid) Disease of the Kidney . . . . .	404
Pyelitis, Pyelonephritis, and Pyonephrosis . . . . .	405
Hydronephrosis . . . . .	407
Renal Calculus . . . . .	408
Renal Tuberculosis . . . . .	410
Renal Tumours . . . . .	411
Movable Kidney . . . . .	412
 XVI. DISEASES OF THE NERVOUS SYSTEM . . . . .	413
1. Introductory . . . . .	413
The Neurone . . . . .	413
The Spinal Cord . . . . .	414
The Motor Path . . . . .	417
The Sensory Path . . . . .	418
2. General Symptomatology . . . . .	422
3. Diseases of the Spinal Cord and its Membranes . . . . .	428
Spinal Meningitis . . . . .	429
Meningeal Hæmorrhage . . . . .	431
Acute Myelitis . . . . .	433
Syringomyelia . . . . .	435
Tumours of the Cord . . . . .	436
System Diseases of the Cord : Anterior Poliomyelitis (Infantile Paralysis) . . . . .	436
System Diseases of the Cord : Chronic . . . . .	439
Locomotor Ataxia . . . . .	439
Spastic Paraplegia . . . . .	443
Progressive Muscular Atrophy . . . . .	445



	PAGE
Amyotrophic Lateral Sclerosis . . . . .	447
Ataxic Paraplegia . . . . .	449
Hereditary Ataxia . . . . .	450
4. Diseases of the Medulla Oblongata . . . . .	452
Progressive Bulbar Paralysis . . . . .	452
5. Diseases of the Brain . . . . .	455
Anatomy of the Brain . . . . .	455
Blood Supply of the Brain . . . . .	457
Functions of the Brain . . . . .	459
Cerebral Meningitis . . . . .	464
Hydrocephalus . . . . .	468
Sinus Thrombosis . . . . .	469
Encephalitis . . . . .	470
Abscess of the Brain . . . . .	471
Cerebral Hæmorrhage . . . . .	472
Cerebral Embolism and Thrombosis . . . . .	476
Hemiplegia . . . . .	478
Cerebral Palsies of Infancy . . . . .	480
Cerebral Syphilis . . . . .	481
Tumours of the Brain . . . . .	483
Aphasia . . . . .	485
Disseminated Sclerosis . . . . .	488
General Paralysis . . . . .	490
6. Diseases of Peripheral Nerves . . . . .	493
Cranial Nerves . . . . .	493
Olfactory Nerves . . . . .	493
Optic Nerves . . . . .	493
Third, Fourth, and Sixth Nerves . . . . .	496
Fifth Nerve . . . . .	501
Seventh Nerve . . . . .	503
Eighth Nerve . . . . .	506
Ninth to Twelfth Nerves . . . . .	508
Spinal Nerves . . . . .	510
Phrenic Nerve . . . . .	510
Neuritis . . . . .	510
Multiple (Peripheral) Neuritis . . . . .	511
7. Functional Diseases of the Nervous System . . . . .	513
Neuralgia . . . . .	513
Migraine . . . . .	514
Epilepsy . . . . .	515
Jacksonian Epilepsy . . . . .	519
Infantile Convulsions . . . . .	520
Tetany . . . . .	521
Hysteria . . . . .	522
Neurasthenia . . . . .	525
Chorea . . . . .	526
Spasmodic Torticollis . . . . .	528
Paralysis Agitans . . . . .	529
Raynaud's Disease . . . . .	531
XVII. DISEASES OF THE MUSCLES . . . . .	533
The Muscular Dystrophies . . . . .	533
Thomsen's Disease . . . . .	535



# LIST OF ILLUSTRATIONS

FIG.	PAGE
1. Continued Fever : Case of Acute Miliary Tuberculosis . . . . .	6
2. Remittent Fever : Case of Phthisis Pulmonalis . . . . .	6
3. Intermittent Fever : Case of Tertian Ague . . . . .	7
4. Gradual Rise of Temperature at Onset of Enteric Fever . . . . .	7
5. Decline of Fever by Crisis : Case of Lobar Pneumonia . . . . .	7
6. Decline of Fever by Lysis : Case of Bronchopneumonia . . . . .	7
7. Riva-Rocci Sphygmomanometer : Martin's Modification . . . . .	250
8. Mackenzie's Clinical Polygraph . . . . .	250
9. Pulse of Aortic Stenosis . . . . .	270
10. Pulse of Aortic Regurgitation . . . . .	270
11. Irregular Pulse of Mitral Regurgitation . . . . .	271
12. Pulsus Alternans . . . . .	284
13. Pulse of High Blood-pressure, from a Case of Bright's Disease . . . . .	398
14. Descending Tracts in the Spinal Cord . . . . .	415
15. Ascending Tracts in the Spinal Cord . . . . .	416
16. Position of Tracts of Fibres in the Internal Capsule . . . . .	417
17. The Motor Decussation in the Bulb . . . . .	418
18. Connections of Nerve Fibres in the Spinal Cord . . . . .	419
19. The Sensory Decussation in the Bulb . . . . .	420
20. Connections of the Crus with the Basal Ganglia . . . . .	421
21. Connections of Posterior Root-fibres with Cells of the Cord . . . . .	422
22. Blood Supply of the Internal Capsule . . . . .	458
23. Localisation of Cortical Motor Areas . . . . .	461
24. Localisation of Cortical Sensory Areas : Outer Surface of Cerebrum . . . . .	461
25. Localisation of Cortical Sensory Areas : Inner Surface of Cerebrum . . . . .	461
26. Effects of Lesions of the Motor Path at Different Levels . . . . .	463
27. Nuclei of Origin of the Cranial Nerves . . . . .	498
28. Diagram to Illustrate the Action of the Ocular Muscles . . . . .	500

## FULL-PAGE ILLUSTRATIONS

The Heart and Great Vessels in Situ . . . . .	248
Situation of Cardiac Valves and Areas of Valvular Murmurs . . . . .	248
Diagram to show the Results of Backward Pressure in Cardiac Disease . . . . .	273
The Optic and Visual Tracts . . . . .	494

LIST OF ILLUSTRATIONS

1	1
2	2
3	3
4	4
5	5
6	6
7	7
8	8
9	9
10	10
11	11
12	12
13	13
14	14
15	15
16	16
17	17
18	18
19	19
20	20
21	21
22	22
23	23
24	24
25	25
26	26
27	27
28	28
29	29
30	30
31	31
32	32
33	33
34	34
35	35
36	36
37	37
38	38
39	39
40	40
41	41
42	42
43	43
44	44
45	45
46	46
47	47
48	48
49	49
50	50
51	51
52	52
53	53
54	54
55	55
56	56
57	57
58	58
59	59
60	60
61	61
62	62
63	63
64	64
65	65
66	66
67	67
68	68
69	69
70	70
71	71
72	72
73	73
74	74
75	75
76	76
77	77
78	78
79	79
80	80
81	81
82	82
83	83
84	84
85	85
86	86
87	87
88	88
89	89
90	90
91	91
92	92
93	93
94	94
95	95
96	96
97	97
98	98
99	99
100	100



# HANDBOOK OF MEDICINE

## FEVER

A FEVER, as the term is semi-popularly used, is a disease characterised by a grave disturbance of the system attended with *increase of temperature* and *diminished secretion*, or, in other words, more fire and greater accumulation of ashes. Fever, in its more limited sense, means simply pyrexia or elevation of temperature, and hence the word has come to be used as the designation of the group of diseases of which pyrexia is one of the striking symptoms. But in this sense, as has just been said, the term is now semi-popular, and the group of ailments known as "the fevers" is to-day included in the wider class of *specific infectious diseases*. Their common feature is that they are due each to a special organism, for the most part bacterial, but sometimes (*e.g.* malaria, sleeping sickness) caused by an animal parasite. In most of these diseases the causative organism has been identified, but the cause of others is still unknown, and these are classed among the specific infections from their general similarity to other members of the group.

Broadly speaking, the most common causes of fever are two : —(1) central disturbance of the heat-regulating apparatus, as in certain forms of nervous disease (*e.g.* lesions of the pons Varolii); (2) the presence in the blood-stream of poisons chiefly of bacterial origin. It is with the latter cause that we have here to do. Suppose we put into a sterile bottle water, sugar, and barm; then close it with a sterile plug, and let it stand in a warm place. The result is the formation of  $\text{CO}_2$  and alcohol, and of a sediment which sinks to the bottom. That is, *the sugar has been changed by the living yeast cells into poisonous compounds*. The process ultimately stops, as so many waste and poisonous products are formed that *the yeast cells themselves are killed*.

Again,—put into a bottle some sugar and a solution of



nitrogenous material (say clear mutton broth), and instead of corking, *expose it to the air*. A scum soon forms on the surface, and on microscopic examination the scum is found to consist of masses of unicellular organisms (bacteria). The fluid itself, once innocuous, has at the same time become *highly poisonous*, and gives off a putrefactive odour.

In these two simple instances of fermentation, the one induced by cells introduced by human agency, the other by bacteria which entered from the air, we have examples of the formation of poisonous products as the result of the vital activity of low organisms. *In vitro* the process tends to spontaneous arrest, for the organisms either are ultimately killed by their own poisonous products (*toxins*), or die from exhaustion of their suitable food. But in the body, where their food supply is unlimited, and where their toxins are washed away by the lymph or by the blood-stream, there is no such natural limitation of the process, and its end is brought about by other means.

If we apply what has been said to the case of any specific fever of definite microbic origin, for instance enteric fever, it will be seen that a period must elapse after the settlement of the organisms, during which they multiply and elaborate their toxins, before definite symptoms are produced. The patient will probably in that stage have symptoms of the kind known as *malaise*. Later, when either the organisms or the toxins have got into the blood-stream, there occurs the protective reaction, accompanied by fever and by the specific symptoms of the particular disease. If the reaction is adequate, there follows a decline of symptoms, leading to convalescence. If it is inadequate, there follows an overcoming of the systemic resistance by the toxins, leading to the ultimate stage, still known as the "typhoid state" (for symptoms, *see* p. 8), and finally to death.

Acute infective diseases arising from microbic infection therefore have—

1. A period of multiplication of the organisms, known as the period of *incubation*.

2. A period known as the *fastigium*, in which the circulation of the organisms or their toxins evokes the characteristic symptoms of the particular disease.

3. A period, in favourable cases, of decline, where the bodily resistance has overcome the microbe attack. The temperature may fall rapidly, by *crisis* (κρίσις, a decision), or more gradually, by *lysis* (λύσις, a remission).

It is not, however, only by the direct action of the organisms



that fever is produced. They may be entirely localised to a particular part of the body, but the products of their vitality may enter the circulation, and cause, besides fever, the phenomena of toxæmia, on which many of the nervous and cardiac symptoms largely depend (delirium, cardiac irregularity or tachycardia, and so on). Fever, it may be more than plausibly said, is a reaction of the previously healthy body against the organisms of disease and their products, or, as Adami puts it, it is "the process of adaptation to such toxic agencies as can be neutralised by the development of antibodies." In many instances the reaction is excessive, and destroys by its over-exuberance the life it was meant to guard. But that it is a protective reaction is shown in the diseases of the old. In them, where the body has no longer its former resistant power, there is little or no fever, even in such affections as pneumonia. A young man, previously healthy, may regain perfect health after a pneumonic attack, although in its duration his temperature may have risen to 105° F. or more. An old man suffering from pneumonia may have a temperature but little elevated, and yet the disease almost inevitably tends towards death. In the one case the febrile reaction expresses the resistance of the body; in the other its absence expresses a failing power to combat either micro-organisms or their toxins.

It is evident from these remarks that fevers must be fought upon two lines :—

- (1) *Prophylaxis*. Microbic life does not flourish except upon suitable soils, which must be rendered unsuitable by measures of hygiene and sanitation. In the actual presence of an epidemic, those exposed to infection can sometimes be immunised by serum or vaccine treatment (see p. 17).
- (2) *Treatment*. (a) The body of a patient attacked by one of the fevers may be looked upon as a suitable soil for the specific organism. We must endeavour to make the soil *unsuitable* by such means as internal antiseptics, tonics, and stimulants; in other words, to fight the bacteria by making the resistance greater, through increasing the vitality of the tissues.
- (b) In certain instances (diphtheria, etc.), by the use of antitoxins, we may administer a direct antidote to the toxins formed by the specific micro-organism. In other instances (pneumonia), by the use of vaccines, we may attempt to promote the bactericidal power of the blood.



To sum up: decrease if possible the number of bacteria attacking, and make the soil more resistant; also support the patient's strength till the fight is finished. Remember, too, that in the fight against the acute infections, victories have been won by prevention rather than by "cure." We owe it to sanitation and to better feeding, not to treatment, that typhus fever is now so uncommon.

**Morbid Anatomy of Fever. General Changes.**—Each specific infection has its special foci, but usually in addition to their peculiar lesions, *all, when fever is prolonged*, show more or less change in the tissues generally, viz.—

1. Except in cholera, where there is an excessive loss of water from the bowel, the blood is dark and more fluid, and the red corpuscles are diminished in number. There is usually an increase in the number of leucocytes, known as leucocytosis.

2. The muscles are dark-coloured, and show granular degeneration (*cloudy swelling*).

3. The heart is softened, perhaps dilated.

4. The viscera are softened and congested, the *spleen, liver, and kidneys* particularly.

5. There may be congestion of the lungs, in part produced by position (*hypostatic congestion*).

**Physiology of Fever.**—In fever we have to do not only with the direct action of the toxins upon the tissues, but also with a disturbance of the heat-regulating mechanism, which in health maintains a balance between heat production and heat loss. An excess of heat production is the result, and this is usually accompanied by a diminished loss of heat. The excess of heat production cannot be accounted for by increased intake and combustion of food stuffs, for the diet in fevers is generally restricted; and it must therefore be due to increased metabolic changes, *i.e.*, to increased disintegration of tissues, and consequent accumulation within the blood of the products of tissue waste, which are imperfectly excreted owing to the engorgement and cloudy swelling of the excretory glands. Thus the disturbance of the heat-regulating mechanism set up by the action of the toxins upon the centre or centres in the brain or cord, or in both, leads to a number of secondary disturbances, of which the following are the chief. The effect upon the central nervous system is shown in headache and delirium



on the one hand, or in apathy, prostration, and ultimately coma on the other. All the secretions, except that of sweat, are diminished in amount. The skin may be dry, or sweating may be profuse, and in the latter instance, although there is great loss of heat by the skin, it is still over-balanced by the excessive heat-production. The urine is scanty, high-coloured, often albuminous (*febrile albuminuria*), and toxic to animals. With a few notable exceptions (enteric, cholera) constipation is the rule. The flow of saliva is lessened, and the mouth and tongue are dry. The diminished secretion of the trachea and bronchi permits the readier passage towards the lung of organisms from the mouth. Respiration is accelerated owing to the effect of high temperatures upon the respiratory centre. There is an increase in the rate of the heart, and the pulse may be either full and bounding, or soft and dicrotic.

Excessive heat production, along with diminished loss, is maintained as long as the curative toxin continues to circulate in the blood. It is this disproportion which accounts for the rigor so often occurring at the beginning of fevers, spasm of the cutaneous vessels producing a sense of cold, and giving rise to shivering by stimulation of the sensory nerves. The rigor ceases so soon as the muscular spasm producing it has caused sufficient heat to warm the skin. From similar causes, the apparent heat of the skin forms no true indication of the internal temperature. Thus in ague the skin is at first quite cold, while the rectal temperature is many degrees above normal.

**Types of Fever.**—In all but the most fleeting febrile disturbances, the fever assumes one of three types. It is continued, remittent, or intermittent. It is *continued* when the temperature throughout the illness remains well above normal, and the daily range of temperature is not much greater than that of health (fig. 1); it is *remittent* when, although the temperature remains persistently above normal, there is a wide diurnal range (fig. 2); and it is *intermittent* when, at some period in the course of the twenty-four hours, the temperature falls to normal or below it (fig. 3). Instances of continued fever are found in typhus, pneumonia, small-pox; of remittent in tuberculosis and the septic diseases; and of intermittent typically in malaria. Fever may intermit periodically as in malaria, where a paroxysm occurs once in each twenty-four or forty-eight hours, or irregularly as in pyæmia. In most instances, and very typically in the hectic fever of tuberculosis, the evening is higher



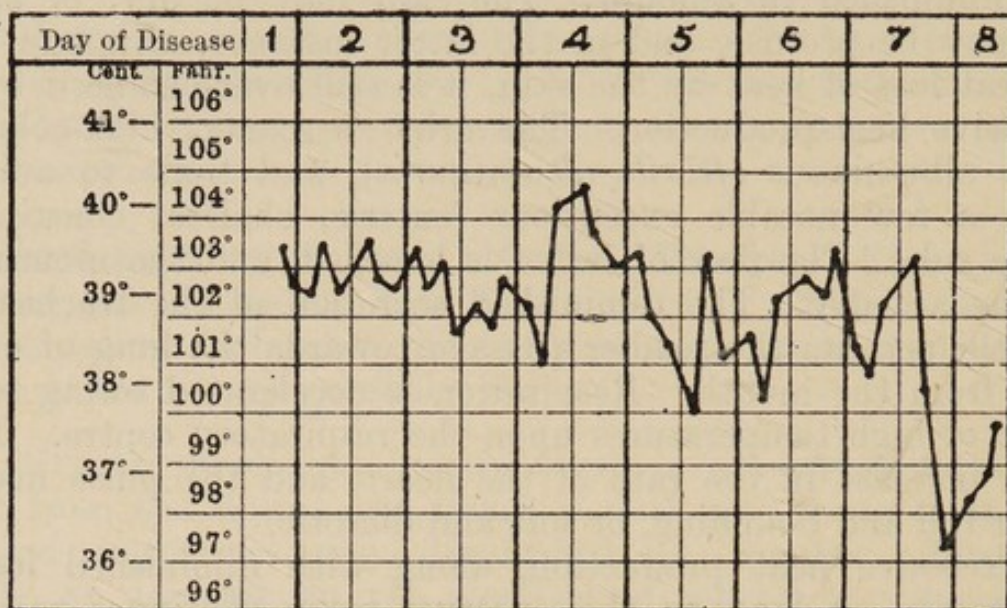


FIG. 1.—Continued fever; from a case of acute miliary tuberculosis.

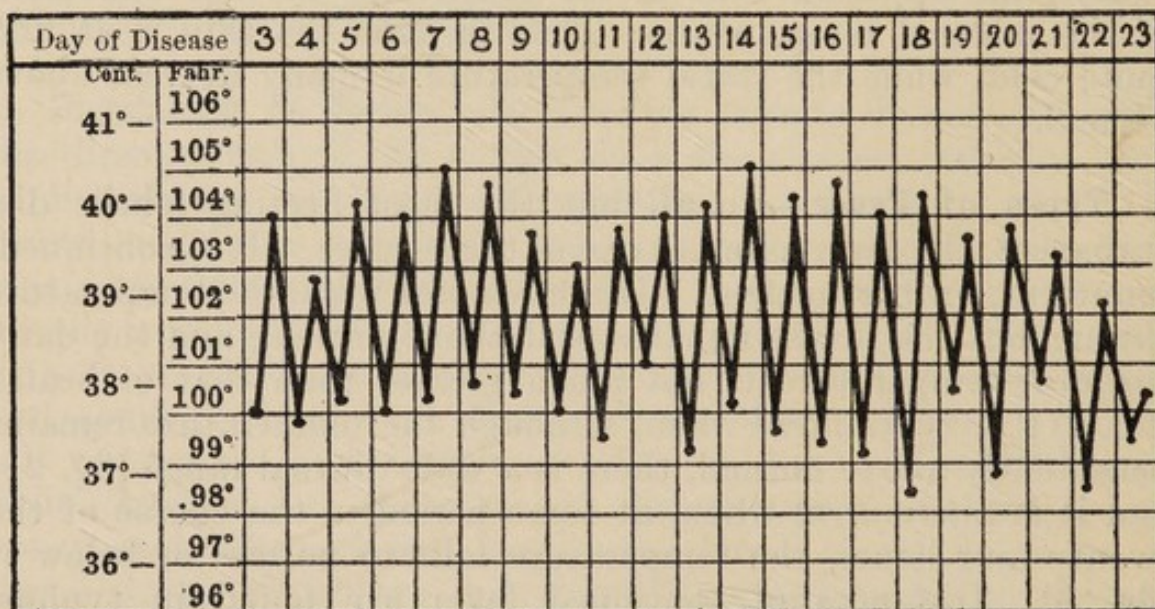


FIG. 2.—Remittent fever; from a case of phthisis pulmonalis.



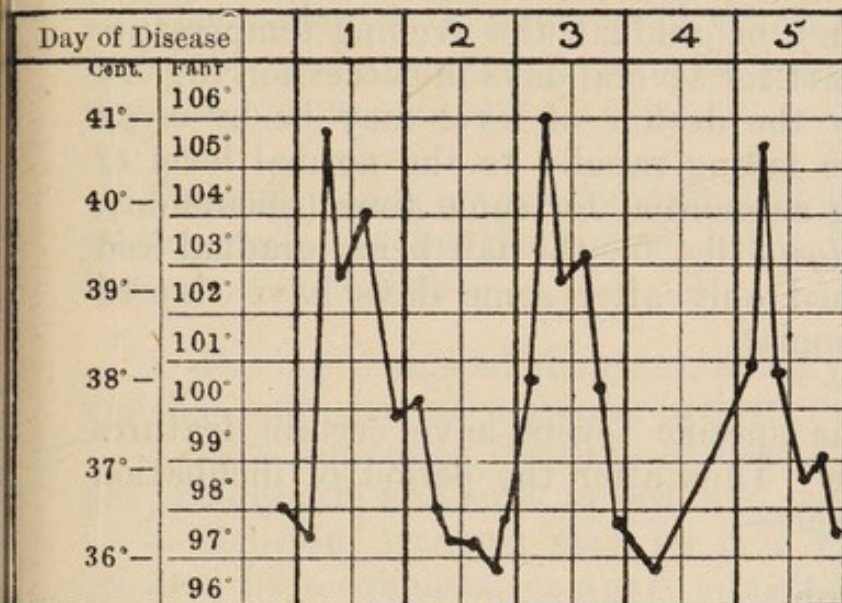


FIG. 3.—Intermittent fever ; from a case of tertian ague.

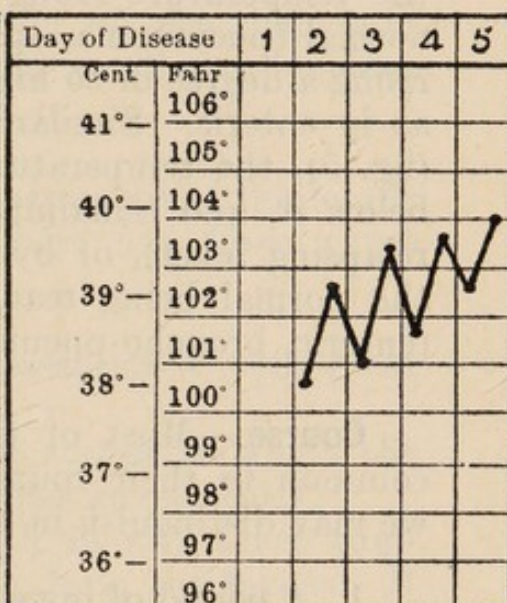


FIG. 4.—Gradual rise of temperature at onset of enteric fever.

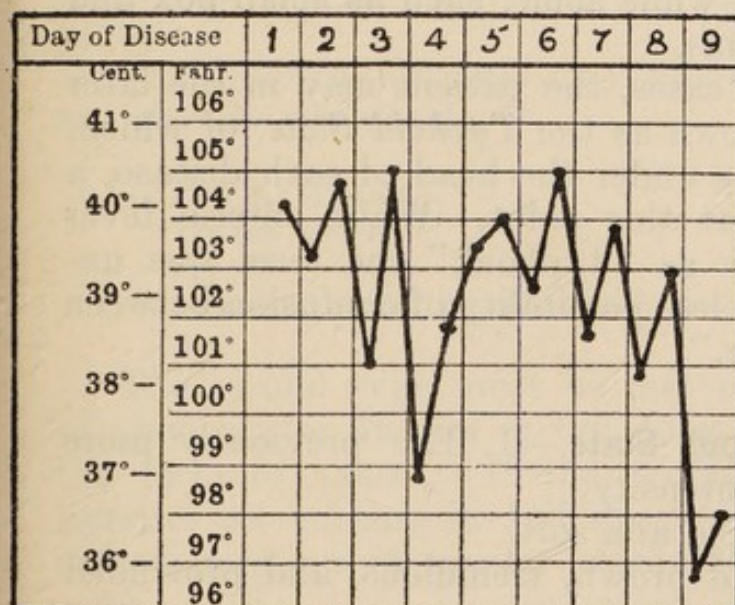


FIG. 5.—Decline of fever by crisis ; from a case of lobar pneumonia.

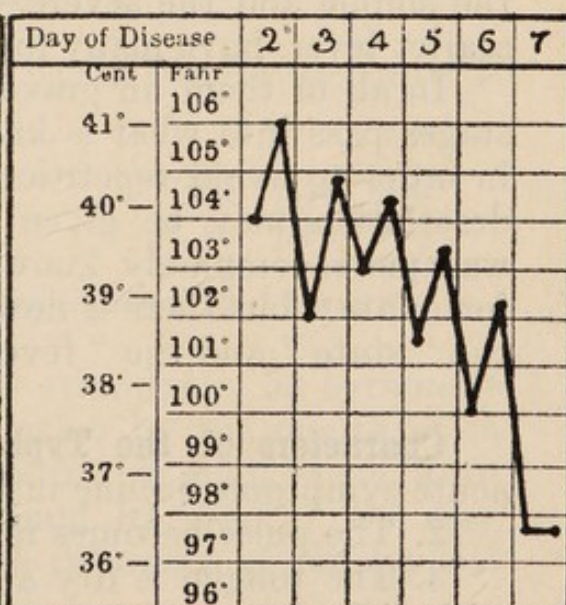


FIG. 6.—Decline of fever by lysis ; from a case of broncho-pneumonia.



than the morning temperature, as it is in health ; but occasionally the reverse is the case. The onset of fever may be sudden, the temperature rising in a few hours to many degrees above normal (pneumonia, ague), or gradual, the evening temperature rising a degree or so higher for several days in succession (fig. 4), as in enteric. Similarly the decline of fever may be by *crisis* (fig. 5), the temperature falling rapidly to the normal level or below it, and remaining subnormal for some time (pneumonia, relapsing fever), or by *lysis* (fig. 6), the fall being gradual and the normal being reached only after some days have elapsed (enteric, broncho-pneumonia).

**Course.**—Most of the specific fevers have certain features common to their course. Thus after the period of incubation we may distinguish in each—

1. A period of invasion.
2. A period of eruption, in which the special symptoms of the disease are prominent.
3. A period of defervescence, leading to recovery.
4. A tendency to complications and sequelæ more or less characteristic of each disease.

All of them have more than one type ; always at least two—the simple and the severe,—while some, such as small-pox and scarlet fever, have many forms.

In all of them, in grave cases, the patient may in the later stages pass into what is known as the *Typhoid State*, of which, in order to avoid repetitions under the head of each disease, a description may be given at this point. While enteric fever was more commonly known as “typhoid,” the term was unfortunate ; but there is now less possibility of confusion between the “state” and the “fever.”

**Characters of the Typhoid State.**—1. The previously more acute symptoms decline in intensity.

2. The pulse becomes rapid and soft.
3. The tongue is dry and brown, tremulous, and protruded with difficulty.
4. Sordes (a mixture of dried mucus and bacteria) collect around the teeth and lips.
5. Muscular prostration increases ; tremors and subsultus tendinum appear.
6. The patient becomes semi-comatose. Though the eyes are open and the pupils dilated, he does not see (*coma vigil*).



7. He picks at the bed-clothes (*carphologia*), and lies in a state of muttering delirium.

8. He tends to slip down to the foot of the bed.

This condition becomes well marked in *all* the malignant forms of fevers, and is very well seen in small-pox, typhus, typhoid, pneumonia, etc.

**Delirium**, being a very common accompaniment of the febrile state, may also be mentioned here. There are three types commonly met with—

1. Raving maniacal delirium, usually associated with great violence and extreme muscular activity.

2. Low muttering delirium of the later stages, in which the patient lies still and sunk in bed, and babbles incoherently.

3. Delirium tremens may be met with in any fever complicated by alcoholism, or apart altogether from the fevers. It is characterised in particular by restlessness, tremor, and hallucinations of sight and hearing.

In the first case we may employ sedatives or depressants, as bromide of potassium, either alone or with chloral hydrate, opium, or antipyretics to control the fever which causes the delirium. Ice to the shaved head, and cold sponging are also useful.

The following formula may prove useful, where the heart is not already weakened—

R Chloral Hydrat. . . . .	5ss.	(grm. 2·0)
Potass. Brom. . . . .	gr. xl.	(grm. 2·5)
Aq. ad . . . . .	5ij.	(cc. 60·0)
One half at once, the other half repeated in one hour if required.		

The second type must be met by stimulants, as carbonate of ammonia or alcohol in full doses either by the mouth or in the form of enema. If the temperature is high, such antipyretics as quinine in full doses should be used. We may indeed sum up in similar words the *treatment of the typhoid state*.

Delirium tremens is to be treated by careful nursing, frequent giving of fluid food, and the use of diaphoretics, sedatives, or hypnotics. Bromide, or bromide and chloral, or paraldehyde in large doses may be used, and if they fail, hyoscine injections beginning with not more than  $\frac{1}{200}$  gr. (grm. 0·00032).



## GENERAL TREATMENT OF FEVER

**I. Medicinal.** — The indications for treatment are the following—

1. Where possible, attack the exciting cause (use of quinine in malaria, etc.).
2. Diminish the heat production.
3. Increase heat loss.
4. Help the secretory organs.
5. Maintain the strength of the heart.
6. Treat complications as they arise.

Under the second heading we may employ antipyretics—

Drugs—(1) Antipyrin group.

(2) Salicylates.

(3) Quinine.

(4) Aconite.

(5) Stimulants.

Of these, such as do not depress the heart are to be preferred, and should generally be given only when the fever is prolonged or excessively high. Cold sponging is comforting, and useful in moderate degrees of fever. In hyperpyrexia cold packs, or better, the cold bath must be used. The patient is immersed in a bath at about blood-heat, and its temperature is gradually reduced to 60° F. by the addition of cold water. When the patient's temperature has fallen to 101° F. he is put back to bed. His pulse must be watched, and stimulants should be at hand.

Under the third heading we may choose—

Diaphoretics—Liq. Ammon. Acet.

Alkaline group generally.

To help the secreting organs—

- (1) Diminish the amount of nitrogenous food, and substitute milk and farinaceous material.
- (2) Keep the bowels moderately open. *Make the urates more soluble* by administration of citrates, tartrates, etc.

And lastly, to support the heart, we may employ—

(1) Digitalis.

(2) Strophanthus.

(3) Strychnine.

(4) Diffusible stimulants, brandy, ether.



No routine treatment can be prescribed, because of the many complications and peculiarities of each case. In most cases we may start with a purge, if there be no contra-indication. Calomel, gr. iii.-iv. (grm. 0.2-0.25), followed in four hours by a saline or a dose of senna, is a useful aperient. This may be combined, in mild cases, with a mixture such as the following, taken every four hours—

R Liq. Ammon. Acet.	. . .	5ij.	(cc. 7.5)
Spt. Ætheris Nitrosi	. . .	5ss.	(cc. 2.0)
Potass. Citratis	. . .	gr. xx	(grm. 1.3)
Aq. Ment. Pip. ad.	. . .	5ss.	(cc. 15.0)

This keeps the skin and kidneys acting, while the citrate renders the urates less irritating.

Slight degrees of fever do not, however, require active treatment, as they are simply the expression of the protective reaction of the body against the cause of the disease.

**II. Hygiene.**—Drugs are frequently the least important factor in successful treatment, and in *all* cases their action is assisted by careful diet and strict attention to hygienic principles.

The essentials are—

1. A large well-ventilated room, with blinds which may be so arranged as to let in plenty of light when wanted, or to exclude light if necessary.

2. An absence of unnecessary furniture, which only serves to form a nidus for retention of the germs. Carpets and curtains are particularly objectionable, and should be removed in any serious fever.

3. A well-trained, non-officious nurse.

4. Absolute cleanliness.

5. Strict attention to physician's orders.

6. Disinfection of all excreta.

**Alcohol in Disease.**—The great diminution in the use of alcohol in disease, which has characterised recent practice in every country, has been attended by a diminution in mortality, and the old routine administration of alcohol in fevers cannot be too strongly condemned. Many cases recover perfectly without a drop of alcohol from onset to convalescence. But the rigorous prohibition of it in every case would seem to be almost as unreasonable. In those who have for long been accustomed to its use in health, its sudden withdrawal in illness may lead to anorexia, and hence to lowered vitality. There are, too, conditions of emergency in which the temporary use of alcohol may



tide over a pressing danger. The individual symptoms must be studied in every case, and to-day, as when it was written, Brunton's summary applies :—If the alcohol tends to bring the patient nearer his normal condition, it is doing good ; if it takes him away farther from his healthy condition, it is doing harm. For instance, if alcohol renders the tongue moist, slows the *quickened* pulse or the hurried breathing, if it renders the skin cooler when hot and dry, and if it lessens delirium and brings on sleep, then use it. If the converse happens, then withhold it, whether in the typhoid state or in any other condition.

Alcohol is chiefly indicated during the small hours of the morning, when attendants are sleepy ; the fire perhaps gets low, and the external temperature is generally lowered.

### IMMUNITY<sup>1</sup>

The subject of immunity has such important relationships to that of the treatment, both preventive and curative, and even in some instances to that of the diagnosis of the specific infections, that a brief account of it is necessary in this place. We have seen (p. 2) that *in vitro* the multiplication of bacteria tends ultimately to spontaneous arrest, either through the toxins they produce or through exhaustion of their appropriate food ; but we have also seen that these methods of arrest do not exist in the body. Yet many people recover from bacterial infections, and some, though constantly exposed to a particular disease, never take it, and are said to be *immune* to it. Such immunity to a particular organism or its toxins is spoken of as *natural immunity*. Again, in many instances, those who have recovered from one of the specific infections are protected against a second attack, and, although in some of the infections no lasting protection is conferred, there must be produced at least a brief period of immunity to render recovery possible at all. In these instances we speak of *acquired immunity*. The protection thus obtained can also be brought about artificially, and of this *artificial immunity* there are two varieties, *active* and *passive*.

*Active immunity* may be produced (a) by repeated injections of attenuated living cultures of the particular organism, or of less than fatal doses of the non-attenuated organism, (b) by repeated injections of the dead organisms, and (c) by similar

<sup>1</sup> This *résumé* of the subject is largely based upon the admirable chapter on Immunity in Muir and Ritchie's *Manual of Bacteriology*.



injections of less than fatal doses of the *toxins* of the organism, *i.e.* the fluid portions of the culture, from which the organisms have been separated by filtration. These doses being gradually increased, or more and more virulent organisms being used, a high degree of immunity is reached, and lasts for some considerable time.

*Passive immunity* is obtained by injecting into another animal the serum of one which has already been actively immunised by one or other of these methods. It is comparatively transitory, and hence an *antiserum* of this nature is most likely to *cure* if injected along with, or very soon after, the entrance of the microbe or toxin that it combats. If the serum injected is derived from an animal actively immunised by the injection of toxins, it in turn protects against these toxins, and hence is known as *antitoxic serum*, but it has little or no bactericidal effect; if, on the other hand, the active immunity has been caused by the injection of living organisms, the serum has little antitoxic effect, but protects against these organisms (*antibacterial serum*).

Each of these classes of serum obviously differs from a normal serum in containing either (*a*) a substance which acts as an antidote to the toxin, or (*b*) a substance which is antagonistic to the organism. Such substances are known as *anti-substances* or *antibodies*, and the toxins or organisms which give rise to them as *antigens*. Besides the toxins, there are many varieties of antigens, probably all, like the toxins, of a proteid nature. Inorganic or alkaloidal poisons are not antigens, *i.e.*, they do not cause the formation of antibodies; but the relation of toxin and antitoxin is like that between an ordinary poison and its antidote in so far that, when they meet, they form an innocuous *chemical* combination. Whether this combination is firm, as Ehrlich maintains, or loose and reversible, as others hold, is for us of secondary importance; the point is that it is *chemical*, not vital, and further, that it is *specific*, *i.e.*, each toxin has its corresponding antitoxin. All the evidence goes to show that the antitoxin is a *normal product of the cells of the body*. It is not newly formed in response to the stimulus of the toxin, but when the toxin enters the body, it is formed by the cells *in increased quantity*, and by them *thrown off into the blood*, where it meets with and neutralises the toxin.

But in many instances the toxin is not separable from the organism, not extra-cellular but endo-cellular, bound up with the body of the bacterium while it is alive. Hence the bacterium itself must be attacked. The case of *antibacterial*



*serum* is more complicated than that of antitoxic serum. In such sera we have to do not only with the formation of two classes of protective substances, one having a direct bactericidal action, and the other (*opsonins*) preparing the organisms for readier absorption by the phagocytes, but also with the formation of substances (*agglutinins*) which produce clumping of the organisms, and of allied bodies (*precipitins*) which precipitate the bacterial substances from solution. Of these the bactericidal substances and the opsonins are of value in prophylaxis and treatment, while the agglutinins, in certain instances, are utilised in diagnosis.

*Bactericidal Substances.*—When organisms are introduced into the body of an individual immunised against them they may rapidly swell up and dissolve (*lysogenesis*), or they may be simply killed. The same effect follows if they are introduced into a non-immunised individual along with a dose of immune serum. *In vitro* it does not occur in the presence of immune serum alone, but it does so when normal serum is added to the mixture. The immune serum remains active even after heating to 70° C., but if the normal serum added to it has been previously heated to 60° C., bacteriolysis does not occur. In each immune serum therefore there is developed a specific comparatively stable substance, the *immune body* or *amboceptor*, which exerts a bacteriolytic effect *only in the presence of an unstable substance existing in normal serum*, and known here and in Germany as *complement*, in France as *alexine* or *cytase*. The complement, which is a substance analogous to the ferments, is not increased during the process of immunisation, but through the immune body is probably brought in contact with, or linked up to, the organism to be attacked. From this linking action the immune body is also frequently known as *copula*.

*Opsonins.*—If a mixture of equal volumes of washed leucocytes and a thin emulsion of a bacterial culture in normal salt solution be incubated together at blood-heat and then examined under the microscope, it will be seen that the cells and the organisms remain separate. But if the mixture be one of equal volumes of leucocytes, bacterial emulsion, and normal serum, the polymorphonuclear leucocytes (*phagocytes*) will be seen to have englobed many of the organisms; and if an immune serum be used instead of a normal serum, the number so englobed will be largely increased. This is the process known as *phagocytosis*. Evidently it has been brought about by the addition of the serum, and it has been shown by Wright and Douglas that the



substances in the serum to which it is due act, not by increasing the activity of the phagocytes, but by "sensitising" the bacteria so that the phagocytes more readily absorb them. They are as it were the caterers for the phagocytes, and hence have received the name of *opsonins* (*οψωνέω*, "I cater for"). Their abundance in the serum is increased by immunisation, and immune opsonin differs from normal opsonin in that it is not destroyed, as normal opsonin is, by heating to 55° C., and in that it is specific, while normal opsonin is not. But both can act by themselves, without the presence of complement, and in this they differ from ordinary immune bodies. (*See infra*, Vaccine Treatment.)

*Agglutinins*.—These are bodies developed in an antiserum by virtue of which, when a small quantity of the serum is added to an emulsion of the living organism, the organisms become aggregated into clumps. The same property is possessed by the serum of individuals convalescent from a microbic infection relatively to emulsions of the organism of that infection, and the reaction is developed comparatively early in the process of immunisation. It is made use of in the diagnosis of enteric fever (*q.v.*), paratyphoid fever, and Mediterranean fever.

**Theories of Immunity.**—There are two which hold the field and are mutually complementary—Ehrlich's and Metchnikoff's. Ehrlich's is based upon the facts of organic chemistry, Metchnikoff's on the vital activities of the phagocytes. In *Ehrlich's side-chain theory* the protoplasmic molecules of which the cell is made up are regarded as composed of a central "executive nucleus," and a large number of "*side-chains*" (as they are called by the organic chemist) or *receptors*, which are ready to combine with or "fix" other atomic groups with which they have a chemical affinity. These receptors are the active elements in the nutrition of the cell, to which they fix the food-stuffs, and are of varying degrees of complexity. The molecules of *toxins* possess, on this theory, two combining groups; one, the *haptophore* group, uniting them to the cell, and the other, the *toxophore* group, producing the toxic action. The haptophore group being united with its appropriate receptor, toxic action results because the cell possesses also a *toxophile* receptor, to which the toxophore group becomes fixed. If it does not possess such receptors, no toxic action follows. In the production of immunity by sub-lethal doses of toxin, the toxin molecules become fixed to the side-chains (receptors) of the cells by their haptophore groups, and these receptors become lost for the



purposes of the cell. The defect thus created excites regenerative processes, new receptors of the same nature are produced, and the cell is thus ready once more to deal with a fresh quantity of toxin. But such regeneration is always in excess of the actual requirements of the cell, and the receptors are finally produced in such numbers that they are shed off into the blood, forming free antitoxin. There they unite with the haptophore groups of the toxin molecules, which are thus prevented from becoming anchored to the cells, and hence have no opportunity for the employment of their toxophore groups.

*Metchnikoff's theory* assigns the protective rôle chiefly to the phagocytes, of which he distinguishes two varieties, the *microphages* or polymorphonuclear leucocytes, and the *macrophages* (large lymphocytes, endothelial cells, connective tissue corpuscles, etc.). The former are the chief protective agents in the acute infections, in most of which there is an active leucocytosis; and as Metchnikoff has shown, the process of immunisation is also attended by phagocytosis. The destruction of the bacteria which the phagocytes englobe is performed by their digestive ferments (cytases), which are normally intracellular, but are set free in the blood by injury to the cell, as when the blood is shed, and are then called *alexines* (complement). An antibacterial serum operates through its immune body, which is fixed by the bacteria, and thus acts as an auxiliary ferment to the alexines. Lysogenesis occurs only when the alexines are set free in the blood by damage to the leucocytes (phagolysis); in other cases the bacteria are disposed of by intracellular digestion.

It will be seen that on one or other of these theories the facts of *natural immunity* may be variously explained, and indeed no one explanation covers them all. Immunity to the attack of a particular organism is explained either (1) by the bactericidal power of normal serum, or (2) by Metchnikoff, as due to phagocytosis, and in immune animals the organisms are found to be thus destroyed in large numbers, while there is little phagocytosis in the susceptible. But the work of Wright and others upon opsonins has tended to lessen the importance of the cell, and to show that the activity of the phagocytosis is determined by the relative abundance of opsonins, *i.e.*, that the essential bactericidal power is to be found in the serum. Yet it is evident that the opsonins must be ultimately derived from cells, and probably from the leucocytes. The serum does not secrete its own contents. Its bactericidal power is believed to be due to substances derived from the spleen and lymphatic glands and other leucocyte-containing tissues, substances analogous to



the cytases of Metchnikoff. But this property of the serum has been found not to vary in proportion to the degree of immunity, and in some instances where the bactericidal power is great *in vitro* there is no immunity *in vivo*.

Immunity to *toxins* is explained on Ehrlich's theory either by want of chemical affinity between the receptors and the haptophore group of the toxin molecule, or by the absence of toxophile receptors in the side-chains of the cell, making it impossible for the toxin to exert its action through the toxophores.

**Serum Anaphylaxis.**—Under this name is known a condition of supersensitiveness, induced when a second injection of toxin or of serum is given not less than twelve days or a fortnight after the first. The substances which induce it are those which cause the formation of antibodies, namely, antigens, and it is not produced by chemical poisons. The sensitising substance is not, however, the toxin, but is contained in the serum in which it is injected. The reaction is specific, and occurs only when the same serum is used for the second as for the first injection. In the guinea-pig severe symptoms, especially affecting the nervous system, may be followed by death. In man a somewhat similar condition may occur, though it does not end fatally. The theoretical explanations are as yet conflicting, but the possibility of such an occurrence is of practical importance in serum treatment (*see Serum Disease*, p. 59).

## VACCINE TREATMENT

We have seen (p. 12) that one of the methods of securing active immunity is the repeated injection of dead bacterial cultures. On this procedure is based Wright's method of vaccine treatment. Upon the injection of a suitable dose of a standardised sterile bacterial emulsion (in other words, a *vaccine*), there follow (1) a brief period (*negative phase*) in which the opsonic content of the serum is lowered, the existing opsonin being partially used up in sensitising the organisms, and (2) a longer period (*positive phase*) in which opsonins are produced in increased quantity, in response to the stimulus. This increased production is maintained for from ten days to a fortnight or more (*high tide of immunity*), after which the opsonic production declines to the normal. A second injection during the declining phase produces a repetition of the process, and thus maintains the opsonic content of the serum above the normal level, thereby rendering the organisms of the particular infection more amenable to phagocytosis. A second injection before the preliminary negative phase has passed has a cumulative effect, and dangerously lowers



the opsonic content, producing symptoms of malaise and pyrexia. The amount of opsonins in any individual for a particular organism is measured by (1) counting the number of bacteria ingested at 37° C. by a definite number of leucocytes mixed with the serum of that individual, and (2) comparing this with the number ingested when normal serum is used. The ratio between the patient's phagocyte count and the normal phagocyte count (taken as unity) is the *opsonic index*.

In health, for each individual, the opsonic index is very constant; but it may in different individuals remain permanently either slightly below or slightly above unity, the limits of normal variation lying between 0·8 and 1·2. Variation beyond these limits is pathological. In disease, the index is low in localised infections, and either high or fluctuating in systemic infections. The low index of localised infections precedes and favours the onset of the infection. It continues low because the organisms remain local, and as they do not enter the blood no positive phase or period of immunity is produced. It is high or fluctuating in systemic infections, because the organisms enter the blood and so produce a series of auto-inoculations with negative and positive phase. The doses, however, are sometimes excessive, or the intervals between them too brief, so that the dangerous effects of a prolonged negative phase are obtained; but in other cases the repeated auto-inoculation leads to successful immunisation, and hence to recovery.

Treatment by vaccines is based upon these considerations, the object being to raise the opsonic content of the patient's blood against the organism of the particular disease. When it is practicable, the inoculations should be controlled by estimation of the opsonic index, and they must not be repeated during a negative phase. Although hitherto principally used in local infections, there is an increasing body of evidence to show that the treatment may also give valuable results in some at least of the systemic infections, *e.g.*, pneumonia, enteric fever, septicaemia: but the matter is still *sub judice*. In the prophylaxis of the infectious diseases, on the other hand, the vaccine treatment occupies a prominent place. (*See articles on Enteric Fever, Plague, Cholera, etc.*)

A recent development of the vaccine treatment is the introduction of *sensitised* vaccines. These are bacterial emulsions which are first treated with the serum of an animal immunised against the particular bacterium, and then washed free of the serum. The immunity produced by injection of such sensitised vaccines is said to be of long duration, and the local toxic effects are said to be very slight.



## SPECIFIC INFECTIOUS DISEASES

## INCUBATION AND RASHES

The period of incubation in the specific infections is the time intervening between the reception of the specific virus and the onset of symptoms.

The *average* duration of the period is here given for the more important diseases—

Cholera . . . . .	4 to 5 days.
Diphtheria . . . . .	2 days.
Enteric fever . . . . .	10 to 14 days.
Influenza . . . . .	3 days.
Measles . . . . .	14 days.
Plague . . . . .	3 to 5 days.
Scarlatina . . . . .	2 to 4 days.
Typhus fever . . . . .	12 days.
Varicella (chicken-pox) . . . . .	14 days.
Variola (small-pox) . . . . .	12 days.

(DAWSON WILLIAMS.)

The period of incubation must be distinguished from the period of onset of the characteristic rash, which may occur along with the first symptoms, or some time later.

The following table gives the usual time of appearance of the rash.

Chicken-pox . . . . .	1st day of the disease.
Scarlatina . . . . .	1st or 2nd day „
Small-pox . . . . .	3rd day „
Measles . . . . .	4th day „
Typhus . . . . .	5th day „
Enteric . . . . .	7th to 12th day „

Most rashes disappear on pressure, *if not hæmorrhagic*. If hæmorrhagic they do not disappear on pressure *or after death*.

## ENTERIC OR TYPHOID FEVER

A specific infectious fever marked clinically by diarrhœa and a rose-coloured rash (appearing in successive crops), running a prolonged course of about twenty-one days, and ending by lysis.



It is accompanied by characteristic ulcerations of the small intestine, and by enlargement of the spleen and mesenteric glands.

**Etiology.**—The disease is most prevalent in the autumn months. It attacks both sexes, principally between fifteen and twenty-five years of age. It is rare in infancy or over the age of sixty. Second attacks are uncommon.

*The poison is conveyed principally by—*

1. Contaminated water (or ice).
2. Milk; uncooked vegetables grown on infected soils; food contaminated by flies.
3. Shell fish, especially oysters, grown on river-beds polluted by sewage.
4. Direct contact with stools of patients.
5. Soiled linen of patients in public laundries (stools or urine).
6. Contamination of the soil through defective drains or cesspools.
7. "Typhoid carriers."

The possibility of air-borne typhoid must be remembered, especially in hot countries. The organism may be drawn into the mouth in dust, and afterwards swallowed.

The stools or urine when *freshly passed* are said to be less virulent than after some time has elapsed.

In certain cases, after recovery, the bacilli may persist for years in the intestine or gall bladder, and possibly also in the prostate gland, from which they may reach the urine. The dejecta of such subjects, who are known as "typhoid carriers," may be constant sources of infection.

Sewer gas, filth, etc., do not of themselves cause typhoid fever, but form a suitable soil for the multiplication of the organisms.

*The Specific Germ*, known as the bacillus of Eberth or *bacillus typhosus*, is a Gram-negative organism, *short, thick*, and with rounded ends. It is highly mobile, and possesses numerous flagella. It does not form spores. Cultures are easily destroyed by corrosive sublimate 1-2500. During life the bacilli may be found in the rose-spots, the stools, and the urine, and are discoverable with some difficulty in the peripheral blood, from which they can usually be isolated by culture. *Post mortem* they are found in the intestinal and mesenteric glands, the spleen, the blood, the gall bladder, and frequently in other parts of the body.



**Morbid Anatomy.**—Principally inflammation of the lymphoid tissue of the lower portion of the ileum, with more or less catarrh throughout the bowel.

*Peyer's Patches*—

1st Week.—Are swollen through infiltration of leucocytes, the surfaces raised and fawn-coloured. The infiltration involves the submucous coat, and may extend to the muscular layer. The lesions are most numerous at the lower end of the ileum.

2nd Week.—The surface becomes abraded; sloughs form, which are often bile-stained.

3rd Week.—The sloughs come away, leaving ulcerating surfaces. Typical typhoid ulcers, with undermined edges, are thus formed. A few solitary glands undergo the same process. At the end of the week the ulcers begin to granulate, but healing is usually slow.

*The Mesenteric Glands* may undergo the same changes, but more often become swollen, red, and tender only, or break down into cheesy masses.

*Other Organs.*—The spleen and liver are enlarged; the heart is soft and flabby. The voluntary muscles undergo granular degeneration; in fact, the changes are those found after death from high temperature, etc. (*See p. 4.*)

**Symptoms.**—*Insidious onset*—headache, possibly epistaxis, increasing weakness till the patient is compelled to take to bed.

1st Week.—The temperature usually, but not invariably, rises in the so-called “staircase” manner, *e.g.*, rising two degrees in the evening and falling *one* in the morning (*see fig. 4, p. 7*). The malaise becomes more marked, and exhaustion increases with the onset of—

- (1) Diarrhœa—the stools quickly assuming their characteristic appearance. The bowels may, however, be confined throughout.
- (2) Tenderness in the right iliac fossa, tumidity of the abdomen, and enlarged spleen. This enlargement is more obvious at a later stage.

The specific rash appears on the seventh to twelfth day, and may continue throughout the disease.

The 2nd Week is marked by the symptoms becoming more aggravated; the temperature remains at a uniform level. A



tendency to *typhoid state* comes on, rambling delirium may occur towards the end of the week, and death may take place.

The 3rd Week is marked by even more profound symptoms. The heart is weak and rapid, there is hypostatic congestion of the lungs, the abdomen is distended, and the typhoid state may be marked. Leucopenia (diminution of leucocytes) is common. Perforation is more likely to occur at this than any other period, and intestinal hæmorrhage, epistaxis, or pneumonia may end the scene.

4th Week.—The temperature gradually becomes normal, and usually convalescence may now be said to *commence*. Frequently, however, relapses occur, often about the tenth day after defervescence. They are usually comparatively mild and brief, but may end fatally.

#### **Varieties of enteric fever.—**

1. The “ambulatory” type, in which the patient may be able to go about, sometimes even during the entire illness, or until some complication arises.
2. Cases with sudden onset, often with marked nervous symptoms (headache, etc.).
3. Cases with termination by crisis in the second week (abortive type).
4. Cases where defervescence is postponed till the fifth or sixth week.

#### **Complications.—**

1. Hæmorrhage ; from *bowel*, nose, or mucous surfaces.
2. Perforation, which may occur even in mild cases. It is commonest in the third and fourth weeks.
3. Peritonitis, with or without perforation.
4. Meteorism or tympanites (excessive distension of the abdomen).
5. Thrombosis, usually of the femoral vein, and embolism.
6. Neuritis, and later “typhoid spine.” Meningitis is rare.
7. Bronchitis, and pneumonia, lobar in the early stages, later catarrhal.
8. Acute nephritis.
9. Early “typhoid state,” or early cardiac failure.
10. Bed-sores.

**Special Points.**—The *Rash* comes out, from the seventh to the twelfth day, in successive crops of small rose-coloured spots



raised above the surface, and slightly convex. These appear first on the abdomen and chest; and they fade on pressure. They may be present on the shoulders and back, and not anteriorly. Each spot lasts about four days, and the eruption may persist till the end of the fever. Frequently no rash appears (in about 30 per cent. of the cases).

The *Ulcer* of Typhoid fever—(1) Lies in the longitudinal axis; (2) Edges—thin, undermined; (3) Situation—last yard of the ileum; the ulcers are most numerous near the ileo-colic valve. The distribution may be more widespread.

Its *base* may be formed of—(1) Submucous coat; (2) Muscular tissue; or (3) Peritoneum only.

The ulcers show a tendency to perforate, but *do not cause constriction after they heal*.

The *Stools* are liquid, abundant, and foetid, like pea soup in appearance, and frequently contain, besides the bacillus and faecal matter, blood, shreds of mucous membrane, and sloughs. When hæmorrhage occurs, they become black or tarry in appearance, and sticky. In severe hæmorrhage the temperature falls suddenly, and collapse symptoms appear.

*Leucocytosis* is not a feature of the uncomplicated fever. When it does occur, it indicates some inflammatory complication.

*Signs of perforation*.—Usually there is sudden and severe pain, but sometimes pain is absent. There are rapid and extreme distension of the abdomen, absence of abdominal respiration, tympanitic percussion note, and possibly absence of hepatic and splenic dulness. There are also signs of *shock*, anxious look, very rapid pulse, rapid respiration. Later fluid is present in the abdomen; and leucocytosis may take the place of leucopenia.

*Widal's reaction*.—Serum from a case of enteric fever will, when mixed with an emulsion of *B. typhosus*, produce agglutination of the bacilli into clumps, even when the serum is diluted to 1 in 100 or further with sterile bouillon. To carry out the test, blood is collected from a puncture in the skin by a small pipette, in which it is allowed to coagulate, and the serum is then blown out and dilutions of 1 in 20, 1 in 50, and 1 in 100 are made. Equal parts of these and the emulsion are then mixed together, and the result watched under the microscope in a hanging-drop preparation, or in a small glass tube sealed at one end, and set vertically. If the case be enteric, agglutination should take place



in half an hour to an hour. A control experiment with healthy serum must be made at the same time. The test does not give positive results, as a rule, until at earliest the seventh or eighth day. A negative result does not exclude enteric, and positive results are occasionally found in other diseases.

**The Diazo-reaction of Ehrlich.**—To forty parts of a saturated solution of sulphanilic acid in 5 per cent. hydrochloric acid add one part of a 0·5 per cent. solution of sodium nitrite. Shake up a drachm of this with a drachm of the patient's urine in a test tube, and float upon the surface a layer of liquor ammoniæ. A ring of red coloration is formed at the point of contact. On shaking, a pinkish froth forms at the surface. The reaction appears earlier than Widal's (from the third day onwards), but is not so valuable, as it may occur in typhus, tuberculosis, malaria, and some other febrile diseases. Its persistent absence is a strong argument against the existence of enteric.

**Russo's test.**—Four drops of a 0·1 per cent. watery solution of methylene blue are added to 4 or 5 cc. of the patient's urine, and thoroughly mixed. When held against the light the resulting mixture has an emerald green colour without a trace of blue. The reaction is somewhat more sensitive than Ehrlich's, and appears about the same time; but it also occurs in measles, small-pox, and sometimes in tuberculosis.

During the first week or ten days, a positive diagnosis may be made in most cases by cultivation of the bacillus from the blood or stools.

The diagnosis from typhus fever is usually simple. In typhus the onset is sudden, the fever high and continuous, the rash conspicuous, the nervous symptoms marked, the typhoid state develops early, and defervescence is abrupt. Widal's reaction is negative. For the diagnosis from acute miliary tuberculosis, see p. 116.

**Paratyphoid Fever.**—This disease is clinically almost identical with enteric fever. Enlargement of the spleen, rose-spots, diarrhœa, and sometimes hæmorrhage are its symptoms, and its course is like that of enteric, though it may be briefer. Ulceration may be present, but perforation does not occur, and the mortality is small. It is due to the paratyphoid bacillus, which microscopically resembles the bacillus of Eberth, but differs from it in cultural characters and agglutinating reaction.

**Special Points of Treatment.**—Here we have to do with a diseased alimentary canal: our treatment must be mainly dietetic. Give no solid food, or that which would not readily



pass through a fine sieve. Milk, *if it agrees*, albumin-water made from white of egg, beef-tea, and chicken-broth are the principal foods. Milk must not be used to relieve thirst, but must be given as a food only, at regular intervals, and in definite doses. If it disagrees, a 5 per cent. solution of gelatin may be used instead, and has been found of considerable value. Thirst should be met by water, mineral waters, lemonade, or Potus Imperialis (for formula see footnote<sup>1</sup>). Of late some authorities have advocated a light solid diet throughout, but nothing must be given that would increase the risk of perforation. Such additions as arrowroot, cornflour, custard, mashed potato, and whipped eggs are permissible. The stools should be daily inspected. If undigested curd is found, milk is being given too often, or the gastric function is impaired. The milk may then be given with lime-water or barley-water. Beef-tea should be sparingly used, lest it excite diarrhœa.

Alcohol is required mainly in the later stages, when the typhoid state has set in, and the heart is weak. Many cases do well without it altogether. Other stimulants are strychnine, ammonia, ether, etc.

**Medicinal.**—General principles hold good. If the diarrhœa becomes excessive give bismuth and opium, or lead acetate and morphia, or an enema of starch and opium. When the motions are very offensive intestinal antiseptics (calomel in small doses, salol, etc.) may be given. They are also useful in meteorism.

If constipation be troublesome, give enemata. No purgatives should be given after the first week.

Hæmorrhage—opium, lead acetate, or calcium lactate gr. xv (grm. 1·0) every four hours, hypodermic injection of morphia or ergotin, ice-bag to the cæcum. Subcutaneous or intramuscular injection of normal serum (10 cc.) is valuable in severe cases.

Perforation.—The main chance of recovery lies in early laparotomy and suture of the affected bowel. Every hour lost after the diagnosis is made, and the initial shock has passed off, increases the danger to life. Cases operated on within twenty-four hours may recover; later, recovery is very rare. In anticipation of the operation, morphia may be given to relieve pain and diminish peristalsis.

Bed-sores—water-bed, cleanliness, stimulant and antiseptic lotions, dry dressings if the slough is large.

High fever—quinine and cold baths. The antipyrin group

<sup>1</sup> Bitartrate of potassium,  $\mathfrak{z}$ i (grm. 4·0); syrup of lemon,  $\mathfrak{z}$ iss (cc. 45); water to  $\mathfrak{z}$ xx (cc. 600).



is dangerous to the heart, but aspirin in doses of five grains (grm. 0.3) every four hours is useful in some cases.

The stools and urine must be carefully disinfected.

During convalescence the diet must be increased with the utmost caution, and the possibility of relapse must always be remembered.

**Specific Treatment.**—Chantemesse's anti-typhoid serum has been successfully used in France. Prophylactically, Wright's vaccine, in two doses (500 million and 1000 million organisms) with an interval of ten days, has proved of much service, and the results obtained from its employment in the British Expeditionary Force conclusively demonstrate its efficacy. The vaccine is also increasingly used in treatment, and Army statistics show a much reduced mortality. The dose varies in different hands from 100 million to 500 million every fourth day.

## TYPHUS FEVER

An acute specific contagious fever, characterised by sudden onset, marked nervous symptoms, and a maculated rash, and ending by crisis.

**Etiology.**—The specific organism is unknown, and is probably ultra-microscopic, but the disease has been transmitted to the chimpanzee by typhus blood, and from it to lower apes. Body-lice can carry it from an infected to a healthy ape. The guinea-pig is also susceptible. The older names,—jail-fever, ship-fever, etc.—suggest contributory causes. It occurs amongst the poor, in overcrowded and dirty districts, and affects principally those between ten and thirty years of age. It is very infectious, by contact or through bedding and clothing. Patients remain infective throughout the illness and during convalescence. Comparatively rare under modern sanitary conditions in time of peace, it may become epidemic in such circumstances as those of Serbia in the present war. "Brill's disease," which occurs in New York, and the *tabardillo* of Mexico, have now been shown to be typhus in an attenuated form.

**Morbid Changes.**—Those of intense fever, plus the petechial rash. (See p. 4.) Leucocytosis is not constant.

**Symptoms.**—After the incubation period (twelve days) the fever rapidly develops. The early symptoms are intense headache, nausea, and vomiting, with rapid elevation of temperature.



There may be rigors, pains all over the body, constipation, contracted pupils, thick-furred tongue, and rapid exhaustion. The skin gives off a mousy odour. Delirium is at first noisy. The face is congested, and the appearance dull, heavy, and apathetic. The rash appears on about the fifth day—first, on the abdomen, the upper part of the chest, and the extensor aspects of the hands and wrists. Later, it spreads over the trunk and extends to the extremities and face. As the fever progresses, the typhoid state comes on rapidly: the delirium becomes of the low muttering type; the pupils, before contracted, may now be dilated; coma vigil, retention of urine and paralysis of the sphincters, or the grave complications tabulated may appear and help to terminate the disease. If the patient does not succumb, the temperature falls, usually about the thirteenth or fourteenth day; profuse sweating, a critical diarrhoea, and an abundance of urates usually usher in a crisis; after which the patient gains strength rapidly, and may be, in a short time, in better health than before the attack. Second attacks are very rare.

*The Rash*—comes out rapidly about the fifth day. It shows—

1. A sub-cuticular mottling, *dusky-red* in colour (*measly eruption*).
2. Distinct papular rose-spots, which tend to *become petechial*, and, therefore, disappear neither on pressure nor after death.

#### Complications.—

1. Retention of urine.
2. Hyperpyrexia.
3. Gangrene of extremities, or bed-sores.
4. Hypostatic congestion of the lungs, bronchitis, and broncho-pneumonia are the most common complications. They are often latent.
5. Parotid bubo, and pyæmic abscesses.
6. Thrombosis of the femoral vein, more common than in typhoid.

It should be noted that, notwithstanding the severity of the cerebral symptoms, meningitis is rare. So is nephritis, but albuminuria may be present.

**Treatment.**—*Special points to bear in mind.*—As the disease is intensely contagious, free ventilation is indicated both in prophylaxis and treatment; and a plentiful supply of air is



perhaps more necessary than in any other fever. The position of the patient should be changed at times, to avoid hypostatic congestion. Experienced nurses should be constantly present.

This fever seldom calls for lowering treatment, and frequently stimulants are required from the beginning. They should not be given as a matter of routine, but to meet special indications. Employ the general principles detailed in the opening chapter. A good mixture is the following—

R Acid Hydrobromic Dil.	. . . . .	℥ss.	(cc. 15·0)
Quininæ Sulph.	. . . . .	℥ss.	(grm. 2·0)
Aq. ad	. . . . .	℥viij.	(cc. 240·0)

Sig. :—A tablespoonful every four hours.

The contracted pupils and delirium suggest absence of excessive light, at least in the earlier stages. All noise must be avoided in the sickroom. Ice or cold affusion to the head is often useful, and chloral alone or with bromide of potassium, may be given in maniacal delirium. Both for enteric and for typhus, Osler strongly advocates hydrotherapy, *i.e.*, the systematic use of baths cooled down to 60° F. (See Treatment of Fever, p. 10.) The food must be liquid, and as plentiful as the assimilative powers will admit. Milk, or milk and soda-water, and broths may be freely given, and when necessary, white of egg.

## RELAPSING FEVER

A specific infectious disease, occurring in epidemics, and characterised by terminating suddenly on the sixth or seventh day, but *followed by a relapse after an interval of a week*. The fever is always associated with specific organisms in the blood.

**Etiology.**—Epidemics have a close connection with overcrowding and destitution. The disease is endemic in parts of India. It used to be epidemic in temperate climates, and especially in Ireland during periods of famine. Like typhus, it occurred amongst the poor and filthy, but was more associated with *poverty and famine* than filth. It attacks all ages, but males more than females. It is transmitted from person to person, and may be carried by *fomites* (clothing, bedding, etc. which has been in contact with the patient). It is probably transmitted by the body louse, and possibly also by the bed-bug.

**The Organism.**—The *spirillum* or *spirochæte Obermeieri* is a long, slender spiral organism with pointed ends. Its length varies from 10 to 40  $\mu$ , and the spirals number about 10. It is in constant movement of a lashing



character. The spirilla, which tend to adhere around the red corpuscles, are absent during the non-febrile interval, but return again when the relapse occurs. When they disappear from the blood, they accumulate in the spleen, and according to Metchnikoff they are there destroyed by phagocytosis. This process is aided by the development of antibodies in the serum.

Noguchi has successfully cultivated several varieties of the spirochæte on sterile ascitic fluid to which a piece of fresh tissue has been added. They multiply both by longitudinal and transverse division.

The incubation period varies between one and twenty-one days.

**Morbid Anatomy.**—That of intense fever. The spleen is notably enlarged.

**Symptoms.**—The fever is ushered in suddenly, with rigors, frontal headache, backache, and rapid elevation of temperature, which may reach, even on the first day, 104° F. The pulse is very rapid, and respirations are also quickened. The tongue has a marked white fur, and there is much thirst. The skin may be dry or sweating. The *spleen* and liver are enlarged and tender. Jaundice is common. About the fifth night the symptoms become greatly aggravated and the temperature may reach 107° or 108° F.; the delirium is increased, and a fatal termination seems imminent, when a profuse sweating takes place, the bad symptoms rapidly abate, and the crisis is established. In a few hours the patient feels comparatively well; he is ravenously hungry, and has apparently fully recovered. Then in about a week, he is seized with symptoms similar to those of the first attack. Usually the second attack, however, *runs a less severe course, and is of shorter duration than the first attack*, but sometimes the exhaustion is so profound, that the “typhoid” condition rapidly supervenes, and a fatal issue results. Two, or even more, relapses have occurred. Convalescence is usually slow. The disease is much less fatal than typhus, but immunity is short, and second attacks are common.

**Complications.**—

1. Bronchitis, pneumonia, and pleurisy.
2. Dysenteric diarrhœa.
3. Severe jaundice.
4. Ophthalmia.
5. Hæmorrhages.
6. Pregnant women usually abort.



**Special Points to note—**

1. The spirochæte.
2. Absence of rash. (There may be petechial spots.)
3. The speedy, *apparent* recovery after the first attack.
4. The slow, *real* convalescence after a relapse.
5. Association of the fever with famine.

**Treatment.**—Salvarsan used intravenously has given excellent results in Russia, causing an early crisis and preventing relapses. Other indications are to relieve symptoms and maintain the general strength. Quinine and the mineral acids may be given. Opium or morphine may be required for the severe pains. Collapse at the crisis must be met by stimulants. As many of the patients have been in a state of starvation, only fluid and easily digestible foods are at first allowable, and the supply must be very carefully increased. In a disease conveyed by parasites, disinfection of the patient's clothing is an obvious measure.

**VARIOLA OR SMALL-POX**

An acute, infectious disease, characterised by a rash, which usually appears on the third day, and runs through four stages, viz.:—(1) Papular; (2) Vesicular; (3) Pustular; (4) Drying up or Scabbing.

**Etiology.**—The disease is common to all ages and both sexes, and may attack the foetus of an infected mother. Planted in a virgin soil it is very virulent. Complete or partial immunity is conferred by successful vaccination.

The contagion spreads by contact with fomites, patients or those who have been in touch with them, and also by contact with the contents of the pustule, with scabs, and especially with the scales of desquamating skin. As the primary fever declines on the third day, when the rash appears, the patients in the early stages frequently walk about and apply at hospital for advice respecting a "rash," and they may thus infect all in the waiting-room. A mild case of "varioid" may cause a virulent infection. The virus lingers long in infected places, and it is given off from the dead. It is probable that the disease is contagious even before the eruption appears.

**Specific organism.**—No specific organism has as yet been unquestionably demonstrated, although Guarnieri describes a small protozoon-like organism from 1 to 8  $\mu$  in diameter, occurring in the deeper epithelial cells of the skin-lesions, and also in the corneal ulcerations, which he names the *Cytoryctes variola*. The appearances are probably due to nuclear changes in the cells, but similar changes have been seen in no other skin-lesions.



**Morbid Anatomy.**—The general changes in the tissues are those of fever. The spleen is much enlarged. In hæmorrhagic small-pox there are hæmorrhages into internal organs, and on serous and mucous surfaces.

The *Rash* affects the mucous surfaces as well as the skin. (1) The *papular stage* is marked by proliferation of the cells of the rete mucosum, with an exudate, partly serous, partly cellular, which compresses the peripheral cells into trabeculæ, and so forms (2) a loculated *vesicle*. At the same time coagulation-necrosis occurs in the centre of the papule, which, when it becomes a vesicle, is thus *umbilicated* or depressed in the centre. The cutis vera becomes hyperæmic, and this leads to infiltration of leucocytes, forming (3) the *pustular stage*. Pyogenic organisms are found in the pustules. Fresh epidermis is formed under the pustule, which dries in, and becomes a scab or crust, on the separation of which scars or pits are left behind, if the cutis has been involved in the pustulation.

The *blood* shows a marked leucocytosis, the lymphocytes being chiefly increased, the polymorphonuclear leucocytes diminished.

**Symptoms.**—After twelve days' incubation, the malaise of onset comes on. Its most notable features are—*shivering, frontal headache, pains in the back, vomiting*. In children there may be *convulsions*. The temperature runs up rapidly, with all the phenomena of "fever." On the third day the fever usually declines, and the rash appears as a papule. Soon, however, the symptoms become worse and more pronounced than before, the fever increases, and on the eighth or ninth day, when the rash becomes pustular, the so-called secondary or suppurative fever is fully developed, and assumes a septic type with oscillatory temperatures. There may be severe rigors and a rapid assumption of the typhoid state. There is great swelling of the face—the eyes may be even closed up—and too frequently the patient dies. When recovery takes place, the temperature falls gradually, by lysis, and the pocks, after remaining pustular for two or three days, begin to dry, forming scabs which fall off finally on the eighteenth to twentieth day, and leave a more or less pitted appearance.

The *Specific Rash* first appears on the third day of fever, on the face, forehead, and scalp, as slightly raised red papules, which give a *shotty* feeling to the skin even before they are visible. It afterwards appears on the back of the wrists, then



on the trunk and arms, and lastly on the legs. The mucous membranes are also affected.

Three days later (sixth day of fever), the "papules" become vesicular. The vesicles are at first clear and transparent, then turbid, depressed in the middle or *umbilicated*. Each vesicle is also *loculated*, that is, divided into compartments by delicate connective-tissue partitions derived from proliferation of the cells of the rete Malpighii.

Usually about five days after the appearance of the eruption (eighth day of fever), the vesicle becomes *purulent*. Round each pustule there is an inflammatory ring, which causes great swelling and disfigurement of the parts affected. This is attended with exacerbation of symptoms due to absorption, *i.e.*, secondary fever. The pustule then begins to dry, and ultimately a black-brown scab forms and drops off, leaving a depressed scar as before mentioned. A characteristic greasy *odour* is present in small-pox from an early stage.

#### Varieties.—

1. *Varioloid*, or modified small-pox, is the form most frequently seen after vaccination. The initial fever is usually slight, and the rash scanty. The papules vesicate on the fifth day, and dry up in a day or two. There is no secondary fever.
2. *Simple* or *discrete*—the pocks being few.
3. *Confluent*—where the pocks run into one another on the face, feet, and hands. The initial fever is severe, the eruption appears early, and the temperature subsides only partially on the third day. The face and hands are much swollen. Noisy delirium is frequent. Death takes place about the ninth day in unvaccinated subjects, and about the eleventh in the vaccinated, as a rule.
4. *Hæmorrhagic*, assuming one of two forms. (1) *Purpura variolosa* or malignant small-pox. The specific eruption is preceded by an erythematous rash which becomes hæmorrhagic or petechial. Hæmorrhages from mucous membranes (*hæmaturia*, *hæmatemesis*, etc.) are frequent. Death sometimes occurs before the specific eruption appears. (2) Hæmorrhages take place *into the pocks*, or from mucous membranes. Both forms are very fatal.



5. *Corymbose*, where the pocks are grouped in grape-like clusters. This is a rare but severe form.

Other varieties have been tabulated, but they only confuse the student and serve no practical purpose.

Of the above forms the hæmorrhagic is the most fatal, and after it the confluent, in which the danger is in proportion to the number of pustules *upon the face*. The typhoid state is apt to appear early in the latter form.

*Initial Rashes*.—The specific rash may be preceded by other *prodromal* rashes (which may appear on the second day)—

1. *Hæmorrhagic or petechial*, occupying the lower half of the abdomen, and extending downwards in a triangular form to an apex in Scarpa's triangle.
2. *Petechio-erythematous*, of wider distribution, extending up the sides of the chest.
3. *Erythematous*, similar to scarlet fever or to measles. In this case, the true eruption when it occurs seems to spare the site of the initial rash.

The erythematous form, which vanishes in a day or two, indicates a slight attack, and the prognosis is gravest in the petechio-erythematous.

#### Complications.—

1. Conjunctivitis, keratitis, corneal ulceration.
2. Laryngitis and laryngeal ulceration.
3. Bronchitis, broncho-pneumonia, pleurisy.
4. Hæmorrhage of *all* kinds.
5. Gangrene, usually of the penis; erysipelas.
6. Orchitis.
7. Purulent arthritis and pyæmia.
8. Albuminuria; rarely nephritis.

#### Sequelæ.—

Otitis media.  
Boils and abscesses.  
Deep pitting.  
Blindness.  
Permanent baldness.

**Treatment.**—Vaccination or re-vaccination carried out within three days of exposure to infection will either prevent or modify the ensuing attack. Later it is useless. All those in contact



with the patient must be vaccinated, and rigid isolation must be insisted upon. There is no specific drug, and treatment must be carried out on general principles.

As small-pox is infective even in the prodromal stage, patients with suspicious symptoms should be at once isolated.

*Special Points are*—1. Cut the hair and beard close.

2. Sponge daily with tepid water, or in confluent cases use the warm bath.

3. Ointments for the prevention of itching or pitting are useless, but benefit has followed the use of a 10 per cent. solution of iodine in glycerine, painted thrice daily over the pustules. Cold moist applications are grateful to the patient. The windows may be covered with red curtains to exclude the ultra-violet rays.

4. The eyelids may be covered with cold compresses, to reduce oedema. The conjunctivæ should be frequently irrigated with sterile water, or a weak antiseptic lotion. Where the cornea is ulcerated, a single application of the yellow oxide of mercury to the ulcer should be followed by the frequent use of a lotion of Ext. Belladonnæ gr. x-3j (2 per cent.), applied warm.

5. Treat throat complications by glycerine of borax, and laryngeal troubles by a carbolic acid spray.

6. In hæmorrhagic cases, stimulate freely, and administer quinine and perchloride of iron.

7. Later, keep the crusts soft by bathing and daily oiling. Tonics containing quinine and iron are useful in convalescence.

## VACCINATION IN MAN

Vaccinia or cow-pox is an eruptive disease occurring in the cow. When its virus is introduced into man by lymph taken from the pustules, a local vesicle is formed. Mild constitutional symptoms appear, and the result is immunity against small-pox. Though it is not yet definitely settled that vaccinia is variola modified by its passage through the cow, there is strong ground for believing that the two are the same disease.

Vaccination in man may be performed either with lymph obtained from a vaccinated person (humanised lymph), or with lymph obtained direct from the calf. The latter is now the usual method, owing to the possibility of transmitting other diseases by humanised lymph. The inoculation is generally performed upon the arm. After inoculation—

A papule forms on the second or third day.

It becomes an umbilicated vesicle on the fifth or sixth day.



It enlarges up to the eighth day, and by the ninth it becomes purulent and an areola forms round it.

It dries up towards the end of the second week, and the scab falls at the end of the third week, leaving a permanent pitted scar.

The neighbouring lymphatic glands become enlarged. After inoculation the arm usually swells locally and more or less fever is occasioned.

British law requires all healthy children, except those of "conscientious objectors," to be vaccinated before the age of six months.

In ordinary circumstances, vaccination should be postponed if the child is feverish, if it has any specific infectious disease, any cutaneous disease, especially eczema, or if it has diarrhoea. Where there has been exposure to small-pox it must be done at all risks, except in the presence of serious acute disease.

The protection afforded diminishes in completeness with the lapse of years. It is therefore desirable that children should be re-vaccinated at the age of seven or eight, and adults in the presence of a small-pox epidemic.

## VARICELLA OR CHICKEN-POX

An eruptive fever, occurring principally amongst children, and characterised by a rash at first papular, but rapidly becoming vesicular, which appears on the first day, and is repeated in successive crops during the next two or three days.

**Etiology.**—Epidemics are very common, and the disease once it starts usually spreads with great rapidity. Sporadic cases, however, are not uncommon. No specific organism has been isolated. The disease is contagious, but not inoculable. One attack confers immunity. The usual incubation period is fourteen days.

*The Rash* consists of small papular rose-spots, of very varying number, which within twenty-four hours become raised vesicles, containing either transparent or turbid fluid. It usually appears first on the neck and chest, but quickly spreads over the entire body. Each crop requires from five to six days to complete itself, and thus the illness lasts rather over a week. Modified spots may appear on the palate, buccal mucous membrane, and tongue.



The rash differs from that of small-pox in—

1. Not being umbilicated or loculated.
2. Having no inflammatory areola around the vesicles.
3. Appearing on the first instead of the third day.
4. Being vesicular almost from the beginning.
5. The vesicles usually beginning to dry up as brownish scabs on the fourth day, leaving no scarring or pitting, unless they have been ruptured by scratching.
6. In small-pox the fever declines when the eruption appears ; not so in chicken-pox.

**Symptoms** are not severe. There is usually slight fever and fretfulness, with a very furred tongue and rarely vomiting. The disease is usually more severe in adults.

**Complications.**—1. *Severe itching*, causing the child to scratch, producing deep scars, or even ulceration.

2. Gangrene around the vesicles in debilitated children (*varicella gangrænosa*). This is not uncommon in the tuberculous, and in congenital syphilitics.

**Treatment.**—A gentle saline purge, careful dieting, and where there is much itching, soothing lotions on lint—morphine and lead.

## SCARLATINA OR SCARLET FEVER

An acute infectious disease, characterised by fever, sore throat, and an erythematous rash, followed during convalescence by free desquamation, and often complicated by otitis, nephritis, or arthritis.

**Etiology.**—Epidemics prevail at all seasons, but are most prominent during the latter half of the year. The disease is commonest in children between two and ten years old, though adults are often attacked. It is usually spread by direct communication, but may be carried by infected clothing or by a third person, the virus remaining active for a long time. It may also be spread by milk, which has been contaminated either by man or by the cow. Cows and swine are said to suffer from a disease which may produce scarlet fever in man, but Savage regards the condition in cows as “a local infection with organisms of human origin,” superadded to a bovine disease which is not dangerous to man.

None of the organisms described by Klein, Class, or Mallory has maintained its claim to be specific. The “inclusion bodies” found in the



polymorphonuclear leucocytes are round, oval, or curved, and of a size varying between that of a coccus and that of a large bacillus, but they are not protozoal, and are not peculiar to scarlet fever. Their absence, however, is practically exclusive of the disease.

The usual *incubation period* is from two to three days.

One attack generally confers immunity, but second and even third attacks may occur.

**Morbid Anatomy.**—Besides the usual indications of fever, there are inflammatory changes in the throat, varying in degree, and accompanied by lymphadenitis, which may go on to supuration or gangrene. Endocarditis and pericarditis are not uncommon. Nephritis is frequent.

The rash disappears at death except in hæmorrhagic cases.

**Symptoms.**—The principal features of the onset are—vomiting, pains in the back and limbs, soreness of throat, high fever, and headache. In infants, convulsions are not uncommon. Soon the throat becomes very sore, the neck feels stiff, and the glands at the angle of the jaw are swollen.

On the first or second day the rash comes out, first on the chest, then rapidly spreads over the face, body, and extremities. Even when the rash is abundant on the face, the skin round the mouth escapes, and this “circum-oral pallor” is very characteristic. The appearance of the rash may be delayed for a day or two.

As the fever progresses, the throat symptoms become more severe; the tonsils approach the middle line, and yellowish points appear. These may coalesce and form a “patch” resembling that of diphtheria. Other severe complications (*see* tabulated list) often arise and bring about a fatal issue; or the temperature may fall gradually with the fading of the rash, and a slow convalescence is established.

*The Tongue* is at first covered with thick white fur, with papillæ projecting—*i.e.* the white “strawberry” tongue; later, the fur peels off, leaving the typical red “strawberry” or “raspberry” appearance.

*The Urine.*—There may be ordinary febrile albuminuria, even when there is no nephritis. The diazo-reaction may be present.

*The Blood.*—There is generally a marked leucocytosis, the polynuclear cells being chiefly affected.

*The Rash* appears on the second day, first as a scarlet blush



but if looked into carefully it is seen to consist of small red spots, surrounded by a diffuse erythema.

The severity of the rash varies ; it may be absent or only present in the flexures of limbs as bright red lines. Diagnosis is then often difficult, but later on desquamation settles the question.

*Desquamation* begins as early as the sixth day ; it may be in the shape of fine and branny scales, or the epidermis may peel off in large flakes, according to the intensity of the eruption. It continues longest on the palms and soles. The process lasts from four to eight weeks, and sometimes longer.

#### Varieties.—

1. Latent or abortive forms, in which the throat symptoms, fever, etc., are so slight as to escape notice. After they have infected others, the cases may be recognised by desquamation, or by the occurrence of nephritis.

2. *Scarlatina Benigna*, or Simple Scarlet Fever. The rash fades in a few days, and the temperature is normal within a week.

3. *Scarlatina Anginosa*, or Septic Scarlet Fever. The throat symptoms are severe and a membranous exudation forms upon the tonsils and palate, leading to necrosis of the tissues of the throat, with intense fœtor. There is extensive sloughing, which may cause perforation of the ascending pharyngeal artery or even the common carotid. The rash lasts a week or more, and the temperature may not be quite normal for several weeks.

4. *Scarlatina Maligna*, or Toxic Scarlet Fever. The symptoms are grave from the beginning, and the rash abundant, but sore throat is not marked. Delirium passes into coma, and death takes place within a week.

5. *The hæmorrhagic form*, in which extensive hæmorrhages occur (epistaxis, hæmaturia, etc.), is now very rare.

#### Complications.—

1. Scarlatinal arthritis, in the forms of—(a) scarlatinal rheumatism, occurring about the end of the first week, and sometimes causing cardiac mischief ; (b) suppuration of one or two large joints ; (c) general pyæmic infection.

2. Endocarditis and pericarditis.

3. Broncho-pneumonia.



4. Ear troubles are exceedingly common. The suppurative process going on in the middle ear may cause—

- (1) Perforation of the drum.
- (2) Permanent deafness, through suppuration extending to the internal ear (not common).
- (3) Facial paralysis.
- (4) Meningitis or abscess of the brain.

5. Extensive suppuration of glands, particularly the cervical glands.

6. *Scarlatinal nephritis* is rather a sequela than a complication. It most often begins about the second or third week of illness, when the skin is freely desquamating. The symptoms are—(1) High-coloured or smoky albuminous urine; (2) Presence of epithelial casts, blood casts, and blood corpuscles; (3) Dropsy of eyelids and ankles, or general dropsy (*anasarca*); (4) In grave cases uræmia may occur, and lead to convulsions, coma, and death.

**Special Points in Scarlet Fever.**—The skin is peculiarly hot and pungent to the touch. The frequency of nephritis (10 to 20 per cent. of the cases) is probably due partly to the cessation of elimination by the skin, owing to the extensive eruption, and partly to a specially irritating effect of the scarlatinal toxins upon the kidneys. It may follow either mild or severe cases, and there is no reason to suppose that it is due to “catching cold.” It seldom leads to chronic affection of the kidney. It tends to affect the glomeruli more than ordinary nephritis, and hence is often called glomerulo-nephritis.

**Treatment.**—The same general principles as those detailed in the opening chapter apply here.

*Special Points are*—1. *Serum treatment.*—Serum from convalescents, injected in doses up to 20 cc., has sometimes proved successful. Good results have also been obtained, particularly in toxic cases, with polyvalent antistreptococcic sera (*i.e.*, sera prepared from several different strains of cocci).

2. The danger of spreading the disease is greatest during the desquamative period. Isolation must therefore be kept up for at least six weeks from the onset, or till all desquamation and discharges (nasal, aural, etc.) have ceased. Such discharges may be cut short, in many instances, by the use of *staphylococcus aureus* vaccines, preferably autogenous.

Milne has recently claimed that inunction with eucalyptus



oil, twice daily for the first ten days, and afterwards once daily, along with frequent swabbing of the throat with carbolic oil, reduces the period of infectivity, and permits of patients being discharged within three or four weeks, although desquamation has not ceased. But there is reason to believe that the number of "return" cases is increased under this system, and it is safer to follow the older rule.

3. A minimum amount of *nitrogenous* food, to avoid irritation of the kidneys. This caution does not affect milk, which may be freely used.

4. Daily toilet, tepid sponging, or tepid baths.

5. Examine the urine daily for signs of nephritis.

6. The condition of the ears must be carefully watched.

*Complications.*—*Arthritis* demands warm and sedative applications. Wrap the joints in cotton wool; alkalies are better than salicylates, as the latter tend to irritate the kidneys.

*Throat.*—Pure ice to suck; antiseptic sprays; glycerine of carbolic acid; warm applications externally.

For the severe types.—Avoid caustic applications; relieve pain by cocaine solutions; internally, tincture of ferric chloride, with free administration of ammonia; quinine, either alone or with ferric chloride. Stimulants may also be required. Nasal feeding often gives much relief.

*Nephritis.*—Milk diet, hydragogue purgatives (avoid mercurials), hot-air baths, hot packs, etc., according to the severity of the dropsy.

## MORBILLI OR MEASLES

An acute infectious disease attended by an eruption and by catarrh of the upper air passages.

**Etiology.**—Contagion is communicated by the nasal secretions and breath; also by fomites, or by a third person. Epidemics occur oftenest in spring and autumn. The disease is extremely infectious, both during incubation, and still more so when the rash is out. It is communicable to monkeys by means of injection of the infected blood, and sometimes also through the nasal and buccal secretions in the eruptive period. No specific organism is known, and the virus is probably ultra-microscopic, as it passes through a Berkefeld filter. The incubation period is from eight to twelve days. One attack does not confer complete immunity, but second attacks are very rare before adult life.



**Morbid Anatomy.**—The first of the pathological changes is a specific catarrhal inflammation of the mucous membranes of the respiratory and intestinal tracts. The papule, according to Unna, is due to œdema of the cutis and hypoderm, not, as in small-pox, to epithelial changes. Cellular exudation is almost completely absent.

The blood shows a leukopenia during the eruptive stage. Death is due to respiratory complications; bronchitis, bronchopneumonia, or collapse of the lung are therefore found.

**Symptoms.**—The onset is usually abrupt, with smart fever, sneezing, running at the nose, watering and redness of the eyes, photophobia, and cough. There is often catarrhal diarrhœa, with green offensive stools. The fever falls on the second or third day, but as a rule not completely. It rises sharply with the appearance of the rash on the fourth day, increases as the rash develops, and disappears as it fades at the end of the week. A fine branny desquamation follows, and lasts about a fortnight.

*Koplik's spots* very often appear on the buccal mucous membrane, within the lips, and even on the gums. They are small rounded red spots with a bluish central area. They precede the true rash by one to three days, and as they are very constant, they are of importance to an early diagnosis.

The *Rash* first appears on the *fourth* day at the roots of the hair, and on the forehead and face. It consists of faintly raised, dark-red papules, coalescing into crescentic patches, and giving a velvety feeling to the touch. There is usually slight subcutaneous œdema, and the child presents a peculiar blotchy, swollen appearance. After extending to the neck, trunk, and limbs, the rash fades in about three days, leaving yellowish stains which soon pass away. The branny desquamation which follows is generally slight, and is best seen on the face. The rash differs from that of scarlet fever by—1. Appearing on the fourth instead of the second day; 2. Being darker in colour; 3. The velvety feeling; 4. The crescentic arrangement; 5. The manner of spreading; but diagnosis from this point is sometimes difficult; 6. The subcutaneous œdema.

*The rash may be slightly hæmorrhagic even in cases which are not severe.*

In simple cases convalescence is usually complete in eighteen days.



**Varieties.**—

1. Simple.
2. Malignant, comprising
  - (a) Hæmorrhagic measles, characterised like all other hæmorrhagic forms of fever by bleeding from mucous surfaces, hæmaturia, and an early assumption of the typhoid state. This is a rare form.
  - (b) Adynamic measles, in which the symptoms are grave from the outset, but without hæmorrhages, and the typhoid state is early present.

**Complications.**—

1. The extension of bronchial catarrh, leading to broncho-pneumonia and collapse of the lung. Lobar pneumonia is much less frequent. In children, laryngitis is often formidable. The initial coryza may become chronic, and lead to adenoids.
2. Ophthalmia and keratitis.
3. Otitis media.
4. Hemiplegia is rare, but important.
5. Enlargement of cervical or thoracic glands.
6. Gangrene of skin or vulva ; cancrum oris.
7. Co-existent diphtheria.
8. Intestinal catarrh.

The sequelæ are very numerous and whooping-cough is frequent among them. Tuberculosis is also liable to follow, and occasionally chronic endocarditis.

**Treatment.**—General principles hold good. Owing to the condition of the respiratory organs, the greatest care must be taken to avoid chill until all catarrhal symptoms have passed away. Simple cases need little more than careful nursing. In convalescence cod-liver oil, Syr. Ferri Iodid., Syr. Hypophosph. and similar drugs are indicated. Cases should be at once isolated, and may be released at the end of three weeks after thorough disinfection, if all catarrhal symptoms have gone.

Milne advises a treatment similar to that which he advocates for scarlet fever (*see pp. 39–40*).

**RÖTHELN—GERMAN MEASLES—RUBELLA**

An infectious eruptive fever, not attended by catarrh, but accompanied by swelling of the cervical glands.



**Etiology.**—Rubella was formerly thought to be a mixture of scarlet fever and measles, but is now held to be a separate disease. It spreads by contagion, and epidemics are most common in the spring. The average incubation period is fourteen days. One attack does not confer immunity.

**Symptoms.**—Those of slight fever and headache, sore throat, and swollen and tender cervical and occipital glands; *rarely are there severe complications*. Many patients do not feel ill at all, but Cheadle reported some severe cases attended with albuminuria.

*The Rash* consists of round or oval, slightly raised, pinkish-red spots, discrete at first, but afterwards coalescing. It appears on the first or second day, begins on the face, and extends to the body and limbs while fading on the face. Fever and rash subside together on the third day.

**Diagnosis.**—From Measles by—

1. Short prodromal stage.
2. Absence of the dark colour and crescentic form of the measles rash.
3. Absence of Koplik's spots.

From Scarlet Fever by—

1. Large size of the spots.
2. Absence of severe symptoms and desquamation.

**Treatment.**—General principles. A child exposed to infection should not be allowed to mix with others for twenty days. A convalescent is free from infection in not less than ten days from the appearance of the rash.

## PERTUSSIS OR WHOOPING-COUGH

A specific infectious disease, affecting the respiratory organs, and attended with a peculiar paroxysmal cough and whoop.

**Etiology.**—The disease is contagious from person to person, and may also be spread by fomites. The virus is chiefly disseminated in the sputum. Children between the first and second dentitions are most frequently attacked, but unprotected adults and infants are both liable, although in the adult the disease is rare.

Whooping-cough is most infectious in the first week, and becomes gradually less so. Epidemics occur chiefly in spring



and early summer, and are often closely associated with measles. One attack procures future immunity.

*Specific organism.*—The bacillus described by Bordet and Gengou has strong claims to be considered as the cause. It is a minute oval rod, about the same size as the influenza bacillus, Gram-negative, and growing freely on a modified form of blood-agar. It is most abundant in the sputum in the first week of the disease.

**Morbid Anatomy.**—The spasmodic nature of the cough is due to implication of the nervous system, probably the result of toxic invasion. *Post mortem*, evidences of bronchial catarrh, and of pulmonary or other complications, may be found. Leucocytosis is well marked (15,000–30,000 per cmm.), the lymphocytes being chiefly involved.

**Symptoms.**—The disease is usually divided into three stages—1. Stage of invasion (catarrhal stage); 2. Paroxysmal or spasmodic stage; 3. Period of decline.

The average incubation period is from eight to ten days.

1. *Stage of Invasion.*—The onset is either insidious or abrupt, the temperature in the latter case rising smartly to 100°–102° F. The symptoms are merely those of bronchial catarrh with coryza, and last from seven to ten days.

2. *Spasmodic or Whooping Stage.*—The paroxysm consists of a series of short coughs or expiratory puffs, with no intervening inspiration till about fifteen or more expulsive efforts have been made in a brief space of time. Then occurs a deep, prolonged inspiration, attended with the characteristic *whoop*; a second bout of short coughs succeeds with another whoop, and after three or four such sequences, a little plug of mucus is expelled; or more frequently vomiting takes place, and the child is apparently well until the next paroxysm.

During the severe cough the patient is perfectly helpless, and when the paroxysms are very violent we may get—1. Hæmorrhages from the nose, frænum of the tongue, under the conjunctivæ, or even in the brain; 2. Ulceration of the frænum; 3. Collapse of the lung; 4. Convulsions in infants; 5. Fatal asphyxia (rare).

During the paroxysm the face becomes blue and swollen, and the eyeballs protrude. If the fits are frequent, there may be cyanosis, with œdema of the face and neck. The whoop is due to a spasmodic partial closure of the glottis. There may be as many as eighty attacks in the twenty-four hours, but they range from four upwards. The prognosis is grave in proportion to the



number of attacks. The paroxysmal stage lasts three to six weeks. During the expiratory spasm, the percussion note is defective. Râles may be heard over the chest throughout the paroxysm. In the intervals there are usually no physical signs, but the rule laid down by Niemeyer is a good one:—"If a child has a violent, *prolonged cough* attended with *vomiting*, suspect and treat as if it were whooping-cough."

3. *Period of Decline*.—This stage is marked by a gradual decrease in the number of paroxysms, the disease lasting from six weeks to two months or more. Convalescence is slow, and may be protracted over several months, but patients may be considered non-infectious five weeks after the occurrence of the first whoop. During convalescence there is an increased liability to tuberculosis.

**Complications**.—Besides those mentioned as possibly occurring during the paroxysm there may be—

1. Bronchitis, bronchopneumonia, and rarely subcutaneous emphysema from rupture of air-cells.
2. Convulsions, and more rarely cerebral paralysis following hæmorrhage.

**Diagnosis** is difficult in the catarrhal stage, but the early lymphocytosis is helpful. The lymphocytes number about 60 per cent., the polymorphonuclears about 40 per cent.

**Treatment**.—Prompt isolation; and to begin with, a simple saline with paregoric. When the whoop develops we may try anti-spasmodics; bromoform, belladonna, chloral, or antipyrin. Of these belladonna is the most generally useful; others advise in addition—quinine, emetics, swabbing the throat with a 2 per cent. solution of resorcin, etc. The number of drugs recommended by various authorities shows that none of them is specific. Even the anti-spasmodics have little influence on the paroxysms unless given in such doses as to be otherwise harmful. Creosote, carbolic acid, sulphurous acid, etc., vaporised in the sick-room, have often proved useful.

Since the discovery of the bacillus, vaccines have been used with promising results.

Adequate ventilation and avoidance of cold and draughts are very important. In warm weather the child should be much in the open air, unless serious complications are present.

If recovery be tedious, *change of air*, cod-liver oil, and Easton's syrup, may be recommended.



*Special Points* to be noticed—1. In *infants*, the tendency to convulsions; 2. In *older children*, the liability to bronchopneumonia, causing—(1) Collapse of the lung; (2) Deformity of the chest (pigeon shape); 3. Children exposed to infection should be disinfected, and isolated for at least three weeks, as the disease cannot be diagnosed in the catarrhal stage.

## MUMPS OR EPIDEMIC PAROTITIS

An acute infectious disease, characterised by inflammation of the salivary glands, especially the parotid gland, with a tendency to metastatic inflammation of the testes in males, or the breasts in females.

**Etiology.**—The specific organism has not yet been discovered. The disease is frequently contagious, especially in schools, and may be curiously localised in one district. It generally attacks children from four to twelve years of age, and young male adults, but no age is exempt. The disease is infectious even before the glands are affected, and for two or three weeks afterwards. The incubation period lasts from two to three weeks. Second attacks are rare.

**Morbid Anatomy.**—The inflammation is interstitial rather than parenchymatous, the connective tissue in and around the glands being attacked, while the acini escape. It rarely goes on to suppuration.

**Symptoms.**—After a period of incubation, pain is felt under one ear, with stiffness or soreness of neck and jaw. This is usually accompanied by smart fever ( $101^{\circ}$  F. to  $103^{\circ}$  or  $104^{\circ}$ ) which subsides on the third or fourth day. The swelling first appears in the hollow between the angle of the jaw and the mastoid process, gradually extends, and may involve the submaxillary and sublingual glands. It is elastic and tender on pressure, but not fluctuant. In about two days the other side may undergo the same changes, and the swellings combine to form a "collar," giving the child a ludicrous appearance. Oftener the swelling is confined to one side, or appears on the other when the first is going down. Deglutition and mastication are often very painful, the breath is foul, and the tongue is very furred. In about nine days the swelling resolves, and rapid improvement takes place. The glands seldom suppurate.

**Complications.**—1. Unilateral orchitis occurs as the glandular affection subsides, or later, in nearly one-third of the cases



seen in adult males. It subsides quickly, but may lead to atrophy of the testicle. Epididymitis is uncommon; 2. In the female, mastitis and œdema of the vulva may occur; 3. Meningitis is a rare complication; 4. Endocarditis has also been recorded.

**Sequelæ.**—After severe cases the following have happened—

1. Permanent deafness, following otitis media or interna; 2. Arthritis, rarely purulent.

**Treatment.**—A simple saline purge, hot fomentations, and an antiseptic wash for the mouth are all that is usually required. Treat complications as they arise.

### GLANDULAR FEVER

An acute infectious disease characterised by fever and by swelling of the cervical and other groups of glands.

**Etiology.**—The disease occurs mainly in children under fourteen, and sometimes in epidemic form. No specific organism is known, and it is still maintained by some authorities that the affection is a non-specific adenitis, due to infection from the buccal mucosa.

**Symptoms.**—The incubation period is said to be from five to seven days. The onset is sudden, stiffness of the neck, dysphagia, and fever being the first symptoms. Within three days the cervical glands of the anterior triangle enlarge and become tender, at first unilaterally, and with but little inflammation of the fauces; those of the other side are next affected, and then the posterior cervical, axillary, and inguinal glands. The liver and spleen are enlarged; the mesenteric glands may be implicated; and there are marked debility and constipation. The glands do not suppurate, and the swelling subsides in about five days, the fever disappearing along with it. Convalescence is often slow.

**Treatment.**—Rest in bed and fever diet, with saline or mercurial laxatives. Iron may be needed to meet the anæmia of convalescence.

### INFLUENZA

An acute specific infectious disease characterised by fever, by symptoms affecting mainly the respiratory, digestive, and nervous systems, and by prolonged prostration following upon defervescence.



**Etiology.**—Epidemics are most frequent in the winter months, when respiratory diseases are prevalent. Adults between the ages of twenty and forty are oftenest attacked, young children and the aged less often. Sedentary occupations predispose to the disease.

The *bacillus influenzae*, discovered by Pfeiffer in 1892, is a very small rod-shaped organism ( $0.2\ \mu$  wide,  $0.5\ \mu$  long), Gram-negative, and occurring singly, or in pairs or clumps. It is non-mobile, and does not bear spores. It can be cultivated on blood-agar, on which it forms very minute colonies.

It is found in the respiratory secretions, and less commonly in the lung, heart, central nervous system, etc., but very rarely in the blood. Contagion is conveyed by the moist secretions of the nasal and bronchial mucosa.

The incubation period is from two to six days. One attack confers no immunity.

**Morbid Anatomy.**—The characteristic changes are—1. Inflammatory swelling of the nose and neighbouring sinuses; 2. Hyperæmia of the trachea and bronchi, the surface of which is covered with tenacious muco-pus; 3. In fatal cases patches of broncho-pneumonia; 4. When the central nervous system is involved, hyperæmia of the meninges of brain and cord.

Leucopenia is present in uncomplicated cases.

**Symptoms.**—The onset is sudden. There are severe frontal headache and backache, pains in the bones, and marked weakness. The temperature rises smartly to  $102^{\circ}$  F. or even  $104^{\circ}$ . Coryza and catarrh of the upper air-passages are present, and the larynx or pharynx is often involved. In mild cases convalescence sets in in a few days, but a feeling of prostration persists for a considerable time. In severer cases, after the first few days, the disease assumes one of three types :—

1. *Respiratory type*, in which bronchitis, broncho-pneumonia, or croupous pneumonia develops.
2. *Gastro-intestinal type*.—Epigastric pain, vomiting, diarrhoea, and anorexia, associated sometimes with jaundice and enlargement of the spleen, are the commonest symptoms.
3. *Nervous type*.—The initial pains are more severe, and after defervescence the heart becomes slow or irregular, and there is sometimes anginoid pain. Great depression and insomnia follow. In grave cases there may be coma or delirium. Meningitis or encephalitis may be found *post mortem*.



**Complications and Sequelæ.—**

1. Any form of nervous disorder may follow or complicate the nervous or the other types of influenza :—hæmorrhage, embolism, epilepsy, insanity: myelitis or degeneration of the cord: neuritis, local or general, and neuralgia.
2. Otitis media is not uncommon.
3. Albuminuria, sometimes leading to chronic nephritis.
4. Pulmonary and cardiac complications.
5. Arthritic pains.
6. Accidental cutaneous rashes. Influenza has no proper rash.
7. Hyperpyrexia.

**Prognosis** in uncomplicated cases is good, except in the elderly, to whom the disease proves very fatal, usually from the supervention of pneumonia. Quiescent chronic diseases are apt to be kindled into activity by an intercurrent attack. Relapses are common.

**Treatment.**—Rest in bed should be insisted on even in the mild cases, and continued for a day or two after the temperature has become normal, in order to avoid the risk of relapses or complications. The sputum and nasal secretion must be disinfected. Great care must be taken during convalescence, especially in the elderly.

The pains of the first stage are best relieved by salicylate of soda, or where headache is severe by phenacetin (acetphenetidin). Quinine is also useful at this stage. Diarrhœa may be checked by warmth to the abdomen and by fluid foods; bismuth and sedatives are of little use. For insomnia, bromides may be given, particularly the bromide of ammonium. In convalescence, tonics, and especially strychnine, are of importance.

Complications must be treated as they arise.

**DENGUE**

An acute infectious disease of warm climates, characterised by severe and shifting pain in the muscles and joints, an early erythematous and a late rubeolar eruption, and catarrhal symptoms.

**Etiology.**—The disease occurs in all the warmer countries of the East, in the West Indies, North and South America, and Australia. It attacks persons of all ages. Sporadic cases are frequent, and epidemics occur at intervals, and spread with



great rapidity and suddenness. It is not transmitted by direct infection, but can be communicated by inoculation of infected blood, and by the bites of mosquitoes which have fed upon the sick. Two species—*Culex fatigans* and *Stegomyia fasciata*—have been shown to transmit it. The organism has not been discovered, and is probably ultramicroscopic.

The disease is rarely fatal, and no characteristic morbid appearances are recorded.

**Symptoms.**—After an *incubation period* of from three to five days, the symptoms set in abruptly with severe pain in some joint, often a finger, extending rapidly to all the joints and bones (hence the name “break-bone fever”), and shifting from one to the other. Smart fever, redness and swelling of the face, injection of the conjunctivæ, sore throat, and a general erythematous rash, are the other initial symptoms. After twenty-four to forty-eight hours the rash and fever subside, and the remission lasts from two to four days. There is then a mild return of fever, and a macular or rubeolar rash appears, first on the palms, and spreads over the arms, face, trunk, and lower limbs. It slowly fades, and is followed by a slight or profuse branny desquamation. The other symptoms also disappear within eight days of the onset, but the patient is left weak and exhausted. The pains, especially in the smaller joints, may persist for a long time, so that the gait of convalescents is stiff and affected (“dandy fever”).

Relapses may occur, and one attack does not confer immunity. Hæmorrhages from mucous surfaces are the chief complication.

Leucopenia is present after the second day, with a relative increase of the lymphocytes at the expense of the polymorphonuclears. Harnett also describes a considerable eosinophilia, which begins about the fourth day, is well marked by the tenth, and persists for some time longer.

**Treatment** is that of the febrile condition. Purgatives should be avoided, as the muscular movements they entail aggravate the already intense pain, for which phenacetin (acetphenetidin) or antipyrin may be given. In convalescence, tonics are indicated.

## CEREBRO-SPINAL FEVER

### (Epidemic Cerebro-spinal Meningitis)

An acute specific fever characterised by inflammation of the cerebro-spinal meninges, and clinically by sudden invasion and extreme nervous shock, attended by painful contractions



of muscles, cutaneous eruptions, and various grave nervous complications.

**Etiology.**—The disease occurs both in limited epidemics and sporadically. It affects for the most part children and young adults. Infection occurs chiefly through the nasal passages. A report by the city bacteriologist of Glasgow upon a recent epidemic states that the meningococcus was found in the naso-pharynx of 20 out of 81 "contacts"; and in the naso-pharynx of patients and convalescents, in one instance as long as the seventy-fifth day after the onset. The infection may thus "be carried by individuals who offer no evidence of its presence, and may remain a long time in the throats of individuals stricken with the disease, or who have recovered from it." Cases are recorded of a similar disease, in horses, during its epidemic prevalence in man. Extreme fatigue appears to increase the liability.

The micro-organism is the *Diplococcus intracellularis meningitidis* of Weichselbaum, which is found mainly within the polymorphonuclear leucocytes of the exudate. It is aërobic, and can be grown on serum-agar or blood-agar. It stains easily with ordinary stains, and is decolorised by Gram's method.

**Morbid Anatomy.**—In rapidly fatal cases there may be nothing more than intense congestion of the meninges. In those of longer duration there is an abundant whitish or yellowish fibrino-purulent deposit at the base of the brain and in the lumbar portion of the cord. The membranes are much thickened; the ventricles are dilated and contain pus or turbid fluid. The veins of Galen may be thrombosed, causing hydrocephalus.

The changes in other internal organs are those of the febrile state.

**Symptoms.**—The invasion is sudden; there are intolerable pains in the back of the head and neck, extending later down the spine, vertigo, vomiting, and noisy delirium, which is succeeded by apathy or even by coma. There is often intense hyperæsthesia. The *head is strongly retracted*, and the limbs and trunk are rigid, causing opisthotonos or orthotonos. The knee-jerks are commonly exaggerated, but may be absent; and Kernig's sign (*see below*) is frequently present. In young children, convulsions may occur. The temperature is sometimes little raised, in other cases it may reach 104° F. or 106°. The pulse varies similarly. The skin may therefore be either cold or warm. Cutaneous eruptions may appear from the first to the third day,



about which time consciousness may return, and there may be a transient improvement in the symptoms. They are soon aggravated, coma succeeds the delirium, the fever rises, and diarrhoea may set in. There may be paralysis of one or more limbs. Exhaustion and emaciation are rapid, and the typhoid state is early established. Death may take place in the first week, or after a protracted illness. If the patient survives, the symptoms subside only slowly, and convalescence is long and perilous.

*Cutaneous eruptions.*—These are usually of one of two forms:—1. *Herpes facialis et cervicalis*, and *herpes zoster*. The distribution is often wide, and the eruption need not be limited to one side; 2. *A purpuric or petechial rash* (hence the American name of “spotted fever”) usually beginning on the legs. It may involve the entire surface.

Erythematous and pemphigoid rashes occasionally occur.

*Leucocytosis* or increase in the number of leucocytes (25,000 to 40,000 per cmm.) is constantly present. The cells are polymorphonuclear.

In the so-called *malignant* form of the disease, death takes place in from a few hours to three or four days from the onset.

#### **Complications.**—

1. Pleurisy, pericarditis, pneumonia.
2. Purulent effusions into joints.
3. Optic neuritis, purulent keratitis, or panophthalmia (usually in the right eye).
4. Otitis media; permanent deafness from labyrinthine disease.

*Special Points* to note in this meningitis are—

The marked rigidity, and tendency to opisthotonos or orthotonos.

The varied skin affections.

The sudden onset and extremely rapid course.

**Diagnosis.**—Apart from the symptoms, there are two important aids to diagnosis—

1. *Kernig's sign.*—If the thigh be flexed at right angles to the abdomen, the leg cannot be fully extended upon the thigh, as it can in health, owing to strong contraction of the flexors.

2. *Lumbar puncture.*—The skin over the lumbar region being anæsthetised, the patient is laid on the left side with the



back bent and the knees drawn up. The third lumbar spine is found, and a small aspirating needle thrust deeply upwards and inwards in the third interspace, till the subarachnoid space is reached. The cerebro-spinal fluid, in meningitis turbid or purulent, can thus be collected and examined for the presence of the diplococcus. With ordinary aseptic precautions, this operation is perfectly safe.

**Treatment.**—The use of antimeningococcic serum is now the chief means of treatment, and has reduced the mortality from 70 or 80 per cent. to about 30 per cent. It is injected by means of lumbar puncture into the spinal canal in doses of 20 to 30 cc. for an adult. The injection is repeated daily, or every second or third day, according to the severity of the case, and is always preceded by the withdrawal, for the relief of pressure, of a somewhat larger quantity of cerebrospinal fluid than it is intended to inject of serum. The earlier the treatment is instituted, the better the prospect of success.

All other means of treatment are subsidiary to the use of the serum, but in its absence repeated lumbar puncture may relieve symptoms by relieving pressure. The intense pain may call for morphia. Leeches to the nape of the neck, and ice to the head are also useful, and an ice-bag may be applied along the spine. High temperature and the other symptoms of fever must be treated on the principles already indicated.

The nasal passages must be disinfected, and nasal discharges burnt.

The period of quarantine for contacts is one week.

## POSTERIOR BASIC MENINGITIS

This is a form of cerebro-spinal fever sporadically or epidemically attacking infants, usually under one year of age.

The *diplococcus* to which it is due is identical, morphologically and culturally, with the diplococcus intracellularis, but its agglutinative and opsonic reactions are different. The disease is commonest in the first four months of the year.

The changes found are, in the early stages (1) an inflammatory exudate in the posterior fossa of the base, around the medulla and cerebellum, and extending forward to the optic chiasma; (2) distension of the ventricles with fluid. Later, the exudate may be absorbed, but much thickening of the meninges remains.

Fever, vomiting, convulsions, and retraction of the head, setting in abruptly, are the early symptoms; emaciation follows.



Blindness is frequent, but not due to optic neuritis. The deep reflexes are exaggerated. Death is generally due to exhaustion, after an illness of a few weeks or months. Hydrocephalus is the most frequent complication, and in the cases which recover (about 10 per cent.) may be permanent. Mental impairment is also a frequent result.

The repeated use of *antimeningococcic* serum, combined with lumbar puncture, is the most valuable measure of treatment. Feeding may have to be done through a nasal tube.

## DIPHTHERIA

An acute specific infectious disease attended with grave throat symptoms and general symptoms, and the formation of a false membrane or fibrinous exudation on mucous and abraded surfaces, and often followed by paralysis in various situations.

**Etiology.**—The disease is endemic in the larger towns, and becomes often epidemic at various seasons. It is highly contagious, and the poison is very concentrated in the *pharyngeal secretion*. The disease is often contracted by doctors and nurses. The spluttering of secretion into the face whilst examining or swabbing out the patient's throat accounts for this great tendency to attack the attendants. The bacillus may be conveyed to the healthy from the discharges of the sick, from the throats of convalescents in whom it persists, and from the throats or clothing of healthy people where it has found an accidental lodging. Schools are a fruitful source of infection. The organism is extremely tenacious of life, and infected articles may remain dangerous for months. Epidemics are often traceable to contaminated milk supply, but there is no evidence for water-borne infection, although exposure to sewage effluvia may act as a predisposing cause.

The disease is most prevalent in the third and fourth quarters of the year. Children between the ages of two and ten are oftenest attacked, but it is quite common in adults. Individual susceptibility varies greatly, the organism being often found in the throats of healthy people, especially when they have been associated with patients. Other diseases, such as scarlet fever and measles, weakening both the fauces and the system, render the liability greater. One attack does not confer immunity. The incubation period varies from two to seven days.

The *specific organism* is known as the Klebs-Loeffler bacillus. It is non-mobile, slightly bent, and knobbed; it stains with ordinary dyes, and is



not decolorised by Gram's method. It grows on all ordinary media, and multiplies readily in milk. It is found in the false membrane associated with other germs, such as streptococci, and staphylococci ("mixed infection"). It does not penetrate the mucosa. Diphtheria is, therefore, primarily a local disease, the constitutional symptoms being produced by the absorption of toxins.

The streptococcus pyogenes is very commonly associated with the Klebs-Loeffler bacillus, and may even occur singly, when it may give rise to "diphtheroid" conditions of the throat.

**Morbid Anatomy.**—The false membrane first appears upon the fauces, and may spread thence to the pharynx and larynx, and sometimes to the posterior nares and middle ear. Less commonly it is limited to the larynx or posterior nares. It may also invade the trachea, bronchi, or œsophagus, rarely other mucous membranes. The conjunctiva may be attacked. The membrane consists of a network of fibrin enclosing round cells and connective-tissue corpuscles, the specific organism, and masses of streptococci. In the fauces and nares it includes the superficial layers of the mucosa, and hence is adherent; in the larynx the necrosis does not extend beyond the epithelium, and the membrane can be stripped off.

Histologically, the changes are as follows:—The poison first induces a necrosis of the cells with which it comes in contact; the superficial epithelium thus first disappears. The deeper cells become similarly affected, and a zone of inflammation forms around the dead cells; the membrane thus is really a mass of dead cells undergoing hyaline degeneration, and mingled with fibrin, and it presents the peculiar laminated appearance considered characteristic.

The neighbouring *lymphatic glands* become much enlarged.

**Heart.**—Fatty or hyaline degeneration of the myocardium is somewhat common.

**Lungs.**—Broncho-pneumonia is often found.

**Kidneys.**—Either the ordinary cloudy swelling or intense and acute nephritis may be present. The latter is not common.

**Nervous System.**—At a later stage, the peripheral nerves supplying the parts that may be paralysed are found in a state of partial degeneration of their medullated fibres (toxic neuritis).

The other visceral changes are similar to those of any other intense or malignant fever.

Very marked leucocytosis is common. It affects chiefly the polymorphonuclear cells.



**Symptoms.**—After an incubation of two to seven days a general malaise sets in; there are *slight* fever, stiffness of the neck, and swelling of the glands at the angles of the jaws. The soft palate is deeply congested, and whitish patches soon appear, first on the fauces; these patches coalesce, become “wash-leather-like” in colour, and the false membrane is fully formed. It may extend *all over* the fauces, or may begin on the tonsils first, and creeping forward surround the uvula, like a finger of a glove. Patches on the soft palate may also occur early, and these are often encircled by an area of intense congestion. The membrane is at first easily stripped, but soon re-forms; then it becomes firmly adherent, and if torn away leaves a bleeding surface. If left alone it may slough off.

The glands in the neighbourhood enlarge, but do not usually suppurate.

Anæmia and weakness set in early, and are rapidly progressive. The pulse is very frequent and feeble.

The temperature varies—it may be 103° F. or higher, but is usually from 100° F. to 102°, and indeed sometimes *subnormal*. This is peculiar, as most acute throat affections have high temperatures.

The exudation may now extend in any direction, upwards or downwards, into—

1. The naso-pharynx, attended with coryza, regurgitation through the nose, epistaxis, and a nasal twang of voice.

2. The larynx; symptoms are stridor, brassy or “croupy” cough, great dyspnœa, and sucking in of the intercostal spaces. The membrane may *first appear in the larynx* (“diphtheritic croup”), and hence the symptoms just mentioned may be present before any characteristic appearances in the fauces. It may remain limited to the larynx, or may extend upwards to the pharynx or downwards to the bronchi.

3. The bronchi, with all the symptoms of severe capillary bronchitis or broncho-pneumonia. The membrane, after extending to the first bifurcation of the bronchus, speedily becomes purulent.

4. The middle ear, along the Eustachian tube.

In many instances the false membrane remains confined to the fauces.

*Albuminuria* is frequently present from the earliest period. There is usually no dropsy. The diazo-reaction is seldom present.



Three forms of diphtheria may be distinguished, according to their severity :—

1. *Benign* form. The general symptoms are very mild, and there are only a few scattered patches of membrane on the tonsils. Patients may be convalescent in a few days. No doctor may be consulted, and hence they are very likely to spread infection.

2. The ordinary form just described. If the membrane does not spread, a gradual convalescence may set in about the fourteenth day; or death may result from asthenia or cardiac failure. Laryngeal diphtheria leads to death from asphyxia.

3. The *malignant* form. Symptoms are severe from the start; there is great prostration, the typhoid state develops early, and there is a marked tendency to hæmorrhages. Death takes place in a few days.

**Complications**, besides those described, are—1. Formation of membrane on external wounds; 2. Severe ulceration of the throat; 3. Cardiac failure, and rarely ulcerative endocarditis; 4. Thrombosis of veins; 5. Pulmonary affections (bronchitis, collapse, broncho-pneumonia); 6. Nephritis; 7. Meningitis; 8. Otitis media; 9. Conjunctival diphtheria (rare); 10. Streptococcus septicæmia (“mixed infection”).

The most important sequela is *post-diphtheritic paralysis*. This is a peripheral neuritis, and may vary much in its distribution and severity—usually coming on within three weeks of apparent recovery. The more constant symptoms are—1. Anæsthesia and paralysis of the soft palate; 2. Loss of accommodation, with squint or diplopia; 3. Loss of the deep reflexes; 4. More or less wasting of the paralysed muscles, with reaction of degeneration.

The paralysis may be much more extensive, and if the intercostal muscles or vagi become affected, then the prognosis is very unfavourable. Usually, however, the paralysis passes off in time under suitable treatment.

*Special Points.*—Note—1. The tendency to cardiac failure (myocardial affections), and to septic infection (streptococcus); 2. The marked depression; 3. The red areolæ around the exudation.

*Remember* the membrane may be very localised, and not visible without a post-nasal or laryngoscopic examination.

**Diagnosis** is easy in well-marked cases. Wherever there is doubt, a bacteriological examination of the exudate, removed on



a sterilised swab, should be at once undertaken. The swab is rubbed over the surface of blood-serum in a culture-tube, and is ready for examination after incubation for twelve hours at 37° C. In doubtful cases antitoxin should be given without waiting for this confirmatory evidence.

### **Treatment.—**

1. Isolation must be continued for at least three weeks after the throat is clear, or till all discharge from the throat, mouth, or ears has ceased.

2. The *antitoxic serum* has greatly reduced the mortality from diphtheria. The treatment should be begun early, and the dose must vary with the period of the disease and the gravity of the case. From 2000 to 8000 units should be at once injected, another dose of 2000 to 4000 given in twelve hours in urgent cases, and a third twenty-four hours afterwards. Much larger doses may be given if the case is seen for the first time in a late stage. The age of the patient does not affect the dose. The membrane usually begins to separate in from twenty-four to thirty-six hours, and daily injections should be given until this effect is produced. Strict antiseptic precautions must be observed.

In some divergence from these principles, Park considers that "amounts of antitoxin beyond 25,000 units in a child, and 50,000 in an adult, are absolutely unnecessary . . . ; and that an initial dose of 10,000 in a child and 20,000 in an adult is probably sufficient for the whole course of the disease." He recommends that all the antitoxin needed be given at the outset, in a single dose ; because absorption from subcutaneous injections is very slow, and what is given on the first day makes the body fluids more and more antitoxic till the third or fourth day. The recent introduction of concentrated antitoxin makes it easy to give such large amounts in a single dose.

Intramuscular injection has of late been advocated as causing more rapid absorption than the subcutaneous method, and less rapid excretion than the intravenous.

As the serum of an immunised animal not only has an important curative influence in cases of diphtheria, but also protects "contacts" against infection, it may also be used for prophylaxis. For this purpose a dose of 300 units may be given to a child, 500 to an adult. "The antitoxin unit is the amount of antitoxin which, injected into a guinea-pig of 250 grammes in weight, neutralises 100 times the minimum fatal dose of toxin of standard weight and strength."



The immunity is transitory, and lasts no more than six weeks.

3. Locally, frequent swabbing or irrigation of the fauces and nasal fossæ with antiseptic solutions, such as carbolic acid (3 per cent.), formalin (1 in 200), or Loeffler's solution (menthol 10 grammes dissolved in toluol to 36 cc., Liq. Ferri Sesquichlor. 4 cc., absolute alcohol, 60 cc.). In the early stages of laryngeal cases, hot sponges to the larynx, and the bronchitis kettle. Intubation or tracheotomy must be resorted to when the larynx is seriously involved.

4. Avoid all lowering measures, and treat symptoms on general principles. Special attention must be paid to the condition of the heart, and the recumbent posture should be maintained for several weeks.

The "post-diphtheritic" paralysis must be treated with careful hygiene, strychnine, massage, the interrupted current if necessary, and nourishing diet.

*Serum disease.*—A single dose of antitoxic serum usually has no harmful effects, but in about 20 per cent. of cases, if the dose has been large, a reaction follows. Eight to twelve days after the injection, the neighbouring lymphatic glands become swollen, there is some degree of fever and malaise, and an urticarial or erythematous rash appears. The symptoms pass off in a few days. In other cases, no symptoms follow the first injection, but if a second is given not less than twelve days and not more than six months afterwards, (1) there may appear within twenty-four hours intense local œdema, fever, and a general eruption, all these symptoms rapidly passing off (*immediate reaction*); or (2) similar symptoms may appear and pass away after an interval of five to seven days (*accelerated reaction*). The phenomena closely resemble those of anaphylaxis (*see p. 17*), and depend on the presence of an antigen, not in the antitoxin, but in the horse-serum itself. They are of greater severity in persons the subjects of asthma, in some of whom even fatal results have followed, and they form a contra-indication to the routine use of the antitoxin in prophylaxis in such instances.

## CROUP

It is now universally recognised that most of the cases which used to be known as "membranous croup" are really of diphtheritic origin, and that the "croupy" symptoms are due to the existence of a primary laryngeal diphtheria. False membrane may also be formed in the larynx in the course of



other diseases of bacterial origin, such as scarlet fever, small-pox, measles, and whooping-cough; or it may arise as the result of injury from scalds, etc. In such cases, the "croup" is merely a complication of the causative disease. The only independent maladies which can be called "croup" are non-contagious. Croupy symptoms may be due to an acute catarrhal laryngitis precisely similar to that of adults, no false membrane being formed. But since it occurs in young children, in whom the larynx is very narrow, and spasmodic conditions are frequent, the spasm and swelling of the mucosa cause the special symptoms of laryngeal stenosis. Croupy symptoms may also occur in laryngismus stridulus, an affection of young children due to spasm of the adductor muscles of the larynx, without laryngeal catarrh or formation of membrane. These forms will be described under Diseases of the Larynx.

## ERYSIPELAS

An acute infective disease, characterised by a spreading inflammation of the skin and general febrile disturbance, and due to the presence of the *Streptococcus erysipelatis*.

**Etiology.**—Erysipelas is widely distributed, but most common in temperate climates. It may be epidemic, but is usually endemic. It is commonest in springtime. Dirt and insanitary conditions favour its development. Important predisposing causes are chronic alcoholism, chronic Bright's disease, recent delivery, and the existence of wounds or abrasions. The virus clings to rooms and furniture, and can be conveyed by a third person, but is not active at any great distance.

*The micro-organism* is the streptococcus of Fehleisen, which is apparently identical with the streptococcus pyogenes, although erysipelas always begets erysipelas. It is Gram-positive, and grows on ordinary media. It is found in the lymphatics at the advancing border of the rash.

The *incubation period* is from two to seven days.

**Morbid Anatomy**, as regards the skin, is that of simple inflammation, in which the related lymphatic glands take part. There may be visceral complications of a septic nature, as infarctions of lung, spleen, or kidney; malignant endocarditis; pericarditis or pleurisy; and rarely meningitis. There is moderate leucocytosis.

**Symptoms.**—In a typical case of erysipelas of the head and face the onset is abrupt, and often attended with rigor or



vomiting. The temperature rises sharply—it may be to 104° F. or even higher. Very soon a sharp red patch appears on the skin, either where there is a wound, or at the junction of skin and mucous membrane (inner canthus, angle of mouth, etc.). This rapidly spreads in all directions, the centre at the same time becoming paler. The edges are raised and hard, the surface red, tense, and painful, pitting on pressure. Superficial blebs appear on the surface, their contents being hæmorrhagic in bad cases. The face is enormously swollen and the eyes are often closed. The cervical glands are swollen and tender. Mucous membranes may also be affected. The mouth may be attacked by extension from the face, and the larynx, pharynx, or nose may be involved. After a few days the inflammation ceases to spread, desquamation takes place, and defervescence occurs by crisis or by lysis.

Constitutional disturbance is usually slight, but there may be delirium at night even in mild cases. In chronic alcoholics, and in the elderly, the prognosis is less favourable. The typhoid state is prone to develop, and death from toxæmia to result.

Occasionally the inflammation, while healing on the face, spreads to the trunk, and thence in similar fashion to the limbs (*Erysipelas migrans*).

**Complications** (besides those already mentioned).—1. Local suppuration, sometimes leading to—2. Septicæmia; 3. Extension to the larynx; danger of death from asphyxia; 4. *Albuminuria* is very common, though nephritis is rare; 5. Pneumonia (rare); 6. Ulcerative endocarditis.

**Treatment.**—In most cases little is needed beyond isolation and careful nursing. The strength should be maintained by appropriate diet, and alcohol given when indicated, but not as a routine practice. Sedatives may be given for the delirium, or if this is violent, morphia or hyoscine. Cold sponging is to be preferred to antipyretics.

Internally, the tincture of ferric chloride in large doses (ʒss-ʒi, or cc. 2·0-4·0 every four hours) is often recommended, but there is really no specific remedy. Antistreptococcic serum is not an unqualified success, but 20 cc. may be given once daily. Streptococcus vaccines, if possible autogenous, have been found useful by many. Locally, applications should be employed such as will protect the parts from air and relieve pain. For this purpose one may paint with flexile collodion, apply a dusting powder, and wrap in cotton wool; or hot carbolic fomentations (1 in 40) may be used. Netter recommends a 1 per cent. collargol



ointment, and ichthyol ointment (20–30 per cent.) is of value in many cases. Numerous methods are advised to prevent the spread of inflammation, among the best being the injection of antiseptic solutions beyond the spreading edge.

## THE SEPTIC DISEASES

Under this heading we mean the clinical phenomena attending the introduction into the blood of living micro-organisms or their products. The organisms mainly concerned are those of suppuration (*staphylococcus* and *streptococcus pyogenes*), but some others are capable of giving rise to septic conditions, among them being the *pneumococcus*, *gonococcus*, *bacillus typhosus*, and *bacillus influenzae*. There are three types of septic disease:—

1. *Sapræmia* or *septic intoxication*—i.e. the introduction of bacterial products (toxins) from a lesion in which the bacteria themselves remain local. The toxins do not multiply in the blood; the symptoms therefore are in proportion to the size of dose introduced.

2. *Septicæmia*—the introduction into the blood of organisms which have the power of living and multiplying therein, but which do not give rise to the formation of metastatic abscesses. The symptoms are produced by the toxins which the organisms elaborate *in the blood*, and therefore bear no relation to the amount of bacteria originally introduced.

3. *Pyæmia* is a condition in which, from a primary septic focus, septic matter is disseminated throughout the circulation, and leads to the formation of septic emboli and the consequent development of metastatic abscesses in the internal organs or skin.

No fixed line can be drawn between the three conditions. *Sapræmia* and *septicæmia*, for instance, are closely similar in their symptoms, the latter, however, being usually of greater gravity. They not infrequently co-exist.

It should be remembered that the local source of infection is often obscure, and sometimes undiscoverable even after death (“cryptogenetic septicæmia”).

The paths of secondary infection are as follows (Muir and Ritchie):—

1. By lymphatics, with extension to lymphatic glands and serous spaces related to the primary lesion. 2. By natural channels, such as the ureters and bile-ducts (infection of



kidneys and liver). 3. By the blood-vessels: (a) by a few organisms entering the blood from a local lesion and settling in a favourable nidus; (b) by septic phlebitis with suppurative softening of the thrombus and resulting septic emboli; (c) by direct extension along a vein (spreading thrombosis).

**Symptoms.**—*Sapraemia* is generally due to absorption of products of putrefaction, such as decomposed placental remains, blood clots, etc., to suppurating or to poisoned wounds, or to internal suppurations, such as empyema. The onset is rapid. There are local pain and swelling in the wound, chilliness or rigor, the general symptoms of fever, and marked prostration. If not relieved by treatment, diarrhoea and emaciation follow, the typhoid state appears, and the patient dies comatose or from exhaustion. If toxic absorption ceases recovery may be rapid.

*Septicæmia* may originate in such conditions as pneumococcus or gonococcus infection, or in more obvious lesions such as open wounds. After wound infection, the disease begins within at most three days, with one or more rigors, and with high fever, which shows daily remissions or intermissions. The pulse is rapid, soft, and compressible, and there is a marked leucocytosis. Prostration is very great. Jaundice and diarrhoea are common. There may or may not be delirium. Death may occur on the second or third day; if later, the typhoid state appears, and leads to death within a week or ten days. Mild cases may run a more chronic course.

*Pyæmia* arises in connection with infected wounds, or diseases due to the pyogenic organisms (gonorrhœa, otitis media, empyema, intestinal ulceration, puerperal conditions, etc.). It is most frequently due to the streptococcus. The situation of the abscesses depends on the course of the circulation from the seat of infection. The symptoms are repeated rigors, high fever, remittent or intermittent, profuse sweating, great exhaustion, marked emaciation. Jaundice, vomiting, and diarrhoea are frequent. There are local signs of abscess, visceral or subcutaneous. Pleurisy, pericarditis, and bronchopneumonia are frequent complications. In acute cases the typhoid state soon makes its appearance, and death takes place in about ten days. In more chronic cases visceral abscesses are not common, but there may be suppuration in the joints or muscles. Some of these recover, others die of exhaustion after a long and irregular fever.



**Treatment.**—In *sapraemia* the treatment is obviously to stop the manufacture of the poison by attacking the organisms in their local seat (evacuation of abscesses, washing out the uterus, scraping or drainage of wounds, etc.); and to maintain the general strength by tonics and hygienic surroundings.

In *septicæmia*, while attending to the local source of infection, we have also to deal with the living organisms in the circulation. The strength must be maintained by feeding, and by tonics and stimulants. Drugs are of little use, with the exception of *quinine*, which in large doses of 5–10 grains (grm. 0·3–0·6) every four hours has some bactericidal effect, and is a useful antipyretic. Depressant antipyretics should not be given.

Vaccines, prepared if possible from the patient's own organism, have in some instances given good results. Where the infection is demonstrably due to the streptococcus, a polyvalent anti-streptococcic serum should be given once daily, in doses of 20–30 cc.

The treatment of *pyæmia* is that of septicæmia, with the addition that accessible abscesses must be opened and surgically cleansed.

In all cases, any discoverable primary focus of infection must be dealt with surgically.

## RHEUMATIC FEVER (ACUTE RHEUMATISM)

An acute febrile disease characterised by polyarthrititis, a tendency to hyperpyrexia, a special tendency to involve the pericardium and endocardium, and marked anæmia.

**Etiology.**—Rheumatic fever is now regarded as an acute specific infection. It shows a distinct tendency to epidemic prevalence at irregular intervals. Its clinical course is strikingly similar to that of pyæmia, and hence some writers look upon it as a mild pyogenic infection. But rheumatic joints do not suppurate, and the specific effects of the salicylates in rheumatism are absent in pyæmia.

The *organism* which has most claim to specificity is the *Micrococcus rheumaticus* of Poynton and Paine. It is a small streptococcus, growing in short chains, and in the tissues often in pairs. It grows on ordinary media, and is Gram-positive, but more easily decolorised than *Streptococcus pyogenes*. It is most easily found in the substance of inflamed synovial membrane, and may be cultivated from the blood or from cardiac vegetations. It often sets up arthritic and cardiac lesions in experimental animals.

Other theories of rheumatism, that it is due to an excess of



lactic acid in the blood, or to nervous influences, are now discredited.

The disease is most common during the spring and in humid climates, and most frequently affects *young* adults. Males are more often attacked than females, although before the age of twenty it is more common in women. The influence of heredity is often definitely noticeable. Exposure to cold, inclement weather, etc., are frequent determining causes. No immunity, but rather an increased liability, is conferred by previous attacks.

The mode of entrance of the organism is probably by the tonsils.

**Morbid Anatomy.**—The synovial membrane undergoes the changes common to inflammation of serous membranes, viz. hyperæmia, swelling, and effusion of fluid. The fluid, which contains albumin, is usually turbid, but seldom or never becomes purulent. The ligamentous structures are swollen, and often the cartilages are slightly eroded. In secondary affections, such as pericarditis or pleurisy, and in the endocarditic vegetations, the diplococcus and sometimes also pus organisms are to be found. There is nothing to differentiate these from other inflammations. The myocardium is very commonly affected. The blood, though it contains an excess of fibrin, clots more slowly than normal; this, however, occurs in many other conditions. The poison causes a rapid destruction of the red corpuscles, resulting in anæmia. There is a moderate leucocytosis.

**Symptoms.**—After a feeling of malaise, more or less soreness, and general stiffness, and very often after an attack of tonsillitis, the rheumatic pain begins in one of the larger joints: usually the knees, wrists, or ankles.

*The arthritis* extends very rapidly to any of the medium-sized joints. At first the affected joints are red, hot, swollen, and intensely painful; later, the redness becomes less marked, and the joint may assume a dead-white appearance. Frequently the inflammation rapidly subsides in one joint, only to appear in another.

*The pulse* is rapid, full, and soft.

*The temperature* is moderately elevated, ranging between 101° F. and 103°. It usually reaches its height in twenty-four hours, is very irregular, and falls with the subsidence of the arthritis. In some cases, however, and at any stage of the disease, it may rise rapidly to a great height (107° F. or more).



Such hyperpyrexia, though not very common, forms an important complication of acute rheumatism.

*Marked sweating* of a peculiar sourish smell is a constant feature, and the various hair follicles and other cutaneous glands may become inflamed and painful in consequence.

As the disease advances *anæmia becomes very pronounced*. This anæmia may be increased by the salicylates, but is mainly due to the rheumatic poison. There is well-marked leucocytosis.

*The tongue* is usually very large, flat, and covered with such an extremely thick fur that it has been named the "blanket" tongue.

*The urine* is scanty, highly coloured, and often loaded with urates. The chlorides are diminished or absent.

*Murmurs*, either hæmic or organic, may often be heard over the heart, which should be examined every day.

*The more common complications are—*

1. Endocarditis, most frequent in youth, and affecting oftenest the mitral valve. It occurs in about half the cases.
2. Pericarditis, less frequent.
3. Myocarditis, which may be slight or profound.
4. Pleurisy.
5. Hyperpyrexia, often attended with delirium or coma.
6. Certain skin eruptions, such as sudamina, miliaria, erythema multiforme, "pelioses" or small red petechial spots around the ankles, and purpura.
7. Pharyngitis and tonsillitis.
8. Meningitis (very rare).

Frequently subcutaneous fibrous *nodules* develop over bony ridges.

Though rarely fatal, and though the severe symptoms usually subside in about fourteen days, no disease is more often attended with relapses and grave results. The valvular and other diseases of the heart, which so constantly follow in its wake, may be regarded rather as symptoms than as complications. It should be noted that in children rheumatic affections of the heart may occur in the absence of arthritis. Pericarditis is especially insidious. Chorea is also a frequent sequela of rheumatism in children.

**Diagnosis.**—The distinction between acute rheumatism and septic polyarthritis is sometimes difficult. In the latter there



is evidence of a primary septic disease (gonorrhœa, puerperal conditions, etc.), and a much greater tendency to pus formation, and to marked oscillations of temperature.

**Treatment.**—The patient must be kept absolutely at rest in bed. He should lie between blankets, not sheets, and should wear a flannel night-gown. The diet should consist of milk, diluted with mineral water; if it does not agree, chicken-broth may be given. In convalescence it may be more ample, but red meat should be sparingly used.

Salicylate of soda, or salicin, is the specific remedy, the latter being the less depressant form. In severe cases, the salicylate should be given in doses of gr. xx (grm. 1·3) every two hours until the pain is relieved, thereafter every four hours till the temperature falls, and then less frequently and in smaller doses, its withdrawal being gradually accomplished. In less severe attacks gr. xx (grm. 1·3) every four hours may suffice. Lees recommends its being vigorously pushed, and combines it with sodium bicarbonate to lessen the risk of poisoning. He gives adults gr. xv (grm. 1·0), with gr. xxx (grm. 2·0) of the bicarbonate, every two hours from 6 a.m. to 10 p.m., and once during the night, to a child between seven and twelve years old gr. x (grm. 0·6) at the same intervals, and to a younger child gr. v (grm. 0·3). The symptoms of salicylate poisoning are tinnitus aurium, deepened breathing ("air-hunger"), delirium, vomiting, and acetonuria. Should the salicylate disagree, other salicyl compounds (oil of wintergreen, aspirin) may be used.

In the view of the majority, the salicylates shorten the course, protect the heart, and lessen the liability to relapse; but some still prefer the alkaline treatment, in which case large doses (3iss, or grm. 6·0) of sodium bicarbonate are given every three hours. The amount is lessened when the urine becomes alkaline.

If the pain in the joints is severe, they should be fomented, and the limb should be put upon a splint. A lotion of carbonate of soda and laudanum, or a liniment of methyl salicylate, may give relief; and morphia is sometimes required at first.

Hyperpyrexia should be treated by the cold bath.

## PLAGUE—PESTIS

A specific infectious disease characterised by great virulence and rapid course, accompanied by buboes or pulmonary inflammation, and due to the presence in the blood and tissues of the *bacillus pestis*.



**Etiology.**—The disease is liable to become epidemic in the presence of insanitary conditions, filth, and overcrowding. It is now very rare in Europe, the last small epidemic occurring in Glasgow in 1900, but it prevails in India and in Eastern China. It is commonest in warm weather. The contagion attaches itself particularly to houses and to fomites, but it is by no means so virulent as that of small-pox or scarlet fever. Previous to an outbreak, the disease usually appears in rats and mice, which die of it in large numbers. It spreads among them, and from them to man, through the fleas with which they are infested. Except in connection with pneumonic plague, in which infection occurs through the respiratory passages, direct infection from man to man is uncommon.

*The micro-organism* is a minute bacillus, thick, and with rounded ends which stain more deeply than the central portion. It grows on ordinary media, stains with basic aniline dyes, and is Gram-negative.

The average *incubation period* is from three to five days.

**Morbid Anatomy.**—The most characteristic appearances are those of bubo, and of widespread hæmorrhages into all the organs and tissues. Degenerative changes are present in the heart, liver, spleen, and kidneys. The buboes consist of swollen and congested lymphatic glands embedded in a hæmorrhagic exudation into the adjoining areolar tissue. Later, the glandular substance breaks down and becomes purulent. In the pulmonary form scattered pneumonic patches are to be found; and the spleen is enlarged, and may show infarctions.

**Symptoms.**—There may or may not be slight premonitory symptoms, lasting one or two days. The invasion is usually sudden, attended with high fever, vomiting, headache, suffusion of the eyes, and sometimes with rigor. There is great lassitude, the face has an anxious or a dazed expression, and the gait is staggering. Hearing is dulled, and the speech is thick and indistinct. The tongue is swollen, furred, and dry. There may be diarrhœa or constipation. Delirium ensues, the typhoid state sets in, and death may result in a few days.

In about 78 per cent. of the cases (*bubonic plague*), buboes appear between the second and fifth days. They are commonest in the groin, less common in the axilla, and least common in the angle of the jaw. They are usually single, large, and very tender. The skin over them is inflamed.

In favourable cases convalescence occurs from the sixth to



the tenth day, but the bubo continues to enlarge, breaks down, and is discharged in the form of pus and sloughs, which may persist for weeks.

In another form of the disease (*pneumonic plague*) there are no buboes. High fever and severe prostration are present, along with cough, and a profuse, watery, blood-stained (but not rusty) sputum. Moist râles at the bases, and scattered pneumonic patches are to be found, but the physical signs are not proportionate to the severity of the symptoms. The sputum contains the bacillus, often almost in pure culture. The mortality is very high.

Exceptionally severe and exceptionally mild forms (*septicæmic plague* and *ambulatory plague*) are also to be met with.

The mortality varies in different epidemics from 50 per cent. to 95 per cent., but appears to be less among Europeans.

In the Glasgow epidemic, of 28 cases recognised during life 28·5 per cent. died. There were eight others, inferentially plague cases, making 36 in all. The total deaths were 16, or 44·5 per cent. All the cases noted as pneumonic, and all those noted as septicæmic or diarrhœal, ended in death. Only one purely bubonic case proved fatal, and even that is noted as being "probably ultimately septicæmic." Many of the bubonic cases were, however, of a very mild type (*pestis ambulans*).

Only the pneumonic and septicæmic forms gave rise to secondary cases; the bubonic did not. In the former cases the bacilli were disseminated in the sputum or alvine discharges; in the latter locked up in the glands.

In the great Manchurian epidemic of 1910, the cases were almost entirely pneumonic, and the mortality was correspondingly high.

*Special Points.*—1. Rapidity of onset and course; 2. Extreme prostration; 3. Presence of characteristic bubo; 4. In the pneumonic form, diagnosis mainly depends upon bacteriological examination.

*Prophylaxis.*—Rigorous isolation must be carried out, and continued for a month after recovery. All excreta must be disinfected, and also clothes and utensils. Attendants and relatives, where the disease is epidemic, should be inoculated with Haffkine's prophylactic vaccine (a culture grown on broth for six weeks, and then brought for one hour to 70° C. Of this 2 to 5 cc. are injected. Smart febrile reaction follows, and lasts about two days). Rats should as far as possible be exterminated, and their bodies burnt.



**Treatment**, apart from serum-therapy, consists mainly in nursing and in supporting the strength. Ice-bags to the head, cold sponging, avoidance of depressant antipyretics, stimulation by alcohol and ammonia, are the main indications. Locally, pain in the buboes may be relieved by ice-bags or morphia. The ordinary practice is to open them when pus has formed; but Nesfield advises immediate incision to allow of the escape of toxins, and reports striking results.

*Serum-therapy*.—Two sera, Yersin's and Lustig's, have been chiefly employed in the treatment of plague. Of these, the former is bactericidal, the latter antitoxic. Yersin's serum gives the better results, and is the more generally used, but neither has been more than partially successful. Much depends upon the serum being given early in the disease, and on the mode of administration. Part at least of the dose (60–150 cc.) should be given intravenously.

## CHOLERA

A specific infectious disease, occurring in epidemic form, and characterised by violent purging and vomiting, pain, cramps in the legs, suppression of urine, and intense collapse.

The mouth of the Ganges is claimed as the home of cholera, and although epidemics have occurred in most parts of the world, they can always be traced back to India.

**Etiology**.—The disease is not directly contagious. Contagion is conveyed by stools, contaminated water, and vegetables or other food-stuffs washed in it. It breaks out principally in summer and autumn, and attacks all ages and both sexes alike. It spreads along trade routes no faster than the rate of human travel.

The *germ* is a spirochæte, known as the comma bacillus of Koch. It is about half the size of, but thicker than, the tubercle bacillus, and is curved, but may be spiral, or shaped like an S. It grows on ordinary media, stains with the usual aniline dyes, and is Gram-negative. It is found in the dejecta and intestines of all patients affected. It does not occur in the internal organs or blood. The constitutional symptoms are therefore regarded as due to the absorption of toxins from the intestine. The organism survives for a considerable time outside the body, and multiplies freely on moist linen. It remains alive, but does not multiply freely, in sewage water. It is quickly killed by drying. It has sometimes been found in the stools of healthy individuals, who may act as "carriers." Flies also may possibly spread the disease.

**Morbid Anatomy**.—The mucous membrane of the small intestine is generally congested, but may be pale. Peyer's



patches and the solitary follicles are swollen and red. There is no ulceration, but the intestinal epithelium may be extensively denuded. The intestines are filled with a turbid watery fluid, in which the organisms are present in large numbers.

The blood is dark and thick, sometimes almost tarry through the drain of water from the system. The other organs show changes similar to those occurring in other virulent fevers. The spleen, however, is usually small. The tissues are dry, and the serous membranes dry and sticky. *Rigor mortis is rapid*, and may cause movements of the limbs. Sometimes there is *post mortem rise of temperature*.

**Symptoms.**—After a short incubative period (from two to five days) a preliminary diarrhoea sets in, with more or less headache, vertigo, and nausea, and lasts from one to three days; then the characteristic diarrhoea begins. Most clinicians divide the disease into three stages—

1. Evacuative.
2. Algid or collapse.
3. Reaction.

The preliminary diarrhoea is sometimes referred to as the *stage of invasion*, in which case four stages are enumerated. The stage of invasion may, however, be entirely absent, and the disease then begins abruptly with the

**Evacuative Stage.**—This is characterised by violent diarrhoea, and by cramp in the legs. The stools are at first faecal, but soon become fluid, greyish, and watery ("rice-water" stools). Their reaction is generally alkaline. The fluid is of low specific gravity (1006–1012), and contains albumin, mucin and sodium chloride. The flocculent sediment consists of epithelial cells and leucocytes, triple phosphates, shreds of mucus, the specific organism in great abundance, and other microbes. Blood is sometimes present. The evacuations are extremely copious and frequent, and often quite painless, but there may be griping pain. Vomiting begins within a few hours, the food being first brought up, and afterwards matter similar to the stools. It may be almost incessant. There are violent cramps in the legs and feet, and later in the abdominal muscles. The external temperature is subnormal, and the pulse rapid and weak. Prostration is great, and thirst extreme. This stage passes in a few hours into the

**Collapse or Algid Stage.**—The collapse becomes extreme; the features are shrunken, livid, or ashy grey; the eyeballs are sunk



in ; the skin is shrivelled and wrinkled, and covered with a cold, clammy sweat. The surface temperature remains subnormal, but in the rectum it rises to 102° F. or more. The voice is husky ; the pulse is small and flickering, and often absent at the wrist. The purging usually ceases, but the vomiting may continue. Restlessness is conspicuous. There may be complete suppression of urine, coma, and death within a few hours ; or the patient may pass into the third, or

*Reaction Stage.*—The temperature gradually rises, and a red glow replaces the ashy appearance. Erythema and urticaria are frequent. The patient gradually recovers, the urinary secretion returning, or the improvement is checked by the onset of—1. The typhoid state ; 2. Inflammatory complications (pneumonia, enteritis, etc.) ; 3. Recurrence of severe diarrhoea ; 4. Uræmia, coma, and death.

The stage of evacuation may last from two to sixteen hours, that of collapse not more than twenty-four. The duration of the reactive stage is less definite.

The mortality from cholera is greatest at the beginning of an epidemic. Very virulent cases may die before diarrhoea has begun (*cholera sicca*). Pregnant women almost always miscarry.

**Complications.**—1. Bronchitis, pneumonia, pulmonary oedema, pleurisy ; 2. Gangrene of the penis or nose ; diphtheritic inflammation of the vulva. 3. Conjunctivitis ; corneal ulceration ; 4. Meningitis ; 5. Arthritis ; 6. Bed-sores.

**Treatment.**—In the stages of invasion and evacuation diarrhoea should be checked, if possible, by lead acetate and opium, mineral acids, or morphia hypodermically. For vomiting, mustard should be applied to the abdomen, and ice given to suck. Cramps are to be relieved by friction and morphia. No food or stimulant should be allowed, and the thirst must be relieved by ice alone. In the collapse stage, warmth must be applied to the limbs, and if there is no purging, enemata of beef-tea and brandy may be given. Intravenous injection of warm saline solution is often at least temporarily successful (common salt, grm. 3·0, carbonate of soda, grm. 1·5, warm sterilised water, cc. 1000) ; but better results are now obtained by Rogers' method of intravenous injections of *hypertonic* saline solution (sodium chloride two drachms, water one pint), begun whenever the specific gravity of the blood is found to have reached 1063, without waiting for collapse to appear. The



injections may be frequently repeated. By this method, combined with the internal administration of permanganate of potassium in solution or pill, a very considerable reduction in mortality has been effected. The permanganate appears to act by converting the highly toxic nitrites formed in the intestine by the organisms into nitrates. In the stage of reaction, food must be given often, and at first very sparingly. Excessive fever should be checked by sponging, and continued suppression of urine by dry cupping and fomentations over the loins.

*Prophylaxis.*—The sick must be promptly isolated, and all excreta and fomites disinfected. When cholera is epidemic, milk and drinking-water should be boiled. Irritating foods must be avoided. All cases of diarrhoea should be checked as rapidly as possible, by opium, chlorodyne, or other astringents.

Preventive inoculation has been introduced by Haffkine, who injects subcutaneously first a weak and then a strong vaccine, prepared from cultures of the cholera vibrio. The results appear to be distinctly favourable in diminishing liability to the disease.

## DYSENTERY

A term employed to designate various forms of intestinal flux, all characterised by the frequent passage of mucoid or hæmorrhagic stools, tormina, and tenesmus; and pathologically by inflammation, and sometimes ulceration, of the large intestine.

There are two distinct forms, bacillary and amœbic dysentery. Besides these, there are many instances in which dysenteric symptoms occur as a "terminal infection" in the course of acute or chronic disease.

### I. BACILLARY DYSENTERY

**Etiology.**—Dysentery occurs sporadically in temperate climates, and occasionally in epidemic form in large public institutions (jails and asylums) where sanitation is defective. Severe epidemics may occur in times of famine, and in armies in the field. It was very prevalent in the war in South Africa. In tropical climates it is endemic and epidemic. Infection is conveyed by the fæces, by soiled clothing, by flies, and by contaminated water or soil. Convalescents may act as "carriers."

*The bacillus*, known as the bacillus of Shiga, exists in several strains; differing in their fermentative effect on different sugars, and in their degree of agglutination by immune serum. In form and staining reactions it is very like the typhoid bacillus, being like it Gram-negative, but it is somewhat thicker, and is non-mobile.



The *incubation period* is usually two to three days, but may extend to eight.

**Morbid Anatomy.**—In *acute* dysentery the principal changes in the intestinal mucous membrane are—

- (1) Intense hyperæmia, the mucosa being swollen, corrugated, and here and there hæmorrhagic.
- (2) Necrosis, and fibrinous exudate, with formation of a pseudo-membrane.
- (3) Ulceration, at first on the summit of the folds, but extending both deeply and laterally.

In *chronic* dysentery the mucosa is thickened and granular, and the whole gut is thickened. There are chronic ulcers with thickened edges, and areas of scar tissue. Consequently there are partial strictures of the bowel, which is dilated above. A typical dysenteric ulcer therefore has the following characters :—

- (1) Site ; on the ridges of the large intestine.
- (2) Edges ; irregular and thickened.
- (3) Base ; may be formed of any of the coats.
- (4) Tendency to contraction on healing.

The so-called “diphtheritic dysentery” is a form in which necrosis is very extensive and the development of pseudo-membrane pronounced. In advanced cases the whole mucosa may form “a black, rotten, friable, charred mass” (Rokitansky). “Secondary” diphtheritic dysenteries arise as a terminal infection, most frequently in the course of *chronic renal disease* ; but they occur in many other diseases, acute and chronic. They also are of the bacillary type.

**Symptoms.**—In *acute* dysentery the onset may be sudden or there may be previous diarrhoea. There are frequent or incessant calls to stool, with pain, griping (tormina), and constant sense of weight in the rectum (tenesmus). The stools are *very small*, and composed of slimy mucus, which soon becomes blood-stained. Passage of a stool gives no relief ; straining continues, and in grave cases from 50 up to even 200 stools may be passed in twenty-four hours. There are slight or moderate fever, great thirst, dirty tongue, dizziness, and dry skin. The patient loses strength. In the graver forms he may die of exhaustion, or may pass before death into the typhoid state, or death may result from pyæmia or perforation. In milder cases the disease subsides rapidly or slowly, or it may lead to chronic dysentery. Where there is ulceration, the stools



contain clumps of grey or foetid sloughs, and are very offensive; where there is gangrene, the sloughs are larger, the fluid portion is blackish, and the odour intensely disgusting. Pure blood may be passed as the result of ulceration; and in the late stages almost pure pus.

In *chronic* dysentery diarrhœa is less frequent. The stools are loose, partly fæcal, much mixed with mucus, and occasionally with blood. They sometimes appear like "frog's spawn" or "boiled sago." Appetite is absent; the tongue is glazed and red; anæmia and emaciation are progressive; and the patient has a shrunk, cachectic appearance. The spleen is not enlarged. The disease leads ultimately to death.

## Complications.—

- (1) Peritonitis, with or without perforation (rare).
- (2) Pleurisy, pericarditis, endocarditis.
- (3) Arthritis, rarely pyæmia.
- (4) Anæmic dropsy.

*Hepatic abscess does not occur in bacillary dysentery*, but may do so when it is complicated with the amoebic form.

**Treatment.**—In *acute* cases the patient must be confined to bed, and the food must be fluid (milk, chicken-tea, barley-water, etc.). Pain and tenesmus may be met by morphia, or small laudanum enemata. The ipecacuan treatment is much favoured in the tropics. The stomach being kept empty for three hours, ℥ xx (1·2 cc.) of laudanum are given, and in about twenty minutes gr. xx-xxx (grm. 1·3-2·0) of ipecacuan, followed by *absolute rest on the back*. If rejected, the dose is repeated, and the same dose is given every eight hours or so for two or three days. Alternatively Sodii Sulph. ʒi (grm. 4·0) may be given every two hours till a feculent motion is produced, and then so as to produce two or three motions daily. When mucus and blood have gone, bismuth and opium are given till the motions are solid.

Calomel, gr.  $\frac{1}{4}$ - $\frac{1}{2}$  (grm. 0·015-0·03), every hour, or minute doses of perchloride may also be given in the acute stage. Irrigation is contra-indicated by the pain and irritability of the bowel.

*Chronic* cases must be treated at first by rest in bed and careful dieting, combined with irrigation. Two to four pints of a warm (100° F.) solution of nitrate of silver (grm. 1·0-3·0 to the litre) are allowed to flow slowly through a long tube into the bowel, the patient lying on his back with the buttocks raised on a pillow. Large doses of bismuth are also useful.



*Specific Therapy.*—Further experience of the use of polyvalent antidyenteric sera has confirmed the beneficial effects first claimed for them by Shiga. By this means of treatment the mortality has been much reduced, and in all severe cases the serum should be administered at the earliest possible moment. Twenty cc. may be given intravenously to an adult, and ten to a child. Vaccines of the Shiga bacillus have also been used with good results, but they do not appear to be so successful as the serum.

## II. AMŒBIC DYSENTERY (Amœbiasis)

**Etiology.**—*Amœba dysentericæ* is the chief cause of *endemic* dysentery in the tropics and the United States. It occurs in two forms, *Entamœba histolytica* and *E. tetragena*. The latter, which forms cysts containing only four nuclei, is now known to be merely a stage of the former. The obligatory parasite *Entamœba coli* is non-pathogenic. The disease occurs sporadically in temperate climates. The organism is swallowed in contaminated water or upon uncooked vegetables. Convalescents may be carriers.

*Entamœba histolytica* is a rounded cell from three to five times the diameter of a red blood corpuscle, possessing a clear outer ectoplasm, and a granular endoplasm. It has a rounded or oval excentric nucleus, and measures from 10 to 15  $\mu$  in diameter. On the warm stage it shows active amœboid movement. In the resting stage it forms a cyst or cysts, and in this state resists drying for a long time.

The organisms are found chiefly in the large intestine, especially in the rectum and flexures, but they also occur in the ileum and stomach, and in the liver. They have the power of penetrating the tissues.

**Morbid Anatomy.**—The lesions are chiefly seated in the large intestine. They present—

- (1) Small gelatinous swellings of the mucosa, with partial ulceration.
- (2) Necrosis and sloughing of the underlying tissues.

The ulcers of amœbic dysentery thus have *undermined edges*. The amœbæ are found in the ulcerating mucosa, but more abundantly in the tissues beyond the ulcerated area (submucous or muscular coat), where they set up œdema and necrosis. Later, along with the ulcers, cicatrices, leading sometimes to partial stricture, may be found.

Hepatic abscesses, usually single, and hepato-pulmonary abscess, are common complications. Amœbæ are sometimes found in the portal capillaries.

**Symptoms.**—The *acute* form closely resembles acute bacillary



dysentery. Death may occur in a few days, or after weeks or months, from hæmorrhage, perforation, sloughing, hepatic abscess, or exhaustion; or the disease may become chronic. The majority of cases recover.

The *chronic* form may arise out of the acute, or be chronic from the first. The symptoms have a general resemblance to those of chronic bacillary dysentery, but there is a more definite tendency to alternating periods of diarrhoea and constipation. Along with the emaciation, this may suggest cancer of the lower bowel. A digital examination will clear up the difficulty.

Both acute and chronic forms can be distinguished from bacillary dysentery by the discovery of the amœba in the stools, and by the failure of the patient's serum to agglutinate Shiga's bacillus.

The chief *complications* have been mentioned above. Peritonitis may follow perforation. Hepatitis is followed by hepatic abscess, which, if untreated, ends by rupture, usually into the lung, an event signalled by "anchovy-sauce" expectoration. It may also rupture into the pleura, peritoneum, intestines, or stomach, and rarely in other situations.

**Treatment.**—Rest in bed and light or liquid diet are required, according to the severity of the case. Symptoms may be relieved as in bacillary dysentery. A great advance in treatment has been made by Rogers' introduction of *emetine*, the alkaloid of ipecacuanha. The drug, in the form of emetine hydrochloride, can be given either by the mouth or hypodermically, the latter being much the more effective method, and causing no sickness. The amœbæ "can be killed in a few days by about gr. i (grm. 0·065) of emetine hydrochloride each day, in two  $\frac{1}{2}$  gr. doses." In severe cases intravenous injection is desirable. Under this treatment both the dysenteric symptoms and also the hepatitis rapidly disappear. In very obstinate cases it may be supplemented by irrigation of the bowels, and Osler strongly advocates warm solution of quinine in strengths increasing from 1 in 5000 to 1 in 1000. One to two litres should be used twice daily. Nitrate of silver may also be used. Hepatic abscess should be treated by aspiration and injection of emetine hydrochloride into the cavity. Drainage is unnecessary, unless there is a microbic complication.

## YELLOW FEVER

An acute specific fever of limited geographical distribution, characterised by jaundice, albuminuria, and tendency to hæmor-



rhages, particularly from the stomach; and pathologically, by fatty degeneration of the liver and acute nephritis.

**Etiology.**—The disease is endemic in the West Indies, on the coasts of Central and South America, and on the West African coast. From these centres it spreads in epidemic form, usually by means of shipping, to other and sometimes remote regions, where, however, it has never taken a firm hold. It is most severe at low levels, and in crowded towns upon the coast-line, and is especially favoured by warmth and moisture. There is no racial immunity, although negroes in the endemic areas suffer less severely and less frequently than whites. One attack confers almost complete immunity, *so long as the subject remains in the infected area* (repeated inoculation?).

After introduction of a case to a new area, others do not appear for from two to three weeks. The *individual* incubation period, after inoculation, is from three to six days.

Yellow fever is not transmitted by direct contagion or by fomites. It may be communicated by inoculation with the blood or serum of a patient in the first three days of the disease, but not later. Heating the blood to 55° C. for ten minutes destroys its infectivity. It is now proved to be spread by the bite of mosquitoes (*stegomyia fasciata*), which have previously bitten patients at the early stage of the malady. From ten to twelve days are required for incubation in the mosquito before its bite transmits infection, and from four to five or more after the bite before symptoms develop in man.

The specific organism has not been discovered. It is apparently ultra-microscopic, for the blood remains infective after passage through a Berkefeld filter. It is easily destroyed by heat; and it is probably of protozoal nature. The *bacillus icteroides* of Sanarelli has been shown to be a secondary infection.

**Morbid Anatomy.**—The skin is jaundiced. The most important changes are in—

1. *Liver.*—Size about the normal, colour pale yellow with hæmorrhagic patches; cells atrophied, necrotic, and showing *marked fatty degeneration*.
2. *Kidneys.*—In a state of acute glomerulo-nephritis, and much engorged; cells full of fat globules.
3. *Stomach.*—Injected and ecchymosed, coated internally with altered blood; contains “black vomit.”

**Symptoms.**—The onset is usually sudden, accompanied by chilliness or rigor, severe headache, and pains in the back or



limbs. The temperature runs up to 101°–104° F. and sometimes higher. The face is bloated and flushed, the conjunctivæ injected and bright red; there may be a slightly jaundiced tint. The tongue is pointed, red at the tip and edges, and furred in the middle. There may be simple vomiting. Albuminuria may be present from the first day. The pulse is full and strong, but *slow relatively to the temperature, and tends day by day to become slower while the temperature keeps high*. It may fall even below the normal. In mild cases convalescence may begin about the third or fourth day; more commonly there is a remission, lasting from a few hours to a couple of days. The disease then goes on to—

The *second stage*, which commences about the fourth or fifth day. Its prominent features are—

1. "Black vomit." The vomit is at first watery, but becomes more and more mixed with altered blood, and like coffee-grounds in appearance.
2. Other hæmorrhages, as epistaxis, hæmorrhage from the bowel, etc. Metrorrhagia is common, and pregnant women always abort.
3. Highly albuminous and scanty urine. Complete suppression is not uncommon, and if it persists for twenty-four hours the case almost always ends fatally.
4. Jaundice, beginning in the conjunctivæ. The tint is oftenest a faint lemon yellow, but it may be deeper, or may even be a dark orange brown.

There are also asthenic symptoms—intense collapse, shrunken features, etc.; or the typhoid state may occur.

The mortality is very variable in different epidemics and races. "It ranges anywhere from 5 to 75 per cent. of those attacked" (Manson).

**Treatment.**—It is well to begin with a smart purge (calomel or castor oil); afterwards laxatives must be used cautiously. Nothing but small quantities of water or iced water need be given in the first stage; large quantities will be vomited. Stimulants are seldom required so early. They may be used later in the presence of collapse or asthenia, and food may then be given by the rectum. The headache may be relieved by an ice-cap, and sponging may relieve the fever. Drugs are of little use, though astringents may be tried for hæmorrhage. Suppression of urine may be treated by cups to the loins, and hot packs or the hot bath.



*Prophylactic.*—In general, the breeding-places of mosquitoes should be destroyed as in malarial regions, and cisterns protected by wire-gauze. Infected houses should be screened by wire-gauze, and the adult mosquitoes in them destroyed by fumigation. During the fever season the nights should be spent away from the town in a healthy suburb, preferably at some height above the coast-line. These measures have been successful in stamping the disease out of the island of Cuba.

### MEDITERRANEAN (MALTA) FEVER

A specific infectious disease due to the *micrococcus melitensis*, and characterised by a prolonged course of irregularly remittent fever, and by enlargement of the spleen.

**Etiology.**—Endemic on the shores of the Mediterranean, the disease is also found in South Africa, India, China, and parts of America. It prevails chiefly in the summer, attacks the sexes equally, and is commonest between the ages of ten and thirty.

*The organism* is a small flagellated micrococcus, growing on agar and bouillon, and feebly in gelatine. It stains with basic aniline dyes, but is Gram-negative. It is found abundantly in the spleen in fatal cases, and it may often be cultivated from the blood, sometimes from the urine, and occasionally from the faeces. In Malta it is transmitted to the human being through the goat, being found in the milk of at least 10 per cent. of the goats in the island, while the serum of at least half of them agglutinates the organism. There is some evidence that it may be transmitted by mosquitoes.

Fatal cases show a marked enlargement and softening of the spleen. The intestine is not ulcerated. There is no leucocytosis, but on the contrary leucopenia.

The limits of the *incubation period* are not definitely determined. In most cases it is about six to ten days.

**Symptoms.**—For the first week or more headache, sleeplessness, anorexia and sometimes vomiting, constipation, and drenching sweats are the prominent symptoms. Crops of sudamina appear, but no specific eruption. The liver and spleen are enlarged. There is slight cough, and the breath-sounds at the bases of the lungs are harsh and creaking. After this period the acute symptoms disappear, but the sweats continue, and the patient becomes increasingly weak. Constipation continues. Fever remains high, but the curve is irregular and prolonged. It often presents waves of high temperature, lasting two to three weeks, and followed by a few days' intermission and another rise, the total duration being about ninety days.



Convalescence is long and slow. The chief complications are *arthritis*, flitting from joint to joint, *orchitis*, and *neuralgia*.

The *undulatory* type of fever just described is the commonest, but the type may be *intermittent* or *continuous*. Mild cases may recover in a fortnight, in others the illness may be prolonged for six months. In some few *malignant* cases, the typhoid state leads to death in a week or ten days; but only about 2 per cent. of the cases die.

The *diagnosis* from enteric fever depends upon the agglutination test, and from malaria upon examination of the blood.

**Treatment** is symptomatic. The cold bath is indicated in hyperpyrexia. *Vaccines* have been used prophylactically with some degree of success, but their use in treatment has so far given dubious results. Segregation of infected animals is also necessary for prophylaxis, and those animals are considered infected whose milk responds positively to the agglutination test.

## MALARIAL FEVER

A specific infective disease caused by the presence in the blood of the *hæmamoeba* or *plasmodium malarie*, and presenting clinically the following varieties:—(1) periodically recurring paroxysms of intermittent fever; (2) *continued* fever with well-marked *remissions*; (3) certain pernicious, rapidly fatal forms; and (4) a chronic cachexia with anæmia and enlarged spleen.

**Etiology.**—The disease has its headquarters in tropical and subtropical regions, but occurs also in less severe forms in more temperate climates, in which the period of fresh infection is the summer and autumn. Warmth, abundant vegetation, stagnant surface water, and hence low-lying and marshy districts, favour its development, while the opposite conditions retard it.

It is spread by the bites of mosquitoes of the genus *Anopheles*, in the body of which the parasite completes the sexual part of its life-history.

The malarial parasite is a protozoon of the order Sporozoa, and belongs to the group of the Hæmamoebidæ. The term *plasmodium* is applied to it only as it exists in human blood. Its life history is as follows:—In its youngest form it is a small amœboid body (*amœbula* or *trophozoite*) occupying the interior of a red blood corpuscle, and consisting of nucleus, nucleolus, and surrounding protoplasm. The amœbulæ rapidly grow and become pigmented by converting the corpuscular hæmoglobin into melanin. They reach maturity in 48 or 72 hours, according to the species. When mature, some of them become *schizonts* (asexual forms), and others *sporonts* or *gametocytes* (male and female sexual forms). The schizonts then undergo segmentation (*schizogony*), and form a "rosette"-shaped body, consisting



of a variable number of nucleated segments (*merozoites* or *enhæmospores*), which rupture the containing corpuscle, and pass with the liberated melanin into the liquor sanguinis. The melanin is absorbed by the phagocytes, and carried to the central organs; while the merozoites attack fresh corpuscles, and repeat the same cycle of development.

The sexual forms, or gametocytes, do not divide in the body, but circulate unchanged. The female gametocyte (*macrogametocyte*) of tertian and quartan fevers is a large, rounded cell measuring about  $16\ \mu$  in diameter. The male cell (*microgametocyte*) is smaller. In the malignant fevers both male and female cells are crescentic. When the gametocytes are drawn into the stomach of the mosquito, they burst the containing corpuscle. The male gametocyte puts forth four or more actively mobile flagella (*microgametes*), which are really spermatozoa. These detach themselves from the parent cell, and attack the female gametocytes (*macrogametes*). One of them succeeds in penetrating and fertilising a macrogamete, which now becomes elongated and of the shape of a spear-head (*zygote* or *oökinete*). The mobile zygote penetrates the stomach wall, fixes itself on the outer surface of the stomach, and develops there, by segmentation of the nucleus, into a capsule filled with cells called *sporoblasts*, which again divide into numerous delicate thread-like *sporozoites* or *exotospores*. This process occupies at least a week. The capsule ruptures, and the sporozoites are carried by the circulation to the salivary gland and thence to the salivary duct. They are then injected by the mosquito into the next puncture that it makes, and set up a fresh malarial infection in a new host, in whom the sporozoites penetrate the red corpuscles and become amœbulæ.

Three forms of the parasite occur in man, all with a similar life-history, but with individual differences:—

1. The *quartan* parasite (*plasmodium malariae*) occupies medium-sized corpuscles. Its amœboid movement is sluggish. It produces 6–12 merozoites, and its asexual life-cycle lasts 72 hours. The gametocytes resemble the schizonts before segmentation has occurred.
2. The *tertian* parasite (*plasmodium vivax*) occupies large pale corpuscles. The movements are active. It produces 15–20 merozoites, and its life-cycle lasts 48 hours. The gametocytes resemble the schizonts before segmentation.
3. The *æstivo-autumnal* or *malignant tertian* parasite (*hæmomenas* or *plasmodium præcox*) occupies medium-sized corpuscles. The movements are at first active. It produces 6–20 merozoites. It is still doubtful whether there is one or more than one species of this parasite, but the evidence is on the whole in favour of one only, with a life-cycle of 48 hours. The gametocytes are crescentic, with pigment granules in the centre. Only gametocytes and young amœbulæ are found in the peripheral blood, segmentation occurring in the internal organs.

Cultures of the malarial parasite to the fourth generation have recently been obtained by Bass and Johns, the medium used being defibrinated blood, to 10 cc. of which 0.1 cc. of a 50 per cent. solution of dextrose is added. The organisms grow anærobically on the surface of the layer of corpuscles, after the blood has settled. Sexual forms have not yet been obtained.

The liberation of each swarm of merozoites coincides with an attack of fever. Thus with simple tertian or simple quartan



infection a paroxysm occurs every second or every third day. But in tertian ague there may be two broods of merozoites coming to maturity on alternate days, so that what appears to be a quotidian ague is really a double tertian; and in quartan ague there may be two broods, with fever on two successive days, and a day of interval, or three, with a paroxysm every day. A similar statement applies to æstivo-autumnal fever, and thus the paroxysm may here also be quotidian.

Not only fever, but also great destruction of red blood corpuscles follow the escape of the merozoites. Both are probably due to the liberation at that moment of a toxin possessed of heat-producing and hæmolytic properties.

The *incubation period*, as determined experimentally, varies from 6 to 20 days.

**Morbid Anatomy.**—During the paroxysms, the principal changes are—

1. Enlargement of the spleen, sometimes very great. In recent cases the organ is soft; in older cases firm ("ague-cake").
2. Enlargement and congestion of the liver.
3. Congestion of the bone-marrow, the brain and meninges, and the kidneys.

In all these organs the vessels are full of plasmodia and of melanin. There is also present a yellow pigment, not confined to the vessels, which consists of altered hæmoglobin. Marked diminution of the number of red cells, and pigmentation of the leucocytes, are conspicuous features. The lymphocytes are also *relatively* increased for some time after the paroxysm, but leucopenia, not leucocytosis, is a feature of malaria.

In *malarial cachexia* the spleen is not necessarily pigmented. The enlargement is mainly due to thickening of the capsule and trabeculæ, and the organ is hard. The liver is frequently enlarged. Anæmia is a conspicuous feature.

**I. Intermittent Type (Ordinary Ague). The Paroxysm.**—After a day or two of premonitory symptoms of more or less general malaise, produced by the multiplication of the parasites before they are numerous enough to determine an attack of fever, the typical attack comes on, and usually consists of three stages—cold, hot, and sweating stages.

*Cold Stage.*—The patient shivers violently, the teeth chatter, the skin is pale and blue, and the papillæ are raised (goose-skin).



The face is anxious, and the breathing hurried. There is often vomiting. The temperature, though much lowered externally, is raised in the rectum. As may be expected with such an extreme contraction of the superficial capillaries, the urine is pale, copious, and of low specific gravity. This stage lasts from a few minutes to an hour or more. Towards the end of the stage the temperature may be  $103^{\circ}$  to  $106^{\circ}$  F.

The *Hot Stage* may be gradual or sudden in its onset. The skin is hot, red, and burning. Vomiting increases; the carotids throb forcibly, headache is intense, and there may be violent delirium. The temperature is often  $106^{\circ}$  F. or more, and the pulse is rapid and full. Epistaxis or diarrhœa may occur. This stage lasts from one to many hours. Enlargement of the spleen can often be demonstrated. The urine is scanty, of high specific gravity, and contains a large quantity of urates and urea. Albuminuria is frequent.

*Sweating Stage.*—The sweating commences at the roots of the hair, but soon becomes general and profuse. The pulse gets softer, the temperature falls gradually to the normal, and the patient is restored to the normal condition, the enlargement of the spleen diminishing or disappearing. During the sweating stage the urine is of high density, and scanty in quantity; *urates* are more abundant than urea. The patient remains anæmic. This stage lasts from two to four hours.

The paroxysm, as has already been noted, may recur daily, or every second or third day (quotidian, tertian, and quartan ague). The simple tertian and quartan paroxysms last in all ten to twelve hours. Paroxysms are said to “anticipate” when they come on a little earlier each succeeding day, to “postpone” when they come on a little later.

**II. Remittent and Irregularly Intermittent Type** (*æstivo-autumnal fever, bilious remittent fever*).—This form, associated with the presence of the *æstivo-autumnal* parasite, is frequent in the tropics, and also occurs in late summer and autumn in temperate climates. The type of fever is irregular. It may either be (1) definitely intermittent, with irregular intervals between the paroxysms; or (2) more or less continuous, with well-marked remissions, but not intermissions. Of the intermittent type there are two forms, the quotidian, with a paroxysm lasting 6 to 12 hours, and the tertian, with a much longer paroxysm (24–40 hours). The special symptoms of either the



remittent or intermittent type are coated tongue, epigastric pain and tenderness, bilious vomiting, anorexia, constipation or diarrhoea, and jaundice. Either type tends to merge either into an ordinary intermittent attack, or into a typhoid type of grave prognosis.

First attacks of malarial fever, especially in the tropics, are usually either remittent or continued, and only gradually assume the intermittent type. In temperate climates they may be intermittent (and most frequently tertian) from the first.

Both the remittent and the regular intermittent type tend at first, after some weeks of fever, to spontaneous improvement. The symptoms slowly disappear, and the patient may think himself well, and go about his business. But after weeks or months, a relapse occurs, to be followed by another and another, any of which may end in death, or may lead to the malarial cachexia. Spontaneous recovery is uncommon.

**III. Pernicious Attacks** are due to the æstivo-autumnal parasite, and are comparatively rare. They occur mainly in tropical countries, among those exposed to hardship, or in whom resistance has been lowered by intemperance or previous attacks of malaria. They are sudden in onset, and of extreme gravity. The following are the chief forms :—

1. Hyperpyrexial, the temperature running up to  $107^{\circ}$ – $110^{\circ}$  F., or even higher.
2. Cerebral, due to plugging of various cerebral centres by plasmodial emboli. Comatose, convulsive, and paralytic forms are described.
3. Algid. There is no febrile reaction after the cold stage, and the case ends in collapse.
4. Choleraic and dysenteric. The choleraic stools of malaria always contain some bile.

**IV. Blackwater Fever or Hæmoglobinuric Fever** occurs especially in tropical Africa and more rarely in certain parts of India. Recurrence may take place after recovery even when the patient has been removed to a temperate climate. It is uncommon among natives, and usually occurs after a prolonged residence in malarial regions and repeated attacks of malaria, which have led to anæmia and debility. Its etiology is obscure, malarial parasites being absent or scanty in the blood during the attack, and in internal organs after death. Manson inclines “to regard the parasite . . . as being in some respects different from that of ordinary malarial affections.” Many



observers, among them Koch, consider the prolonged use of quinine as the cause of the hæmoglobinuria, but there is much evidence against this view. One attack predisposes to another. Essentially the disease is an acute hæmolysis. Red corpuscles are but sparsely found in the urine, but hæmoglobin is present in abundance. There is cloudy swelling of the hepatic and renal cells. Numerous casts are found in the renal tubules. The skin and conjunctivæ are yellow or more darkly brown from jaundice.

The attack may commence as an ordinary malarial paroxysm, but after a few days there is a severe rigor, and the urine becomes very dark or even black. It is at first copious, but later diminished or suppressed. There is yellow discoloration of the skin and sclerotics, also bilious vomiting, and sharp pains in the loins and epigastrium, usually also bilious diarrhœa. The condition may pass off in a few hours, and not recur. More often there are recurrences with each attack of fever, or there may even be no remission at all. Marked anæmia and prostration develop, and death is very frequent.

While in the malarial zone, the slightest chill or overstrain may provoke recurrence. After the attack is arrested, therefore, the first aim of treatment is to remove the patient to a temperate climate, at a season of the year which is not too cold. Even so, recurrences may take place, and prove fatal; but the tendency generally dies out in about six months.

**V. Malarial Cachexia.**—After repeated or prolonged attacks of malarial fever, and also in those who have been long resident in malarial countries, a peculiar cachexia is apt to develop which is characterised by—

1. Anæmia, often intense, the skin being of a sallow earthy colour.
2. Tendency to hæmorrhages—epistaxis, purpura, etc., and retinal hæmorrhages.
3. Irregular attacks of fever.
4. Great and sometimes enormous enlargement of the spleen; to a less extent enlargement of the liver.

The parasites may be altogether absent in malarial cachexia.

**Diagnosis** of typical forms of ague is easy; in atypical forms the response to quinine, and above all *the results of examination of the blood*, will establish the diagnosis. Those intending to practise in malarious regions should make themselves familiar with the various appearances of malarial blood.



**Treatment of Malaria in general.**—The specific for malarial fevers is quinine, which, properly given, destroys the parasites in the blood. It should be given promptly, and in the early stages in doses up to gr. xxx (grm. 2·0) in the twenty-four hours, and continued in doses sufficient to produce moderate ringing in the ears for a week after the parasites have disappeared from the blood (Ross). The dose may then be gradually reduced, but the drug must be continued for at least three months. It may be given in capsule or in acid solution. In grave or pernicious cases it may have to be given hypodermically, to secure a prompt effect, in doses up to gr. x thrice daily. The injection should be made into muscle, not subcutaneously. Salvarsan, given intravenously in doses of 0·5 grm., repeated after ten days, appears to have been effective in a considerable number of cases of malignant malaria; but quinine remains the staple drug. In chronic cases small doses of quinine, if plasmodia are present, are enough. Arsenic, alone or with iron, is of great service, but iron alone has not so good an effect. Arsenic is useless in acute cases.

In *blackwater fever* quinine may be cautiously given if the malarial parasite is found in the blood. Some, who consider quinine the cause, advocate calomel in large doses; others, salicylate of sodium. Fluids should be freely given, to flush the tubules and avert suppression. Collapse may be met by strychnine or ether. Normal saline should be injected subcutaneously, or given by the rectum. On no account must the patient sit up, owing to the risk of cardiac failure, for which cardiac stimulants (*digitalis*, *caffeine*) are indicated.

*Prophylactically*, 5 or 10 grains (grm. 0·3–0·6) of quinine may be taken in the morning. Mosquito curtains, wire-gauze window screens, etc., must be employed; in tropical towns Europeans should live in a separate quarter; and a crusade against the larvæ of *Anopheles* should be undertaken, by drainage, covering the surface of pools with a film of kerosene, and so forth.

## ACTINOMYCOSIS (STREPTOTRICHOSIS)

A chronic infective disease occurring in man and cattle, and in the pig, and due to the presence and multiplication of the "ray fungus" (*Actinomyces* and allied species of *streptothrix*).

**Etiology.**—There is no evidence of direct infection from the flesh of diseased animals. In man and animals the most frequent site of infection is the mouth. The fungus, which is



common on various cereals, may be taken in with the food, or may find lodgment in those who are in the habit of chewing straw, or who inhale the dust of grain in thrashing or chaff-cutting.

*The organism* forms in the tissues colonies, which consist of—

- (1) Long, thin, branching *filaments*, interwoven in the centre of the colony, radially arranged towards the periphery.
- (2) *Spores* or *gonidia*, spherical bodies formed within the filaments.
- (3) *Clubs*. These are pear-shaped enlargements of the free end of the sheath of the filament. They are found, radially arranged, at the periphery of the colony. They are homogeneous and structureless.

One variety of *Actinomyces* has been cultivated on agar and glycerin-agar, and on gelatine; another is almost strictly anærobic. The disease has been successfully inoculated in cattle.

The filaments and spores are Gram-positive, the clubs usually Gram-negative.

**Morbid Anatomy.**—*In cattle* the disease usually begins in the lower jaw, and causes the development of granulation tissue, forming bulky, nodular, tumour-like masses connected with the bone. Necrotic changes lead at the same time to diffuse puriform infiltration, or irregular abscess formation. The tongue is also affected, indurated nodules being produced, which give a gritty, woody feeling; hence the name “woody tongue.” The lesions may be found in (1) the jaws, tongue, or neck, and the submaxillary glands; (2) the larynx, lungs, or alimentary tract.

*In man* the disease also affects the jaw and neck, and sometimes the tongue. It leads to great connective tissue proliferation, with the formation of nodular masses, which may be mistaken for osteosarcoma. Ultimately suppuration takes place, and deep-seated (sometimes retro-pharyngeal) abscesses are the result.

In internal organs suppuration tends to occur early, and sponge-like abscesses are formed, in the pus of which the colonies may be seen as yellowish or semi-translucent granules, about the size of a small pin's head. The organs most liable to attack are the lungs, intestines, liver, and peritoneum, but almost any organ may exceptionally be involved. The skin is sometimes affected.

*Microscopically* the colonies may be seen to be surrounded by granulation tissue, recalling in its arrangement that of the miliary tubercle. Thus we find (1) the colony in the centre; (2) a layer of epithelioid cells; (3) smaller proliferating cells and leucocytes. Giant-cells may or may not be present.



**Mode of Inoculation.**—The parasite may enter the tissues (1) by the mouth (carious teeth, tonsillar crypts); (2) by the intestine; (3) by the respiratory tract; (4) by the skin; (5) rarely by the female genital tract.

**Symptoms.**—These may almost be inferred from the account of the morbid anatomy. Thus we may have bossy tumours of the face or jaw, ending in suppuration; bronchitis, bronchopneumonia, or abscess of the lung, with cough, fever, and wasting; intestinal ulceration, peritonitis, or abscess of the liver; tumour-like nodules in the skin; or, rarely, disease of the uterus or ovaries.

The *diagnosis* is dependent not so much upon the varying symptoms, as upon the recognition in the purulent discharges of the pinhead-like granules, which under the microscope are found to consist of colonies of the fungus.

**Treatment.**—This is at present unsatisfactory, because the condition, in the first place, is not easily diagnosed; and secondly, when it is diagnosed in man, the extensive inflammatory changes, the numberless adhesions set up, etc., give little chance of the complete success of even surgical treatment. But where the disease is accessible, surgical removal is indicated. Even if only partial, this step, followed by free irrigation with antiseptics, is often of great value. Of drugs, potassium iodide gr. xl to ʒi (grm. 3.0–4.0) daily is the most serviceable, and some cases have been cured by its use. Vaccine treatment has in some instances proved of much benefit.

## ANTHRAX

Anthrax—also called charbon, malignant pustule, wool-sorter's disease, splenic fever, splenic apoplexy—is an acute specific infection, due to the *bacillus anthracis*. It especially attacks cattle, horses, sheep, swine, and deer. It occurs in man chiefly as an industrial disease.

**Etiology.**—Anthrax in animals is the result either of direct inoculation, as by fly-bites, or of grazing in infected pastures. In man it attacks those who have to do with the slaughtering of cattle, or the handling of carcasses or hides, as butchers, tanners, wool-sorters, and hair-combers. The poison may also be carried by flies.

The *bacillus* is a slender rod, varying in length from 5 to 10 or 20  $\mu$ , and having square or slightly rounded ends. The rods, which are non-mobile,



are often joined end to end to form long chains. The organism grows on all ordinary media, stains with the basic aniline dyes, and is Gram-positive. It has comparatively little power of resistance to heat or drying, but its spores are highly resistant. Spores require the presence of free oxygen, as in cultures, for their formation. Thus in infected animals they are found in the secretions of mouth, nose, and intestine, and in shed blood, but not in the circulation.

If a diseased carcass be not opened, spores are not formed, and the bacilli die in a short time; but if it be opened, and the blood and tissues exposed to air, spores are formed and the carcass therefore constitutes a lasting source of infection.

**Symptoms.**—As it occurs in cattle, anthrax is a general disease; in man it may be general or local, the latter being the commoner form.

*In cattle*, symptoms of collapse may rapidly be followed by death; or fever and rigors may be attended with bloody discharge from the bowels and nose, death taking place within forty-eight hours. The most conspicuous changes are found in the spleen, which becomes enormously swollen, dark red, and very soft; hence the name “splenic apoplexy.” The other internal organs are congested, and there may be hæmorrhages into them. The lymphatic glands are also affected, especially the cervical, mediastinal, and mesenteric glands, which are much swollen, and surrounded by œdema. The blood is dark and fluid, and is crowded with bacilli.

*In man*, the organism may enter the tissues (1) through a wound or scratch; (2) by the respiratory tract; (3) by the alimentary tract. The local form is subdivided into *malignant pustule* and *anthrax-œdema*, and the general into respiratory and intestinal forms.

**I. LOCAL FORM.**—(a) *Malignant Pustule* is due to inoculation; hence it occurs on exposed parts—on the face, neck, lips, hands, arms. It commences as a small papule, which becomes vesicular, and round which there is inflammatory induration. In about thirty-six hours the summit of the papule forms an eschar, round which there may be a ring of vesicles. There are great induration and œdema; the lymphatics are inflamed, and the glands swollen. The temperature is at first high, but may afterwards be subnormal. Death may occur in three to five days, or slow recovery may follow upon sloughing out of the eschar. The bacillus is found in the superficial and subcutaneous lymphatics of the affected area, and afterwards in the blood, etc.

(b) *Anthrax-œdema*.—In this form eschar and induration



are absent, the constitutional symptoms are very grave, and the swelling is of the nature of an extensive and spreading oedema, which may go on to gangrene. It is much more fatal than malignant pustule.

II. The GENERAL FORM is rare in man. It may affect—

(a) The *Respiratory Tract*.—Wool-sorter's disease. The primary lesion is usually in the lower part of the trachea and larger bronchi, where there are patches of intense swelling of the mucous membrane, with hæmorrhages and ulcerations. There may also be broncho-pneumonic patches in the lungs. There is great swelling of the mediastinal glands, which, from hæmorrhages, look like blood-clots. In these cases there are few bacilli in the blood, but they are found in great numbers in the bronchi and in lymphatic glands. The disease begins with chill or rigor, fever, headache, vomiting, or diarrhœa, and marked prostration. There are predominant pulmonary symptoms corresponding to the local lesions, hurried breathing, and cyanosis. Delirium is common, but the mind may be clear. Death occurs in three or four days. Survival for more than a week usually means recovery.

(b) The *Gastro-intestinal Tract*.—This form gives rise to hæmorrhagic lesions of the intestinal mucous membrane, tending to necrosis in their central parts, and to implication of the mesenteric glands. The spleen is enlarged. The symptoms are those of intense poisoning—severe vomiting and diarrhœa, and possibly blood-stained stools.

**Treatment.**—In the local form, the swelling should be excised, if not too large, or crucial incisions should be made, and the parts cauterised with pure carbolic acid. Carbolic lotion should be frequently injected into the surrounding tissues. The strength should be supported by stimulants and quinine. Sclavo's anti-anthrax serum should also be used.

Until recently, no treatment had materially influenced the fatal course of the general forms of the disease. Quinine in large doses was the chief measure recommended. But with the advent of Sclavo's anti-anthrax serum the outlook has been changed. The dose (30–40 cc.) should be divided and injected into three or four different places in the flank. In grave cases it may be given intravenously. Malignant pustules often heal under this treatment without surgical interference, although it is safer to combine it with excision. Other methods of treatment recently advocated (pyocyanase, salvarsan) are of inferior



value. Prophylactic treatment consists in preventing anthrax in animals, and preventing the spread of infected material. The bodies of infected animals must be burned or buried unopened, under the supervision of the sanitary authorities.

### GLANDERS (FARCY)

An acute infectious disease, occurring in horses, due to the *bacillus mallei*, and characterised by the formation of granulation-tissue nodules in the nostrils (glanders), or under the skin (farcy). It is sometimes communicated to man.

**Etiology.**—The disease arises in man as the result of inoculation of the discharges from infected animals upon some wound or scratch. It therefore occurs chiefly amongst grooms, stable-lads, and others who have to do with horses.

The *bacillus* is a straight or slightly curved rod, about as long as the tubercle bacillus, but thicker. It stains only feebly with ordinary dyes, more strongly when a mordant is added; but it is readily decolorised by alcohol. It grows on ordinary media.

The *incubation period* is from three days to a fortnight.

**Morbid Anatomy.**—*In the horse*, glanders is characterised by the development in the nasal mucous membrane of grey translucent nodules, surrounded by inflammatory swelling. They cause a profuse discharge from the nose, and ultimately break down and ulcerate. They extend into the trachea and bronchi; the cervical lymphatic glands are affected; and similar nodules may be found in internal organs. *In farcy*, due to inoculation through the skin, the superficial lymphatic vessels and glands are thickened, and the glands tend to suppurate. The internal organs are affected as in glanders.

*In man*, in whom the disease is usually due to inoculation through the skin, the type of farcy is the more common, but that of glanders may occur. The lesions are essentially similar to those in the horse.

**Symptoms.**—The disease may be acute or chronic. In the *acute* form, the site of inoculation—usually the hand or arm—becomes inflamed, swollen, and red, and the inflammation spreads along the neighbouring lymphatics. Soon there is constitutional disturbance, with rigors and sharp fever. A local or general eruption, papular, and later pustular, appears; the nose and face may be secondarily infected; abscesses develop in the subcutaneous tissue and muscles; the joints may suppurate; and the



internal organs may become involved. The condition is one of grave pyæmia, and leads to death in the course of two or three weeks or less.

The *chronic* form is characterised by the development of a local granuloma, which breaks down into an irregular ulcer, with thickened edges and a foul discharge. The lymphatics also tend to ulcerate, and the nasal mucous membrane may become affected. The disease may last for years, but may at any time take on an acute form, and end in speedy death.

The **diagnosis** is difficult. In suspected glanders the agglutination reaction is of service. In a case of glanders the serum agglutinates *bacillus mallei* in a dilution of 1 in 50, while normal serum does not do so even in a dilution of 1 in 5. Or a suspension of the discharge may be injected into the peritoneal cavity of a guinea-pig; if the case be glanders, the animal's testicles become inflamed and swollen within two or three days, and the bacillus can be found in the fluid of the tunica vaginalis. Mallein (a solution of the toxins of glanders) is useful for diagnosis and treatment in animals, but not in man.

**Treatment** consists in cutting out or cauterising the wound, if seen early, treating the lesions by surgical methods, and keeping up the strength. Many of the chronic cases ultimately recover.

### FOOT-AND-MOUTH DISEASE (APHTHA EPIZOOTICA)

An acute specific disease occurring in cattle and sheep, and occasionally transmitted to man.

**Etiology.**—The virus is not yet known, but is a filter-passer and probably belongs to the *chlamydozoa*. The disease occurs in man either through direct contact with the lesions of animals or through the consumption of infected milk.

**Symptoms.**—In animals vesicles and bullæ form on the buccal mucosa and on the tongue, on the borders of the hoofs, and in cows on the udder. There is free salivation. The vesicles break, leaving in the mouth shallow erosions covered by aphthous patches, and causing on the hoofs a purulent discharge, which dries into crusts. The duration is about a fortnight, and recovery is the rule, except in calves.

In man, after an incubation period varying from three to ten days or more, the illness sets in with slight fever, the develop-



ment of aphthæ (vesicles breaking down and leaving shallow oval ulcers with a greyish base, and surrounded by a red areola) on the tongue and inside the mouth, salivation, and sometimes a rash upon the fingers and toes. These parts become swollen, and covered with a dull red elevated eruption, upon which vesicles develop, become pustular, and may be confluent. In slight cases the illness may appear as a mild feverish attack, with no complaint of stomatitis, which must be looked for. The duration is about a week or ten days.

**Treatment** is simple; astringent lotions to the hands and feet, and a mouth-wash of borax or chlorate of potassium. The buccal ulcers may be touched with silver nitrate.

### HYDROPHOBIA—RABIES

An acute specific disease due to the inoculation of a specific virus generated in rabid animals.

**Etiology.**—The disease is almost invariably contracted from the bite of a rabid animal, usually the dog; but the cat, wolf, and fox may also transmit it. The virus is in the saliva, which may be infective for a day or two before symptoms have developed. By no means all who are bitten are affected; wounds on uncovered parts are far more dangerous than wounds through the clothing. Bites on the face lead to specially acute symptoms.

*Specific Organism.*—Negri has described certain rounded eosinophil bodies, varying in diameter from 0.5 to 25 $\mu$  and occupying the interior of the nerve-cells of the central nervous system. They are found in 98 per cent. of cases of street rabies, and are apparently peculiar to the disease. By many, Negri's bodies are looked upon as protozoal, by others, as a cellular reaction against the virus. They are not present in the infected saliva. Still more recently, Noguchi claims to have cultivated the virus, and to have produced rabies in animals by inoculation of his cultures. The organisms he describes are very minute granular nucleated corpuscular bodies of an extremely pleomorphic character, and protozoal in nature.

Since the muzzling order of Mr. Walter Long, the disease has become extinct in Great Britain and Ireland.

The average incubation period is from six weeks to two months. It may be as short as a fortnight, or as long as three months.

**Morbid Anatomy.**—The main feature of rabies is hyperæmia and congestion of the central nervous system. There is also congestion of the pharynx, œsophagus, and stomach.



**Symptoms.**—The wound by which the poison was introduced, as a rule, rapidly heals, and for a time nothing happens to attract the patient's attention to the scar. In about six to eight weeks or so, the scar may become painful and nervous disturbances manifest themselves. The patient becomes sleepless, peevish, irritable, and experiences a choking sensation about the throat. When the disease is fully developed there are intense muscular spasms, the respiratory muscles and those of deglutition being specially involved; but a more or less tetanoid condition may be observed in nearly all the muscles. There may be opisthotonos. The features may be horribly contorted or wear an aspect of extreme terror; the saliva is not swallowed, and as it collects in the mouth along with thick mucus from the congested fauces, it causes noisy attempts at ejection, attended with great difficulty.

The face is usually flushed or livid during the attacks, and there may be raving delirium, delusions, and hallucinations. It should be noted that, though the patient is very thirsty, he is afraid to drink, as any attempt at swallowing brings on the spasms at once; even the sound of running water will excite the attacks. There is generally fever, the temperature ranging from 100° to 103° F. After from two to three days the patient may pass into the "paralytic stage," which, however, is more common in animals. He generally dies of exhaustion in from two to ten days after the development of the characteristic symptoms.

**Diagnosis.**—Until recently, when there was doubt whether a dog was rabid, it was advisable not to kill it, but to keep it isolated and under observation. This has been altered by the discovery of the Negri bodies. The animal should be killed, and its brain should be examined for their presence, which is diagnostic. At the same time a rabbit should be inoculated intracerebrally from the dog's medulla.

**Treatment.**—The bitten person or a bystander should at once suck the wound, and if practicable ligature the part above the injury. The wound should be cleansed and cauterised as soon as possible. The Pasteur treatment should be commenced whenever it is ascertained that the dog was rabid. It is practically certain to prevent the disease if begun within a week of the bite.

When the disease is developed, treatment is merely palliative. Morphia and chloroform for the spasm, and cocaine to diminish the sensitiveness of the throat, are the best remedies.



*Pasteur's Method.*—The virus is intensified by passage through a series of rabbits until a maximum degree of virulence is reached (*virus fixe*). The spinal cords of such rabbits gradually lose their toxic property by drying, and after fourteen days are no longer toxic. The virulence is in inverse proportion to the length of exposure. Pasteur's treatment consists in the injection of an emulsion of spinal cord thus prepared, beginning with a cord which has been exposed for fourteen days, next day using one which has been less attenuated, and so on until a cord dried for only three days is used. In ordinary cases it takes nine days to reach this strength, and the treatment continues till the fifteenth day. Where the incubation is likely to be short, as in bites about the head and face, an "intensive" treatment is employed. The details vary somewhat in different institutions, but the result is the same; the patient is rendered immune during the incubation period, and rabies does not develop. The treatment bears the same relation to hydrophobia as vaccination does to small-pox in those who have been exposed to infection.

An antirabic serum has also been used prophylactically, but does not replace the Pasteur method.

## TETANUS OR LOCK-JAW

A specific infective disease due to the *bacillus tetani*, and characterised clinically by severe tonic spasms of the muscles, especially those of the jaw.

**Etiology.**—Tetanus may occur "idiopathically" or may follow injury. It is most frequent after contused or punctured wounds of the hands or feet, but may result from the most trivial scratch. It has frequently occurred in epidemic form amongst new-born children (*tetanus* or *trismus neonatorum*), the infection entering by the umbilicus. It may follow confinement, especially in hot countries. It has often been prevalent in warfare, and has followed such small operations as tooth-extraction, hypodermic injection, and the like. The term "idiopathic" means that the seat of entry has not been discovered.

The *bacillus tetani*, discovered by Nicolaier, who cultivated it from surface soil, is a slender rod which may grow into long threads. It is slightly mobile, and anærobic in habit. It stains with ordinary dyes, and is Gram-positive. A rounded spore is often situated at one end ("drum-stick bacillus"). It normally infests the intestinal tract of horses and cattle, and therefore abounds in manure and in garden soil.

The organism multiplies only in the wound, the blood and



tissues remaining free. The process is therefore primarily local, the systemic effects being due to the absorption of the poison manufactured in the wound. This is a toxalbumin of extraordinary virulence, which travels to the central nervous system along the axons of the motor nerves.

**Morbid Anatomy.**—Hyperæmia of the central nervous system, and small hæmorrhages into the motor centres are the only important changes.

**Symptoms.**—Usually within ten days of the injury, the patient complains of stiffness in the neck and muscles of the jaw. Gradually lock-jaw (*trismus*) is produced by the tetanic spasm of the muscles; the eyebrows at the same time become elevated, and the angles of the mouth drawn out, causing the so-called *risus sardonicus*. Soon all the muscles may be affected with paroxysms of tetanic spasms. The body may be arched, the patient resting on his head and heels (*opisthotonos*); *pleurothotonos* or *emprothotonos* may sometimes occur. The pain amounts to agony during the paroxysms. Complete relaxation does not occur even in the intervals. The patient may be bathed in sweat, and the temperature reaches sometimes as high as 110° or 112° F. before death. Acute cases usually die within four days; the more chronic and milder cases may recover. The longer the incubation period, the greater is the chance of recovery.

**Diagnosis.**—A diagnosis must be made from poisoning by strychnine. It is difficult to confound tetanus with strychnine poisoning, for the latter never causes the jaws to *remain* tetanically closed between the spasms, nor are the muscles of the jaws involved early. In hydrophobia too there is no continuous rigidity; in tetany the paroxysms attack the hands and feet.

**Treatment.**—The room must be darkened, and all sources of irritation must be excluded. During the severe paroxysms, give chloroform; between, hypodermic injections of morphia or hyoscine may be administered. Chloral should be freely given. The patient may be fed, per rectum, with pancreatised meat extract, etc. The tetanus antitoxin has not proved of much use in acute cases. It may be given hypodermically in repeated doses of 100 cc., but is more effective if given by the intravenous method. Of late, subdural and intracerebral injections have been tried, but the toxin very easily forms so firm a combination with the cells, that the antitoxin cannot withdraw it. The best



results, however, are obtained by intrathecal injection through a lumbar puncture needle. Behring holds that injections given more than thirty hours after onset are useless. Baccelli has had much success with hypodermic injections of carbolic acid. The dose is 2 to 3 cc. of a 5 per cent. aqueous solution, repeated hourly for several days. Carbolic poisoning does not follow in tetanus cases. Magnesium sulphate, administered intrathecally, has also given good results. A safe dose is 5 cc. of a 15 per cent. solution; stronger solutions may produce respiratory paralysis.

If tetanus is feared, repeated hypodermic doses of 20 cc. of the serum may be given. The wound should be incised, swabbed out with 3 per cent. iodine solution, and treated antiseptically. The serum may also be locally applied.

### GONORRHOÆAL INFECTION

In the course of a case of gonorrhœa, sometimes in the acute stage, sometimes when it has become chronic, or even after the discharge has ceased, general symptoms may develop which indicate the spreading of the infective process (*a*) by continuity of tissue, (*b*) by general systemic infection. In class (*a*) are included various forms of disease of the genito-urinary organs, such as salpingitis, endometritis, cystitis; and sometimes, by extension along the ureters, pyelitis, or pyelo-nephritis, which, when due to gonorrhœa, is of very grave significance.

Into class (*b*) there fall forms of disease very similar to those which are found in septic conditions (see *Septic Diseases*, p. 62). Occasionally there may be severe and rapidly fatal general infection, as in a case recorded by Osler, which ended fatally fourteen days after the onset of gonorrhœa. Such cases are associated with foci of suppuration in the urinary tract. More frequently local lesions are to be found—

1. In the heart; ulcerative endocarditis or pericarditis :  
but oftenest—
2. In the joints; gonorrhœal arthritis.

In not a few of these cases the gonococcus has been found in the blood.

*Gonorrhœal Arthritis.*—This affection is much more frequent in the male than the female. The amount of effusion into the joints is not great, but there is considerable peri-articular œdema. The effusion is seldom purulent, but is more like that of synovitis. There is often much tenderness. Recovery is slow, and there is a liability to the formation of fibrous



adhesions, which may lead to ankylosis. Usually several joints are attacked, but sometimes only one. Those which commonly escape in acute rheumatism (sterno-clavicular, intervertebral, etc.) often suffer in "gonorrhœal rheumatism." The gonococcus may be present in the joints affected, most commonly in the synovial fringes. It can sometimes be cultivated from the fluid withdrawn by puncture. The following forms of gonorrhœal arthritis may be distinguished :—

1. *Polyarthritic*, resembling subacute rheumatism.
2. *Acute arthritis*; sudden involvement of one joint, with severe pain and great swelling.
3. *Chronic hydrarthrosis*; one joint only attacked, oftenest the knee; no pain or redness.
4. *Bursal and synovial form*; the bursæ and tendon sheaths are mainly affected.
5. *Septicæmic*; intense symptoms of sepsis along with the arthritis.

**Diagnosis.**—The numerous other causes of synovitis must be excluded. The history of the case, the presence of a urethral discharge, and the obstinacy of the symptoms, will be of service. A persistently abnormal opsonic index to gonococcus is suggestive, and successful culture of the organism conclusive.

**Treatment.**—The use of a gonococcus vaccine has given very encouraging results. If possible, it should be "autogenous" (*i.e.* made from cultures of the patient's own organism). The dosage should be small; from four to ten million cocci every four to eight days are usually sufficient, and higher doses are not altogether safe. With stock vaccines a larger dose is necessary. The case must be taken early: no vaccine will break down old adhesions. Of drugs there is none specific; the failure of the salicylates may even help to distinguish the case from ordinary rheumatism. Iodide of potassium is much vaunted but is often useless. General tonic treatment must be combined with local measures. The urethral discharge must be treated; the joints, if acutely affected, must be kept at rest, while in chronic cases massage is useful. In some cases the thermo-cautery, in others, local baths of superheated air may do good. Bier's method of producing passive hyperæmia may be useful. Later, massage and passive movement are required.

## SYPHILIS

A specific infective disease of slow evolution and long duration, due to the *spirochæte pallida*, and transmitted by (1)



inoculation, whether in sexual intercourse or otherwise (acquired syphilis); or (2) inheritance (congenital syphilis).

### **Etiology.**—

The *spirochæte pallida*, discovered by Schaudinn and Hofmann in 1905, and now also known as the *spironema* or *treponema pallidum*, is a delicate, mobile, spirally-shaped organism, measuring from 4 to 14  $\mu$  in length, and in thickness only 0.25  $\mu$ . The number of turns in the spiral varies from six to twenty-four. The organism is only slightly refractile, and stains with some difficulty. It possesses a delicate flagellum at each end, but is devoid of undulating membrane. In these respects, in its smaller size, and in the greater regularity of its spiral curves, it differs from other spirochætes, notably *spirochæte refringens*, which is found on the surface of lesions of the genital organs. *Spirochæte pallida* is found in all the primary and secondary lesions, and sometimes in the blood during life in secondary syphilis. In tertiary syphilis it is sometimes discoverable in the gummata, but with greater difficulty. In congenital syphilis it is very abundant both in the superficial lesions and the viscera, and also in the blood. Noguchi has of late succeeded in cultivating the parasite on a medium consisting of agar and ascitic fluid to which a piece of sterile rabbit's kidney is added. It is a strict anaerobe, and multiplies by longitudinal fission. Inoculation of his cultures produces the disease in monkeys. He has also shown that the organism exists in different strains. E. H. Ross and also McDonagh have found rounded intracellular bodies in the lymphocytes, and McDonagh claims to have demonstrated that the spirochætes are the male elements (microgametes) and the intracellular bodies the female elements in the development of the parasite, for which he suggests the name *leucocytozoon syphilis*.

**Modes of Infection.**—Infection may be direct (sexual congress, finger of the accoucheur, tattooing, etc.) or mediate, through contaminated instruments or drinking-vessels. The secretions of the chancre and of secondary lesions, and the blood in the secondary stage, all contain the virus. The statement that tertiary lesions are not infective must be modified in view of the discovery of the parasite in them.

The disease may also be transmitted hereditarily, either through the father (sperm inheritance) or the mother (germ inheritance), or both. If the mother is infected after conception, the child may or may not suffer (placental transmission), usually escaping when the infection occurs late in the pregnancy. Though the mother of a syphilitic child may show no signs of the disease, she has acquired immunity from it and cannot be infected (Colles' law). The lesions of congenital syphilis are infective.

**Morbid Anatomy.**—The primary chancre is largely an accumulation of round cells in the corium, with infiltration of the connective tissue. The endothelium of the blood-vessels is swollen, and proliferates, their walls are infiltrated with round



cells, and their lumen is narrowed. The adjacent lymphatic glands are also infiltrated with round cells.

In the secondary stage there are cutaneous lesions with changes of a similar type. The vessels are also implicated. There may be macules, papules, pustules, etc. "Mucous patches" are papules modified by their situation on moist skin or mucous membrane. Where growth is exuberant, especially around the anus, and on the scrotum or vulva, they are called condylomata.

The tertiary stage is characterised by the presence of *gummata*,—hard, nodular, localised masses *adherent to the surrounding tissues*. They may be quite small, or even larger than a hen's egg. They consist of round cells with epithelioid cells and occasional giant cells, and are surrounded by firm connective tissue. They tend to necrosis in the centre, and may discharge through the skin or mucous membrane. When they affect the viscera they may cause much cicatricial deformity.

Profound arterial changes may occur in the later stages of syphilis, favouring the development of aneurysm, atheroma, cerebral thrombosis, etc.

**Acquired Syphilis: Symptoms.**—There are four stages in acquired syphilis:—(1) Incubation; (2) Primary Sore; (3) Secondary Stage; and (4) Tertiary Stage. Of these the following is a very brief summary.

1. The *Incubation Period* varies from two to six weeks, but is usually between three and five.

2. The *Primary Sore*, or chancre, is a papule with an *indurated* base. The surface may or may not ulcerate, but is usually depressed in the centre. The hardness or induration extends to the nearest lymphatic glands, and ultimately all the lymphatic glands may become involved. The glands are freely movable under the skin ("rolling glands"), discrete, and not painful. They rarely suppurate. The chancre or "*hard sore*" finally disappears, leaving a cicatrix behind.

3. *Secondary Symptoms.*—After the appearance of the primary sore, there is an interval ("second incubation") varying from one to three months, but usually lasting about six weeks, before secondary symptoms appear. There is a transient *first stage*, characterised by—

- (1) Fever, usually slight, sometimes sharp and remittent; malaise.



(2) A transitory macular or roseolar rash, chiefly on the abdomen.

(3) Sore throat (congestion of the fauces).

Later, other forms of cutaneous eruption (syphilides) may succeed the roseola, and are distinguished by—

(1) Their *symmetrical distribution* on opposite sides of the body.

(2) Their *colour*; coppery, or like that of raw ham; darker on the legs than elsewhere.

(3) Their *outline*; circular, or where the patches have coalesced, in segments of circles.

(4) Their *polymorphism*; *i.e.* macules, papules, and pustules or scales may be present at the same time in different parts.

The pustular syphilide has been mistaken for small-pox; the squamous resembles psoriasis, but the scales are light and small, and there is no preference for extensor surfaces.

In the late secondary stage, and in the tertiary, ulceration of the skin is common. The ulcers have a sharply-cut circular edge and an ash-grey base. In neglected cases, their discharges may dry into thick greenish crusts, firmly adherent, and often limpet-shaped (*rupia*).

The other changes are—

“Mucous patches” in the mouth, and condylomata,—soft, flat, warty growths with a greyish secretion,—around the anus, on the scrotum, or in the groins.

Ulceration of the throat.

Syphilitic iritis.

Syphilitic periostitis.

Anæmia.

Loss of hair (alopecia) *not* followed usually by permanent baldness. Nocturnal headache and bone-pains.

4. *Third or Tertiary Stage*.—The anæmia has become more marked, and the manifestations of the disease are those of serious malnutrition. Cutaneous syphilides are not symmetrical as in the secondary stage, but scattered, and they tend to form deep ulcers, which leave well-marked scars. The bones are often affected, and periosteal “nodes” are common on the long bones and the calvarium. Syphilitic endarteritis may be the cause of aneurysm or of cerebral thrombosis. Deep-seated organic changes, due to the formation of gummata, are also very common. These growths may be found subcutaneously, in the muscles, or



in any of the internal organs. They are met with in the brain (Jacksonian epilepsy), bones, testicle, liver (syphilitic cirrhosis), heart, nose, and larynx, and elsewhere. Ultimately they may be gradually absorbed, thus causing cicatricial deformity of internal organs; or if they are superficial, they may ulcerate and discharge upon the surface.

Syphilis is of great importance as a factor in the production of other diseases, mainly chronic. While its range may perhaps be over-estimated, it is well to remember that Ricord's chief regret, at the end of his career, was that he had not diagnosed it often enough. There is one group of chronic diseases with which it has a very definite connection. They are known as *parasyphilitic* or *metasyphilitic*, i.e. the lesions are non-specific, but are due to degenerative changes set up by the syphilitic toxin. Chief among them are amyloid disease, epilepsy, and in hereditary syphilis, infantilism. Locomotor ataxia and general paralysis were until quite recently included in this class, but spirochætes have lately been demonstrated in the affected nervous tissues. It is therefore better to regard these diseases not as parasyphilitic, but as manifestations of a quaternary stage of syphilis.

In women, miscarriages or still-births are frequent. Should the child survive, it may be the subject of congenital syphilis.

The secondary stage lasts from six months to a year; the tertiary follows it from a year to three or four years after infection. Tertiary lesions are obstinate, and tend to relapse after treatment.

**Congenital Syphilis**, as we have seen, may be transmitted from the father or mother, or both. If the infection is through the father alone, the mother is immunised, and cannot be subsequently infected. In the tertiary stage the disease is rarely transmitted.

The child is seldom born obviously syphilised, for infants in whom syphilis becomes manifest *in utero* usually die *in utero*; but syphilitic pemphigus is an exception to this rule. It may be present at birth or appear within a week. Such infants usually show other signs (atrophy, snuffles, etc.) and do not long survive.

#### Symptoms.—

1. Born apparently healthy, within three months the child develops rhinitis, impeded breathing, and "snuffles." Necrosis of the nasal bones may cause *depressions at the root of the nose*.



2. General atrophy, slight or marked, with a peculiar "old man" appearance.
3. Fissures (*rhagades*) at the angles of the mouth.
4. Mucous patches in the mouth, condylomata at the anus.
5. Coppery erythema on the buttocks and inner sides of the thighs, or the palms and soles; onychia.
6. *Enlargement of the spleen*; epiphysitis; thickening of bone, or craniotabes.

Passing off in about a year, when the case is not fatal, symptoms tend to return at the second dentition and at puberty. The child is ill-nourished, the brow prominent, the bridge of the nose depressed; the upper central incisors of the permanent set are notched and peg-shaped ("Hutchinson" teeth); interstitial keratitis and iritis are frequent; "nodes" on the long bones are often present; occasionally synovitis of both knees is seen; and gummata of the internal organs may be a late occurrence. General paralysis and locomotor ataxia occasionally occur in juveniles as late manifestations of congenital syphilis.

**Diagnosis.**—Often impossible to mistake, syphilis is at times only to be recognised by a careful search for obscure evidences of its presence, or by minute inquiry into the history of the case. In cases which still remain doubtful, we may employ—

*The Wassermann Reaction.*—If the blood-corpuscles of one animal (ox) be injected into an animal of another species (rabbit), the serum of the latter becomes able to dissolve the corpuscles of animals of the former species (*hæmolysis*). If this hæmolytic or immune serum is heated to 55° C., it loses its hæmolytic property, which is restored to it on the addition of a little normal rabbit's or guinea-pig's serum. This process is exactly similar to that occurring in bacteriolytic sera (see Immunity, p. 14). In the immune serum a *thermo-stable* immune-body has developed, which acts only along with the *thermo-labile* complement; and the latter when destroyed can be replaced from normal serum. If such an immune serum, after heating to 55° C., is added to ox corpuscles, the immune-body present becomes linked to the corpuscles, which are said to be "sensitised," i.e. they are in a state of readiness for lysis on the addition of a normal serum containing complement. Any serum, therefore, which, on being added to the sensitised corpuscles, produces hæmolysis, may be inferred to contain free complement; of any serum which does not do so, it may be inferred that the complement is already



fixed or *deviated* by an antigen-immune-body combination. When syphilitic serum, containing immune-body, is mixed with watery extract of syphilitic liver, containing antigen (spirochætes), a large amount of complement is fixed. Hæmolysis therefore does *not* occur when the mixture is added to the sensitised corpuscles. If normal serum is used instead, the complement is not fixed, and hæmolysis takes place. On this principle of the "deviation of complement" the Wassermann test was based. The test remains valid, giving a positive result in about 90 per cent. of cases of active syphilis; but the principle has been invalidated, for it is found that alcoholic extracts of many other substances of a lipoidal nature fix complement along with syphilitic serum. No true explanation of the reaction is yet available. Positive results have also been recorded in yaws, sleeping-sickness, leprosy, scarlet fever, and some cases of diabetic acidosis and cancer of the stomach. The reaction is therefore not absolutely diagnostic, but must be taken along with the clinical evidence. A negative result does not exclude syphilis.

*Luetin reaction.*—Luetin is a substance extracted by Noguchi from pure cultures of the *spirochæte pallida*. When injected into the skin it produces, in syphilitic cases, a reaction which may assume one of three types:—(1) A *papular* form, in which a large indurated reddish papule, 5–10 mm. in diameter, appears within forty-eight hours, increases in size for three or four days, and then recedes; (2) A *pustular* form, in which the papule becomes first vesicular and after the fourth day pustular; (3) A *torpid* or latent form, in which there is no reaction for about ten days, after which small pustules form at the sites of the injection punctures. In the normal individual there may be a slight erythema at the point of injection, but this disappears within forty-eight hours. The reaction is due to anaphylaxis, and is specific for syphilis. It is less constant than the Wassermann reaction in primary and secondary syphilis, but much more often positive in tertiary, latent, and congenital syphilis, and in parasyphilis.

**Treatment.**—Hitherto there have been only two specific drugs, mercury and iodide of potassium, the first being of the greater value in the earlier stages, the second in the later. To these there now falls to be added a third, salvarsan or "606," the recent discovery of Ehrlich.

1. *Ordinary treatment.*—The management of the primary ore consists mainly in local cleanliness. If the diagnosis is



*absolutely certain* (it is made certain by the discovery of the spirochæte, although a negative result does not exclude syphilis), the use of mercury should be commenced at once, and may avert even secondary symptoms; but it may sometimes be advisable to wait till these appear before commencing specific treatment. The Wassermann test is usually negative in the early primary stage. Treatment consists in the early stages in the administration of one or other of the preparations of mercury. The preparations administered, and the methods of administration, are too numerous to describe in full. In the general run of cases Hutchinson's pill (Hydrarg. c. Cretâ gr. i (grm. 0·07), Pulv. Ipecac. Co. gr. i Sig., one or two thrice daily) is effective, if given for long periods. It does not produce salivation or diarrhœa. Treatment must not be stopped when symptoms have disappeared, but *must be continued for two to three years*. Where symptoms are more urgent, and a rapid effect is desired, inunction of blue ointment, 3i (grm. 4·0) daily, or hypodermic injection of corrosive sublimate gr.  $\frac{1}{8}$  (grm. 0·008) every second day, may be practised, or calomel gr. i–ii (grm. 0·07–0·14), suspended in sterile oil, may be injected into the gluteal muscles once a week. The condition of the mouth must be looked to, and smoking should be forbidden if there are lesions in the mouth. In the tertiary stages, iodide of potassium to a great extent takes the place of mercury, but the two are often advantageously combined. Doses of 5 to 20 grains (grm. 0·3–1·3) thrice daily usually suffice, but in cerebral syphilis the drug must be rapidly pushed to even larger doses.

In cases of pregnancy the mother must be actively treated if the father be syphilitic, even though she herself is apparently healthy. Infants suffering from congenital syphilis are best treated with grey powder gr.  $\frac{1}{8}$ – $\frac{1}{3}$  (grm. 0·008–0·02) thrice daily, or in severer cases by inunction.

In every case, besides treating the specific disease, the general health must be sedulously cared for. Anæmia may be treated by iron or arsenic, the general strength built up by hypophosphites, cod-liver oil, etc., and well-to-do patients are often rapidly improved by a course at such a spa as Aix-les-Bains.

2. *Treatment by salvarsan*.—The complex arsenical compounds known as arylarsonates have for some time been used with good results, but with all of them there has been a risk of optic atrophy and blindness. Salvarsan, or dioxydiamidoarsenobenzol, is free from this objection, and has few contra-indications. It may be given subcutaneously, intravenously, or



intramuscularly. Most of the subcutaneous and intramuscular methods are attended by considerable pain, but Wechsellmann employs subcutaneously a neutral suspension, the injection of which is alleged to be painless. In some instances necrosis of the skin and subcutaneous tissue has followed subcutaneous injection, and when a suspension is used, there is danger of giving a second injection before the first is completely absorbed, and thus causing poisoning by an overdose. The intravenous method, favoured by Ehrlich, is the safest, and is free from pain. For adults, the dose is 0.3 to 0.5 gm. A single injection often suffices to heal the manifest lesions, and to cause disappearance of the spirochætes and of the Wassermann reaction. "Recurrences" observed in nerve-trunk, etc., are attributed to the escape of isolated foci of spirochætes in poorly vascularised areas, and to their subsequent activity. For these, and other cases not yielding to a first injection, a second may be necessary, given not less than a fortnight after the first. The treatment has yielded specially brilliant results in cases refractory to mercury and iodide. A single injection often does as much apparent good as a course of mercury, and where a rapid effect is necessary, salvarsan is particularly indicated. But one injection does not cure, and even after several, although the Wassermann reaction may temporarily disappear, it may return at a later stage. Present opinion tends strongly to the combined use of mercury and salvarsan, and in cases of syphilis of the central nervous system it is advisable to give a preparatory mercurial course before salvarsan is used, with the object of lessening the risk of a marked reaction. Ehrlich regards cancer, the status lymphaticus, and Addison's disease as absolute contraindications to the use of salvarsan; Neisser adds to these, severe alcoholism, cachexia, hepatic affections, and degenerative lesions of the nervous system; and the presence of nephritis also increases its danger. Salvarsan, it will be seen, is coming to be regarded as an adjuvant to mercury; and its use is not entirely free from risk.

Neosalvarsan, the newer form of the drug, is more readily soluble and has a neutral reaction, but its solution rapidly decomposes. It can be given in more concentrated solution than salvarsan, but the solution must be made immediately before use. 0.9 gm. of neosalvarsan corresponds to 0.6 gm. of salvarsan. Whichever preparation is employed, the technique of the injections must be carefully learned.



## YAWS (FRAMBÆSIA)

A chronic infectious disease occurring in tropical countries, and characterised by a peculiar eruption.

**Etiology.**—Yaws is due to the *spirochæte pertenuis* or *pallidula*, an organism which morphologically closely resembles the *spirochæte pallida*, and is found in the lesions of the skin. The disease can be inoculated into apes, and in them the spirochæte is found in the skin lesions, and also in the lymphatic glands and spleen. Yaws has a close relationship to syphilis. It is highly contagious, but not through unbroken skin. A scratch or sore may be infected direct, through infected clothing, or by insects.

The cutaneous lesions are composed of granulation-tissue, and involve the deep layers of the epidermis and the papillary layer of the cutis vera.

**Symptoms.**—After an *incubation period*, varying from a fortnight to two months, there follows a week or so of malaise, and sometimes fever. An eruption then appears. It consists at first of minute itchy subcuticular papules, which rapidly increase in size, protrude through the epidermis, and become purulent at the top. The drying in of the pus produces a crust by which the “yaw” is covered. Some of the lesions remain small, others may grow to a considerable size, and form rounded projections on the surface of the skin. Some may ulcerate, but usually after a few weeks the crust falls off, and healing takes place. The lesions, which are painless throughout, occur in successive crops, and the disease may thus last for several months or even years. The Wassermann reaction is positive. The name frambæsia refers to the raspberry appearance of the granulomata (Fr. *framboise*), but the resemblance is somewhat slight.

The **Treatment** is on the same lines as that of syphilis. Until recent years, mercury and iodide of potassium were the chief remedies used, though arsenic has also proved serviceable. Salvarsan has recently been employed with excellent results, and is now the recognised specific treatment. Local applications, isolation, and disinfection of the clothing are the other necessary measures.

## TRYPANOSOMIASIS (SLEEPING SICKNESS)

A chronic disease of tropical countries, due to a protozoon, and characterised by fever, wasting, lassitude, enlargement of the lymphatic glands, and a terminal stage of lethargy.



**Etiology.**—The trypanosomata are protozoa of the sub-class Flagellata, living in the blood and tissues of various animals. Many species exist, some of them pathogenic, others non-pathogenic, to their vertebrate hosts, to which they are usually transmitted by the bite of a blood-sucking insect, and in which they multiply by longitudinal division. The sexual cycle is completed in the invertebrate host, an insect which has bitten an infected animal not usually becoming infective for several days; but the details of the sexual development of the parasite vary in different species, and are not yet worked out for all.

Besides man, rats, dogs, cattle, horses, and camels are liable to be attacked by different species of trypanosomes, producing diseases more or less similar, and characterised by wasting, anæmia, œdema, and fever. Wild herbivora may harbour the parasite without being seriously infected themselves.

Morphologically, the various species of parasite differ chiefly in size. A trypanosome is a minute, actively mobile, spindle-shaped mass of protoplasm, with a single flagellum at the anterior end. Near the middle there is a large *macronucleus*, and towards the posterior end a small deeply staining *micronucleus*, *kinetoculus*, or *centrosome*, in or near which originates the *undulatory membrane*, which runs forward like a crest above one surface of the protoplasmic mass, and terminates in the flagellum, by which and by the wavy motion of the membrane progression is effected. The parasites of man are the *trypanosoma gambiense*, which measures 17 to 28  $\mu$  in length, and 1.4 to 2  $\mu$  in breadth, and the *trypanosoma rhodesiense*, indistinguishable from it in the human blood, but showing certain morphological peculiarities when passed through the rat. The former is transmitted by the bite of a tsetse fly, the *glossina palpalis*, the latter by the *glossina morsitans*.

The organisms have recently been cultivated on a special medium (rabbit-blood agar).

The disease is prevalent on the West African coast, in the Congo basin, in Uganda, and also in the course of the Niger. It also occurs in Rhodesia and in Nyassaland. Natives are the chief sufferers, but Europeans are not exempt.

**Morbid Anatomy.**—Chronic inflammatory changes in the brain and cord and in their membranes, and enlargement of the lymphatic glands, are the principal features. There is also a relative lymphocytosis.

**Symptoms.**—The organism may be present in the blood for years before symptoms begin, and the disease may develop long after a return to Europe. The first stage may be marked merely by recurrent attacks of fever like that of malaria, and by enlargement of the glands and spleen, with intervals of apparent health. A patchy erythema is often present on the chest and back, and may involve the face. In such cases the terminal



stage may not appear for years. In other cases lassitude is present from the first, the gait and speech are slow, there is headache, the temperature rises at night, and the glands are usually enlarged. Irregular œdema is often present, and there may be dropsy of the cavities. Anæmia becomes prominent, and the blood shows a relative lymphocytosis, fine tremors appear in the tongue and hands, wasting sets in, and lethargy increases. In both types the terminal stage, to which the name "sleeping sickness" properly applies, is one of gradually deepening coma, which may be accompanied by convulsions, paralysis, or bed sores. The duration may vary from months to years, and death is often due to an intercurrent disease.

For early **diagnosis** puncture of an enlarged gland is advisable. The parasite is often found in the glandular juice more readily than in the blood.

**Treatment.**—Cure is only possible in the early stages, and many even of the early cases end fatally. Atoxyl in varying doses may be given hypodermically in 10 per cent. solution. Manson gives gr. iii every third day for two years, interrupting the course if there are symptoms of arsenical poisoning, and obtains good results. Antimony has also proved very successful. It is given intravenously in the form of tartar emetic, the dose being gr. ss. (grm. 0·03) increased to gr. i ss. (grm. 0·1) dissolved in about  $\bar{3}$ vi of normal saline. The two treatments may be combined or alternated.

### LEISHMANIASIS (KALA-AZAR)

A chronic infection of tropical countries, due to a protozoon, and characterised by fever, anæmia, hæmorrhages, wasting, and enlargement of the spleen.

**Etiology.**—The disease occurs chiefly in India, but also in Ceylon, China, Syria, Arabia, and Northern Africa. It rarely attacks Europeans.

The parasite, *Leishmania Donovanii*, discovered by Leishman in 1900, is possibly transmitted by the bed-bug (*cimex macrocephalus*), and in the Mediterranean basin possibly also by the dog-flea, since the dogs of that region are frequently infected with *Leishmania*. It is found in the spleen, liver, bone-marrow, and mesenteric glands, sometimes in the cutaneous ulcers, and occasionally in the peripheral blood. As seen in smears from the spleen, it is a round or oval body 2·5 to 3·5  $\mu$  in diameter, and contains a large rounded or oval chromatin body lying towards one side, and a smaller rod-shaped chromatin body lying at a tangent to it. In smears the "Leishman-Donovan bodies" are often free, in sections they are intracellular, being taken up by the mononuclear leucocytes, and by the endothelial cells of the capillaries and splenic sinuses. In these they multiply



by fission till they burst the cell, when they are again taken up by other cells. In cultures they develop a form resembling a trypanosome in possessing a flagellum at the anterior end, but without an undulatory membrane. From some of these flagellated forms there develops by longitudinal fission a hair-like mobile spirillary organism. The organism thus belongs to the sub-class Flagellata.

A form of kala-azar, confined to children of about two years of age, is found in Tunis and other parts of the Mediterranean coast. An obstinate form of chronic ulceration (*Biskra button*, *Delhi boil*, *Natal sore*), prevalent in tropical and subtropical countries, is also caused by a *Leishmania*; but whether these parasites are identical with *Leishmania Donovanii* is uncertain.

**Morbid Anatomy.**—Enlargement of the liver and spleen is constant, and the bone-marrow shows signs of proliferation. There are frequently intestinal ulcers, chiefly in the colon.

**Symptoms** are largely those of anæmia, and include pallor, hæmorrhages from mucous surfaces or into the skin (purpura), long continued irregular fever, and transitory œdema. There is marked leucopenia, with a relative lymphocytosis. Splenic enlargement is constant, hepatic common. Later, muscular atrophy and great emaciation are present. Cutaneous ulcers are frequent. The disease ends fatally after a few months or years.

For **diagnosis** hepatic puncture should be practised in preference to splenic, which involves the risk of fatal hæmorrhage.

**Treatment** has been hitherto unavailing. Quinine reduces the fever, and the arylarsonates or salvarsan may be tried.

## TUBERCULOSIS

A specific infective disease, produced by the action of the *bacillus tuberculosis*, and characterised by the formation of "tubercles," which tend to undergo degenerative changes in the direction of softening or of calcification. The disease may be local or generalised (acute miliary tuberculosis); in the local forms the lungs are oftenest involved, but any organ may be attacked.

**Etiology.**—Those predisposed to tuberculosis are frequently of feeble physical development, though even the athletic are commonly attacked. Town-dwellers oftener suffer than those living in the country; and insanitary occupations, exposure to dust, and confinement to close rooms are predisposing factors. A special type of thorax—the alar chest—often accompanies the pulmonary form (see Phthisis). The sexes are almost equally



liable. No age is exempt, but it is relatively less common in the old. Phthisis is a disease chiefly of adolescence and adult life; children oftener suffer from tuberculosis of bone, glands, peritoneum, or meninges.

Tuberculosis is not hereditary in the direct sense, although in rare instances infants have been born tuberculous, and the bacillus has been found in the placenta. But a *predisposition* to the disease is possibly inherited; the tissues of the offspring are less resistant to the attack of the bacillus. Those of tuberculous family history are less likely to respond favourably to treatment, if attacked, than those in whom the disease is sporadic. This may, however, be due rather to foetal inoculation with the toxins of tuberculosis than to inherited predisposition; and it must be remembered that tuberculosis *acquired* in infancy may long remain latent before it becomes clinically manifest.

*Modes of Infection.*—1. By inoculation of the tuberculous material (lupus, *post mortem* wounds). The process usually remains local.

2. *By inhalation of the dried and powdered sputum* (dust of apartments, etc.). Crowded and ill-ventilated dwellings, and sedentary occupations thus predispose to the disease.

3. By ingestion of tuberculous material, (a) by swallowing the sputum; or (b) by swallowing infected meat or *milk*. Organisms thus ingested may enter the system by the tonsils or by the intestine. In the first case they pass to the cervical or mediastinal glands, from which they may reach the lungs or the general circulation; in the second case they may cause primary intestinal tuberculosis, or may pass through the intact intestinal wall and infect the mesenteric glands or the lungs. The bovine type of bacillus has been conclusively proved to be infective for man, as is the bacillus of swine tuberculosis; but that of avian tuberculosis is not infective.

*The bacillus* is a small, motionless, rod-shaped organism, 3 to 4  $\mu$  in length, and often slightly curved. It is not mobile, and has no flagella. It stains slowly (best by the Ziehl-Neelsen method) with the aid of heat, and is difficult to decolorise by acids (acid-fast). The appearance of beading often noticed is probably due to the presence of vacuoles, and not to spore-formation. In older cultures long and occasionally branching filaments may be found, forms which ally the bacillus with the higher bacteria. The organism grows well on sterilised blood-serum at 37° C., but more rapidly on glycerin agar or glycerin broth, and on Dorset's egg-medium. Growth is arrested at 28° C. on one hand, and 42° C. on the other.

**Morbid Anatomy (general).**—The tubercle from which the disease gets its name is a small body visible to the naked eye



or with a lens. At first it is semi-transparent, grey, and gelatinous in appearance, later it becomes opaque, softened, and yellowish in the centre. Tubercles may be few or very numerous in the affected organs, and may be isolated or clustered in masses.

Microscopically they show from within outwards—

1. One or more central giant cells, each produced by the enlargement of a single epithelioid cell, the nucleus of which divides, while the protoplasm remains undivided. In rare instances several epithelioid cells may fuse to form a giant cell.

2. A layer of epithelioid cells.

3. A zone chiefly of lymphocytes, often surrounded by a network of fibres.

*There are no blood-vessels in the tubercle*, and therefore it is open to degenerative changes. The cells become fatty and soft, the softened material being discharged in sputum, urine, or stools; or there may be a conservative reaction, leading to the encapsulation (calcification) of the *materies morbi*, and to the survival of those affected with obsolete tubercle. Too often the advantage is on the side of the attack.

The bacilli are found in varying numbers in the giant cells and the epithelioid cells.

*There is nothing distinctive in the early processes of tubercle* except the discovery of the bacillus. Later, secondary inflammatory processes occur around the tubercles.

There are several methods by which tubercle may spread within the body of an infected patient:—(1) along mucous membranes (pharynx to Eustachian tube, middle ear, and meninges, etc.); (2) by swallowed sputum (intestinal ulcers); (3) by the lymphatics; and (4) by the blood. All these demand a local infection for a starting-point. But general tuberculosis, with which alone we deal just now, is usually started by the invasion of the wall of a blood-vessel by a local tuberculous process. The bacilli are carried in the blood-stream to all parts of the body.

## ACUTE MILIARY TUBERCULOSIS

**Etiology.**—The disease is frequent in early childhood, and not uncommon between twenty and thirty. It is usually secondary to chronic tuberculous processes in internal lymphatic glands or bones. It may follow other infective diseases—measles, whooping-cough, enteric.

There are three main types,—acute generalised miliary



tuberculosis, acute pulmonary tuberculosis, acute meningeal tuberculosis. The last will be described under Diseases of the Nervous System.

I. In **acute generalised miliary tuberculosis** tubercles are thickly scattered through all the organs. They are small, miliary or sub-miliary, and very numerous.

*The symptoms* are those of a profound general infection, and the disease is often mistaken for enteric fever (hence called the *typhoid* form). It begins with increasing weakness and anorexia, and after some time irregular fever appears. This may be continuous, remittent, or intermittent; oftenest continuous, with a difference of about  $2^{\circ}$  between morning and evening temperatures. The pulse is rapid and feeble, the tongue dry, the cheeks flushed. Delirium is marked, and usually of the typhoid type, deepening into coma. The pulmonary symptoms are those of slight bronchitis, or less commonly broncho-pneumonia. The respirations are rapid at first, and there is often cyanosis. The sputum is scanty, and may be entirely free of tubercle bacilli. Cheyne-Stokes breathing may appear later.

*The diagnosis* from enteric fever is often difficult. The most important points in favour of tuberculosis are—

- (1) Irregularity of the fever.
- (2) Comparative rarity of diarrhoea.
- (3) Absence of rose-spots.
- (4) Absence of the enteric bacillus in the blood.
- (5) Widal's reaction is negative.
- (6) Possible presence of tubercle in the choroid.

II. **The pulmonary form.**—The nodules are most numerous in the lungs, occurring either in groups or universally diffused. They are thickly set beneath the pleura. The lungs are congested, and often emphysematous. Collapsed patches, and areas of consolidation of varying size are to be found.

*Symptoms.*—The onset is usually sudden, with perhaps a previous history of cough or chronic phthisis. In children measles or whooping-cough may have gone before. Breathing is rapid, and dyspnoea marked. Cough may not be very troublesome. The sputum is mucous in character, but may be muco-purulent or rusty; tubercle bacilli are seldom found. Cyanosis is prominent. Fever is well marked ( $102^{\circ}$ – $104^{\circ}$  F.). The physical signs are those of diffuse bronchitis and acute emphysema—hyper-resonant percussion, varying intensity of



breath-sounds, presence of sibilus or rhonchus, and medium crepitations. There is sometimes diminished resonance at the bases (bronchopneumonia).

The duration of the disease varies from one to several weeks. It is generally fatal.

The diagnosis is assisted by the history, suddenness of onset, and possible discovery of the bacillus in the sputum. When the bacillus is present, however, it is due to the presence of a primary focus of chronic tuberculosis in the lung. In the acute process the newly-formed tubercles have rarely time to break down and discharge their organisms in the sputum before the patient's death.

*Treatment* in both forms is purely symptomatic. Recovery is exceptional.

Local tuberculosis will be dealt with under Lungs, Alimentary Tract, Nervous System, etc.

## LEPROSY

A chronic infectious disease due to the *bacillus lepræ*, and characterised by the appearance of granulomata in the skin and mucous membranes, or by infiltrations of nerve trunks leading to anæsthesia, paralysis, and trophic changes.

**Etiology.**—In Europe the disease is found chiefly in Norway, but also in Southern Russia, Turkey, and Greece. In the rest of the world it is widely spread in tropical and subtropical climates. The generally accepted view is that it is transmitted by contagion, the bacillus being found in the discharges from the skin and mucous membranes, and very abundantly in the nasal discharge; but for such transmission prolonged exposure is necessary. Children below three years of age rarely suffer, but with this exception, persons of any age and either sex are liable. Overcrowding, poverty, and poor feeding are predisposing factors.

The *bacillus lepræ* closely resembles the tubercle bacillus in size and staining reactions, being acid-fast. It may stain uniformly, or the protoplasm may be fragmented. It is non-mobile. Although various observers claim to have cultivated it, their work cannot be considered absolutely conclusive, as the organisms they describe are not uniform, and others have failed to reproduce their results. The organism would seem to be pleomorphic and, like the *b. tuberculosis*, allied to the higher bacteria of the genus *streptothrix*. It is found in great numbers, and mainly intracellularly, in the granulomata of the skin and mucous membranes, in the lymph-spaces and lymphatic glands, and, in the anæsthetic form, in nerve-trunks. Sands and Long have recently discovered it in bed-bugs that have bitten lepers, and this forms a possible method of transmission.



Nothing is definitely known of the incubation period but that it is long.

**Morbid Anatomy.**—In the *tubercular* form, nodules or diffuse infiltrations of granulation-tissue appear in the cutis and in mucous membranes, increase in size, and may lead to ulceration. The richly cellular tissue is supported by a stroma at first delicate, later very dense. The bacilli lie in masses in and between the cells. The face, cornea, interior of the nose and mouth, the backs of the hands and feet, and the extensor surfaces of the arms and legs are chiefly affected. The spleen, liver, and testicles suffer secondarily.

In the *anæsthetic* form, diffuse infiltration of the nerve trunks causes destruction of their fibres. Trophic changes follow:—atrophy or bullæ of the skin, anæsthesia, gangrene and separation of parts, muscular paralysis.

**Symptoms.**—I. *Tubercular or nodular leprosy.* With or without prodromata a febrile attack occurs, during or just after which reddish or bronzed erythematous patches, slightly raised, at first hyperæsthetic, later defective in sensation, appear upon the face and limbs. They fade with the fever, leaving faint brownish stains or slight hardening of the skin. After weeks or months the attack is repeated, and perhaps affects other areas of skin. These “leprous storms” keep recurring, and ultimately raised nodules appear on the site of the former eruption or elsewhere, pink at first, and afterwards of a dirty brown tint. These, too, may fade, or persist till a fresh febrile attack adds to their number. Thus the skin of the face is thickened, and its folds deepened, the whole face becoming broadened, and assuming a “leonine” aspect. Nodules appear on the limbs on the sites already mentioned; the cornea is attacked, and vision may be destroyed; the interior of the nose suffers early and severely; and the fauces, vocal cords, and larynx may be involved. The nodules ultimately ulcerate, open sores and cicatrices being seen upon the skin. The constitution suffers from the febrile attacks; dyspepsia first, and then prostration follow; and the disease frequently ends in phthisis. The total duration is from two to eight years.

II. *Anæsthetic leprosy.* The prodromata, which may last for many months, are neuralgic pains, and sometimes weakness or wasting of the forearm muscles. Then pale or light-yellowish itchy spots, not elevated, and often symmetrical, appear on the back and extensor aspects of the limbs, and sometimes on the face, while the corresponding nerve trunks are thickened



and tender. This stage lasts for two or three years. Later, the patches become anæsthetic, and cease to secrete sweat; their surface is white, and their edges serpiginous. The anæsthesia extends beyond them, up to the elbows or knees, or higher; bullæ appear on the limbs and trunk; the fingers contract, and parts of them may drop off; perforating ulcers attack the feet, spontaneous amputation of the toes is common; and the ears may also be mutilated. The temperature is subnormal, except during the eruption of bullæ. After from six to ten years, the third stage appears, and is marked by muscular paralysis, the third and seventh nerves being often implicated, and by dry or moist gangrene of the extremities. The course of the disease is very slow, patients sometimes surviving for twenty or thirty years.

A "mixed form" is also described. The name is self-explanatory.

**Treatment.**—Of drugs chaulmoogra oil is most generally used, in doses of m. x-xl (cc. 0·6–3·0) thrice daily, and at the same time by inunction. Arsenic, potassium iodide, salicylate of soda, etc., have been advocated. Salvarsan has no advantage over other arsenical preparations. Fresh air, good food, and avoidance of fatigue do much to alleviate the condition, and injections of tuberculin have sometimes produced temporary quiescence. Partial segregation is advisable for the protection of others.

As regards specific remedies, serum treatment has been tried unsuccessfully, but favourable results have been reported from the use of *nastin*. This is a fatty acid-fast substance extracted by means of benzoyl chloride from cultures of a streptothrix (*streptothrix leproides*) isolated by Deycke and Raschid from cases of leprosy. An oily solution of this substance is injected hypodermically. The treatment must be continued for many months, or even up to two years. Vaccines made from the organisms isolated by various observers have given no brilliant results.



# DISORDERS OF NUTRITION

## GOUT (PODAGRA)

A NUTRITIONAL disorder, associated with an excessive amount of uric acid in the blood, and deposits of biurate of sodium in the joints, and manifested clinically by periodical attacks of acute arthritis, certain visceral disturbances, and later, deformity of the joints attacked.

**Etiology.**—1. Heredity is a most important factor, and the disease is curiously developed in the grandchildren. It is more frequent in the male, and it occurs chiefly in middle or later life, although it is not unknown about the age of puberty.

2. Habitual indulgence in heavy or sweet wines, and heavy malt liquors. The consumption of light wine or of spirits does not predispose to the same extent. The disease is more common in England and Germany than in Scotland and Italy.

3. Excessive eating, particularly of nitrogenous food, is as important a factor as excessive drinking, especially when combined with a sedentary life.

4. When associated with defective hygiene, and with excessive consumption of malt liquors, poor or insufficient food may cause the disease (*poor man's gout*).

5. Chronic lead-poisoning is often accompanied by gout.

6. In those predisposed to gout, worry, emotion, or a trivial injury may determine an attack.

**Pathology.**—In no disease can we point to a more constant feature than excess of uric acid in cases of gout. This substance is found in excess in the gouty blood, in the gouty joints, and in the exuded serum of the gouty arthritis. It would therefore seem that the disease is due either to the accumulation of uric acid in excess, or to those conditions which give rise to the excessive formation of uric acid. But the injection of uric acid



into the blood of a healthy individual does not produce gout. Further, excess of uric acid may be always produced by deficient oxidation, and is common in many diseases, such as leukæmia and pernicious anæmia, in which arthritis is by no means a common complication. There must, therefore, be a "something more" than mere excess of uric acid to cause such a condition as gout.

Uric acid is the chief member of the group of "purin bodies," of which the others are xanthin, hypoxanthin, guanin, and adenin. All these bodies are derived from nucleins, *i.e.* from the breaking down of tissues rich in cells, and uric acid represents a further stage in the oxidation of the rest. Obviously, there are two possible sources from which the purin bodies, and therefore uric acid, may be derived, (1) the breaking down of the cellular tissues of the organism itself, and (2) the nucleins ingested in the food (*endogenous* and *exogenous* purin bodies). Endogenous uric acid, then, is the result of tissue waste; the quantity of exogenous uric acid, stated by Osler as from 40 to 60 per cent. of the total purin content, can evidently be modified by modification of the diet.

The excess of uric acid in gout may be accounted for in one of three ways, by increased formation, deficient oxidation, or deficient elimination. Increased formation is a possible factor where the diet is habitually rich in purin bodies (nitrogenous food), although, given normal eliminative capacity, even an excess of exogenous purin bodies will be got rid of in the urine. It also exists where richly cellular tissues are broken down with abnormal rapidity, as in leukæmia; but this condition is not present in gout. There is more evidence for deficient elimination. Sir William Roberts held that, owing to defective alkalinity of the blood, the uric acid, circulating as a soluble sodium quadriurate, became less easily eliminable, being converted into an insoluble biurate, which was deposited in the joints and tissues. It is not now held that the blood is deficient in alkalinity, but a retention of nitrogen has been demonstrated in gout, and it is believed by some that the uric acid circulates in a different organic combination from that of healthy persons, and thus cannot be eliminated by the kidneys. Garrod, on the other hand, attributes its retention to defective action of the kidneys, which in gout are at least very frequently diseased. Deficient oxidation has also been invoked as a cause of gout, the supposition being that the ferment (*oxidase*) in the liver and elsewhere which converts uric acid into urea exists in insufficient quantity.



There are yet other theories, in which the excess of uric acid does not hold such an important place. Ebstein considers a change in the nutrition of the tissues, leading to necrosis, to be the primary factor; urates are then secondarily deposited in the necrosed areas. A nervous origin of gout, supported *inter alia* by the influence of emotion and worry, is maintained by Sir Dyce Duckworth and others. A bacterial toxin has been supposed to account for the symptoms.

It will be clear that the pathology of gout is still unknown.

**Morbid Anatomy.**—The metatarso-phalangeal joint of the great toe is most often affected. If we examine the joint at various stages, we find the following conditions—

A deposit of fine crystalline needles apparently in the *superficial* parts of the cartilages, but really interstitial; next, the synovial membranes, cartilages, and ligaments become covered with a chalk-like deposit of biurate of soda. Underneath the deposit, the tissues are in a more or less necrosed condition. The synovial fluid may contain crystals of the biurate.

Later, the cartilages may be eroded, and the synovial membranes thickened. The ends of the bones are enlarged and the joints deformed. Nodular masses around the joints, consisting of collections of biurate of soda *plus* calcium phosphate, form the so-called chalk stones or *tophi* of chronic gout, which may ulcerate through the skin. These deposits may occur elsewhere than in the joints, as in the lobes of the ear, tendinous aponeurosis of muscles, sclerotic of the eye, etc. Some or all of the other joints of the feet and hands may become involved, and even the larger joints of the limbs, but rarely the shoulder or hip. Interstitial nephritis (cirrhosis of the kidney) is common in gout, and is associated with deposits of biurate in the intertubular tissue. Arteriosclerosis, leading to hypertrophy of the left ventricle, is also common.

**Symptoms of Acute Gout.**—Usually there are some premonitory symptoms, such as giddiness, mental depression, flatulence, irritability of temper, scanty and high-coloured urine, etc. The attack most commonly commences in the early hours of the morning, with severe pain in the big toe. The pain increases to acute agony in some cases, rendering sleep or rest out of the question. The patient becomes very feverish, or he may feel chilly, and shiver violently without any rise of temperature. The joint at first is bright red, and exquisitely tender; later, it is more swollen, and of a livid or dull dusky



red, with distended venules standing out. The swelling extends some distance from the joints. The skin desquamates in thick flakes when the attack is over. An attack lasts from five to twelve days, but the severe pain is not constant, or rather there are lulls, with exacerbations of severe pain at intervals of the day, especially at night-time. The urine at first is scanty and high-coloured, *but the uric acid is diminished in amount*; later, the excretion of *uric acid is very much increased*. During the attack the patient usually is most irritable, the tongue is furred, the breath may be offensive, he has no appetite, and the bowels are constipated. Subsequent attacks may affect the joint first implicated, or a number of joints may become involved. If the attacks are fairly frequent, they cause the so-called chronic gout; that is, a condition recognised by a characteristic deformity of the joints, and important changes in various organs, especially the red granular, cirrhotic, or "gouty kidney."

**Chronic Gout** must not be confounded with rheumatoid arthritis, though the sufferer may be crippled in much the same manner.

The small joints of the toes and fingers are most often affected. The fingers are stiff, swollen, flexed or extended, and sometimes deflected to the ulnar side. Their joints are often irregularly distended by periarticular "tophi," which at first lie under the skin, but may ulcerate through, allowing the "chalk-stones" to escape. Tophi may also form in the bursæ, and in the ears. At a later stage the wrists, ankles, and even the elbows and knees may be affected, and fixation of the joints may follow.

The kidney undergoes a marked cirrhotic change (*see* Chronic Bright's Disease), and the urine becomes correspondingly altered. In such cases, the pyramidal area of the kidney often shows yellowish-white streaks of urate crystals.

**Suppressed Gout** is a condition in which the development of internal symptoms coincides with, and may be attributed to inhibition of the joint affection. The symptoms may be gastrointestinal, cardiac, or cerebral.

**Irregular Gout** is the term applied to various manifestations of the "gouty diathesis" in persons who do not suffer from articular gout. It occurs most frequently in those hereditarily predisposed, but is also common in the acquired disease.



Acute attacks usually affect the digestive, circulatory, respiratory, or nervous system.

1. Digestive system. Œsophageal spasm, gastralgia or gastritis, colic or enteritis, or hepatic diseases may be found.

2. Circulatory phenomena are palpitation or irregularity, cardialgia or angina pectoris, dyspnœa, syncope, and phlebitis.

3. In connection with the respiratory tract, bronchitis is frequent.

4. Neuralgia or neuritis, meningitis or cerebral congestion may affect the nervous system.

Certain skin diseases, especially eczema, are also among the acute symptoms. More chronic conditions are asthma, chronic bronchitis; renal calculus, gravel, or gouty urethritis; and an insidious iritis which may go on to destruction of the eye.

The chief *complications* are those already mentioned, namely chronic renal disease, arteriosclerosis, which may lead to apoplexy, and cardiac hypertrophy, often ending in dilatation.

*Garrod's thread test* is designed to detect excess of uric acid in the blood. Take 5ij of blood serum or of the fluid raised by a blister, and mix in a watch-glass with five or six drops of acetic acid. Immerse in the fluid one or two ultimate strands from a piece of linen, and leave in a warm place until evaporated to a jelly-like consistency. Transfer to the stage of a microscope, and examine the strands with a low power for uric acid crystals. The test is not very reliable.

**Treatment.**—During the acute stage—

1. The limb must be kept at rest and elevated, and hot fomentations should be applied to the joints; or they may be wrapped in cotton-wool, after the application of an anodyne liniment sprinkled on lint.

2. The diet should consist of milk and barley-water, and farinaceous foods. Plain water may be freely taken, and the mineral waters have no advantage over it.

3. Medicinal treatment should begin with a mercurial purge, and the bowels should be daily opened by a saline laxative. Colchicum wine m. xx-xxx (cc. 1·0-2·0), should be given every four hours, along with citrate of potassium and hyoscyamus. It should be stopped when the pain is relieved, and if it fails, morphia may be required. Laudanum or lead and opium lotion may be added to the fomentations.

Atophan (phenyl-chinolin-carboxylic acid), a recently introduced remedy, has been used with much benefit both in acute



and subacute gout. It is given in tablets containing gr.  $7\frac{1}{2}$  (grm. 0.5), an average dose being one tablet thrice daily after meals, and one at night.

When the acute attack subsides, gentle friction or shampooing, and passive movements are beneficial.

*Treatment of Chronic Gout.*—Careful regulation of diet, avoidance of food rich in nucleins (sweetbreads, liver, kidneys), moderation in the use of alcohol, or still better, total abstinence, and proper exercise, are the principal points to remember. Arsenic, strychnine, quinine, guaiacum, and iodide of potassium may all be useful in different cases. A course of waters at a spa is valuable from the regular life that it ensures.

### RICKETS (RHACHITIS)

A nutritional disorder, occurring in infants and young children, attended by changes in the development of the bones, and clinically characterised by wasting, stunted growth, characteristic physiognomy and deformities.

**Etiology.**—The disease occurs chiefly among the poor of overcrowded cities, as a result of improper feeding, want of sunlight, and other bad hygienic conditions, among which is want of exercise. Congenital syphilis is a predisposing cause, and may have to do with its occasional appearance in children of wealthy parents. The factor most frequently associated with its occurrence is faulty (not insufficient) feeding, and the diet is very constantly found to be deficient in animal fats and proteids. Hence infants reared by hand upon proprietary foods, those kept too long at the breast, and those nursed by a sickly mother, are more likely to suffer than the breast-fed children of a healthy woman. Quite recently, Edie and Simpson have adduced evidence to show that rickets is more common where white bread is used in preference to wholemeal bread, and they suggest a deficiency of organic phosphates as the cause. The theory that it is due to a defective supply of lime in the food is now abandoned as contrary to the evidence. The disease is not hereditary.

**Morbid Anatomy.**—1. *The bones.* The long bones, the ribs, and the skull are chiefly affected.

- (1) The calcareous salts are much diminished in amount, from imperfect assimilation.



- (2) The cartilaginous epiphyses are thickened, but ossification is delayed. The bluish cartilaginous zone is increased in thickness, very irregular, and softer than is normal. The line of ossification is irregular, very vascular, and spongy. The fibrous periosteum is thickened, and proliferation of bone beneath it is active; but the bone so formed ossifies imperfectly, while the medullary cavity increases at the normal rate. This normal increase, along with the delayed ossification, causes softening of the shaft; there is no *abnormal* absorption of the earthy salts.
- (3) Periosteal proliferation causes thickening of the flat bones of the skull (frontal and parietal), but ossification is slow, and the fontanelles long remain open. The occipital bone is apt to be thinned by the pressure of the growing brain, so that it may crackle like parchment under the finger (*craniotabes*). The top of the head is flat, and the forehead broad. When ossification is complete the sutures are prominent.

Kassowitz regards the hyperæmia of the bone-forming tissues as the primary lesion, and he has shown that such hyperæmia leads to defective deposition of lime salts.

2. The spleen and lymphatic glands generally show extensive fibroid changes. The spleen is usually enlarged and the liver may also be so. This results in enlargement of the abdomen, increased by flatulent distension.

3. The muscles are small and flabby, and their striæ are indistinct.

4. The blood is often said to be anæmic, but Findlay has shown that this is by no means constant.

**Symptoms.**—The symptoms of a typical case of rickets are very characteristic.

**General configuration.**—The head is elongated from back to front, the forehead is square and overhanging, the fontanelles are slow in closing, and the veins in the skin are distended. The flat bones are usually much thickened, and frequently “bossed” by irregular masses of calcified material. Often, however, the occipital bone and certain parts of the parietal bones are *thinned*. Though the skull as a whole is enlarged, the puny face makes the enlargement more noticeable by comparison; measurements show the enlargement to be more



apparent than real. The eruption of the teeth is delayed by several months.

*The ribs* often show characteristic "beading" at the junction of the costal cartilages (*rhachitic rosary*). Pressure of the external air on the softened anterior ends of the ribs produces the "rickety chest," characterised by a shallow vertical depression on each side of the sternum. "*Pigeon breast*" is due to impeded inspiration, as is "*Harrison's groove*," a transverse depression running from the xiphoid cartilage towards the axilla.

*The lower limbs* are bowed, or sickle-shaped, and show well-marked epiphyseal enlargements, especially at the lower end of the *tibia*.

*The upper limbs* also show the most marked changes at the lower ends of the ulna and radius, but the humerus and clavicle may also be affected.

*The pelvis* is often much deformed, and this may later add serious complications to parturition.

*The spine* is bowed forwards from muscular weakness. There may be lateral curvature.

The deformities, dependent partly on excessive deposits of calcareous material, are largely due to the yielding of the softened bone to mechanical causes; hence the necessity of rickety subjects avoiding any undue or avoidable strain, such as too early walking.

The most prominent *general symptoms* are three:—profuse sweating of the head at night, throwing off the bedclothes even in cold weather, and crying on being moved or handled. The last, usually ascribed to a general tenderness, Eustace Smith thinks to be due to a desire to limit respiratory movement. The mind is often backward; the body is stunted and may be emaciated, but the abdomen is prominent from flatulent distension, and from the enlargement of the liver and spleen. The bowels are sometimes costive, sometimes loose, and the stools are usually pasty, or green like boiled spinach, and very foetid. Taken along with the deformities, these symptoms make a diagnosis easy. Of the many *complications*, pulmonary diseases, convulsions, tetany, and laryngismus stridulus must be specially mentioned. Green-stick fractures are not uncommon.

A form of *late rickets* is described, in which the onset may



occur from the fourth to the twelfth year. It sometimes follows the febrile diseases of childhood.

**Treatment** is mainly dietetic and hygienic. The child should have a daily warm bath, and should be kept much in the fresh air and sunshine. If the mother be unhealthy, a wet nurse should be provided, and if this is impossible, artificial feeding must be resorted to, cows' milk, suitably diluted, being the essential element of the diet, which may be carefully added to after the first year. Excess of carbohydrates must be avoided. Walking must be prohibited to prevent deformity, and for this purpose splints projecting beyond the feet may be applied. The chief medicinal remedies are phosphorus, cod-liver oil, and syrup of the iodide of iron. Kassowitz strongly advocates the following:—phosphorus, 0·01, cod-liver oil, 100; one to three teaspoonfuls in the twenty-four hours. Daily friction with sweet oil is often beneficial.

### BERI-BERI

An endemic and epidemic multiple neuritis, characterised by paralysis and dropsy.

**Etiology.**—While there are still some writers who maintain the view that beri-beri is of microbic or toxic origin, and ought therefore to be placed among the infections, the great majority, as a result of the work of recent years, have accepted the position that it is a nutritional disorder, due to the absence of certain protective elements from the food. It occurs mainly among rice-consuming peoples, and therefore the relationship of rice to its production was first investigated. It has been found that when white or polished rice forms the main ingredient of the dietary, beri-beri frequently develops, whereas it is rare when the whole grain is used; and cases which have occurred on a polished rice dietary can be cured by the use of the whole grain. The pericarp, removed by milling, contains a substance which is probably an organic base, and to which Casimir Funk has given the name of *vitamine*. The absence of *vitamine* from the dietary is responsible for the production of the disease, and the administration of an alcoholic extract of the millings will suffice to cure it. But beri-beri also occurs in certain instances where the dietary does not consist of polished rice—on board ship for example, and notably among the labourers on a Brazilian railway whose diet consisted mainly of tinned vegetables and meal and of white flour. It has been shown, how-



ever, that there is more than one vitamine ; and if the vitamines are removed from any class of food by whatever process (milling, sterilisation, etc.) the result will be the same.

The chief habitat of the disease is Japan, the Malay Archipelago, Burma, and Brazil, but it occurs in other localities. Cases are sometimes brought by sea to the ports of this country, and some originate in ships towards the end of long voyages. Overcrowding, warmth, and moisture are predisposing causes. Males between fifteen and twenty-five are oftenest attacked, but neither sex and no age is exempt. The incidence and mortality of beri-beri have much diminished since its etiology has been definitely established.

**Morbid Anatomy.**—In all cases, inflammatory and degenerative changes are found in the axis cylinders and medullary sheaths of the peripheral nerves. In acute cases, the phrenic and vagus also suffer. Wasting and degeneration of the muscular fibres, both voluntary and cardiac, are present, and in the “wet” form of the disease, œdema, and dropsy of the body-cavities.

**Symptoms.**—The disease has two main forms :—

1. *Atrophic, dry, or paralytic.* Weakness and pains in the limbs are followed by rapid wasting, loss of power, and impairment of sensation or anæsthesia. The phrenic and vagus are sometimes attacked, and the heart may be dilated. Most cases end in slow recovery.

2. *Dropsical or wet.* Wasting is slight, and loss of power not marked. The whole subcutaneous tissues become œdematous, and there is effusion into the serous sacs. Cardiac symptoms are common ; albuminuria is absent.

A *rudimentary* type is described, in which paresis and paræsthesiæ replace paralysis and anæsthesia, dropsy is slight or absent, and cardiac symptoms trifling ; and an acute, *pernicious* or *cardiac* type, marked by symptoms of acute heart failure. The rudimentary type may persist for months, and recur with each warm season ; the cardiac ends in death, sometimes in a few hours, usually in a few weeks.

**Treatment.**—Alteration of the faulty diet is the essential measure, any others being directed to the relief of symptoms. The dietary should be mainly nitrogenous. When polished rice has been responsible, alcoholic extract of the millings, or yeast,



which contains a potent vitamine, should be given. The heart must be supported by strychnine or, if need be, digitalis. In acute heart failure purgation and bleeding are necessary, and in dropsy purgation or aspiration of serous cavities. Electricity and massage are needed for the wasted muscles.

The use of undermilled instead of polished rice is the chief prophylactic measure. It has done away with beri-beri in all the Siam Government institutions and among the gendarmerie.

### CHRONIC RHEUMATISM (FIBROSITIS)

An affection characterised by pain and stiffness of the joints, and occurring principally in the elderly.

**Etiology.**—The disease is most common amongst the middle-aged poor, particularly those who are exposed to cold and wet. Very rarely it follows acute rheumatism.

**Morbid Anatomy.**—The fibrous tissues are chiefly affected. The synovial membranes may be reddened, but effusion is very slight. The capsules and ligaments of joints, the sheaths of adjacent tendons, or the aponeurotic sheaths of muscles may be implicated. These structures become thickened and inflamed, and thus the movements of the joint are limited, but there is little deformity. Bony ankylosis is not met with.

**Symptoms.**—Pain and stiffness of the parts involved are the main features. The joints may be a little swollen. The pain is usually worse on movement, but continued exercise mitigates it. It is more marked at night and after rest. In the morning, on attempting to rise, the pain may be very severe, but after working for a time it becomes tolerable though it does not disappear. Many joints are usually affected, and they can often be felt to creak.

**Treatment.**—Internal medication is unsatisfactory. Guaiacum, iodide of potassium, and arsenic are recommended, but the salicylates are ineffectual. Local measures, such as counter-irritation, massage, passive movement, and hydrotherapy are much more useful. The passage of large electric currents through the affected part is frequently of benefit. Obstinate painful nodules may be excised. A course of baths, and a warm winter climate may be of great service in cases where such measures are possible.



## MUSCULAR RHEUMATISM (MYALGIA)

A painful affection of the voluntary muscles and their fibrous attachments.

**Etiology.**—The disease results from overstrain or from exposure to cold and damp. The gouty habit predisposes to it, and it is most common in men. It has no direct connection with acute rheumatism, but is probably due to inflammatory changes in the muscular fibrous tissues, associated, perhaps, with neuralgic affection of the nerve-endings. Its most frequent *varieties* are—

1. *Lumbago*.—The aponeurosis of the erector spinæ and latissimus dorsi is most frequently involved. The pain is often intense, and may markedly affect the locomotion. Often the onset of the pain is traced to some physical exertion, such as getting up into the saddle, or lifting heavy weights.

2. *Pleurodynia*.—The sheaths of the pectoral muscles, intercostals, or serratus magnus, are most commonly affected. The respiratory movements of the affected side are much embarrassed. This affection may be mistaken for pleurisy, as the movements of the affected muscles often cause a distinct fremitus. The absence of other physical signs should prevent such an error.

3. *Muscular Torticollis or Stiff Neck*.—Here the cervical muscles, especially the sterno-mastoid, are affected. This condition must not be confounded with spasmodic torticollis. Many other varieties are described.

**Treatment.**—At the outset the bowels should be opened by a saline. Rest to the muscles is essential, and may be obtained by strapping, as in pleurodynia, or by plasters in lumbago, in which hot fomentations are also useful. Anodyne liniments or galvanism may be employed. In lumbago acupuncture is of great service. In chronic cases, iodide of potassium or guaiacum should be given. Acetyl-salicylic acid, gr. x-xv (grm. 0.65-1.0) thrice daily, is of value for the relief of pain.

## RHEUMATOID ARTHRITIS

A chronic inflammatory affection of the joints, giving rise to changes in the synovial membrane and the surrounding tissues, and sometimes to changes in the bones.

**Etiology.**—The disease is most common amongst females, and



usually begins between the ages of twenty and forty. It may, however, occur at any age. Heredity, uterine disorders, a tuberculous family history, gout or rheumatism, grief, worry, etc., are looked upon as important etiological factors. It is most prevalent amongst the poor, and is favoured by exposure to cold and damp. It may, in fact, be said that almost any influence tending to lower the vitality may predispose to the disease.

**Pathology.**—There are two main theories. The view that the disease is a tropho-neurosis is supported by the symmetry of the lesions, the nutritional disturbances in the skin and muscles, and the frequently concurrent neuritis.

Evidence, however, accumulates in favour of the second view, namely, that the disease is of microbic origin. It is often the sequel to an acute infection, in certain cases its onset is acute, in children there is often widespread enlargement of lymphatic glands and of the spleen, and organisms of various kinds have been found in the articular fluid. Some consider "that it is due to a settlement of micro-organisms in the affected joints, that they there produce a toxin, and that that toxin, passing into the circulation, is responsible for any nervous symptoms which occur" (Luff). Others, with much recent evidence to justify them, think that the joint condition is due to a chronic sapræmia, the result of a local microbic infection. Poncet has recently pointed out that many cases are really of tuberculous origin, being due to the invasion of the joints by tuberculo-toxins derived from an obvious or latent focus of tuberculosis elsewhere in the body. The etiological importance of a tuberculous family history, and the frequency of arthritic pains in tuberculous subjects without tuberculosis of the joints, become explicable on this assumption.

**Morbid Anatomy.**—The morbid changes found in the joints affected are similar to those of Charcot's joint disease, sometimes a complication in cases of locomotor ataxia. The disease begins in the cartilages and synovial membranes. The cartilage becomes soft and vascular, and is gradually absorbed. The result is approximation of the two articular surfaces of the bones, which, by rubbing together, become very dense, hard, and highly polished. The surfaces at the same time become broadened out and lipped at the edges. Underneath the new and dense ivory-like bone, rarefaction and atrophy may occur, and lead to shortening and deformity.

The synovial membranes are inflamed and thickened: often portions become detached and form loose bodies in the joint.



The ligaments are much thickened, and often contracted; sometimes they calcify and cause more or less ankylosis. At the margins of the joints, where the pressure is less, ossification goes on, resulting in the formation of irregular bony outgrowths, termed osteophytes. The ankylosis is rarely complete, and there is no tendency to suppuration.

A rheumatoid type is described, in which the synovial membranes and ligaments are chiefly affected (*rheumatoid arthritis*); and an osteoid type (*osteo-arthritis*) in which the atrophic changes in the cartilages and bones predominate.

Muscular atrophy is common, as are neuritis and trophic changes in the skin.

### Symptoms.—

*The Ordinary Chronic Form.*—The joints are involved symmetrically, although the process generally begins on one side, and becomes symmetrical by extension. The smaller joints, especially the metacarpo-phalangeal and inter-phalangeal joints of the hands, are usually first affected. At first the joints may be swollen, red, and tender, but the more constant features are stiffness and gradually increasing deformity, without any marked signs of inflammation. In confirmed cases, the joint changes and the accompanying muscular atrophy cause the extremities to assume a very characteristic appearance. The lower ends of the ulna and radius project at the wrist, the metacarpo-phalangeal joints are flexed, the first phalangeal joints are over-extended, the second are flexed, and the fingers deviate to the ulnar side. The joints give forth a creaking sound when moved.

When the disease is confined to the hands, osteophytes may form at the sides of the terminal phalanges, and are known as "Heberden's Nodes." After the hands and wrists, the knees and ankles are most commonly attacked, but all the articulations of the body may be implicated, and joints such as the sternoclavicular and temporo-maxillary, which escape in acute rheumatism, are often affected. The muscular atrophy, though no doubt largely due to trophic influences, is contributed to by non-use.

The *monoarticular* form of the disease affects as a rule the spine, shoulder, or hip-joint, and occurs chiefly in elderly men (*morbus coxæ senilis*). The knee is often affected in women about the menopause.

The *rheumatoid* type, beginning with fusiform swelling of the interphalangeal joints, and extending to the larger joints, in which there is much fluid effusion, is the commoner in young



women. It may set in acutely, with much pain, fever, and a rapid pulse, the acute symptoms disappearing after about a month, while some disorganisation of the joints remains, and is added to in subsequent attacks (*acute rheumatoid arthritis*). The *osteo-arthritic* type, in which the changes chiefly affect the cartilages and bones, creaking is perceptible on movement, and osteophytes may cause locking or fibrous ankylosis immobilise the joint, more frequently affects the elderly. Multiple osteo-arthritis sometimes attacks children, and in them is often associated with enlargement of the spleen and lymphatic glands, anæmia, and fever (*Still's disease*).

**Diagnosis.**—Charcot's joint disease is painless, and ataxic symptoms are present. In chronic gout tophi are present, uric acid is in excess, and there is a history of acute attacks. Acute rheumatism responds to salicylates, acute rheumatoid arthritis does not.

**Treatment.**—No treatment can cure this disease, except in its earliest stage, but it is often arrested by careful attention to the general health. Liberal diet, fresh air, and gentle exercise, avoiding pain, and a dry warm climate are beneficial. Of drugs, cod-liver oil, iron, and arsenic are chiefly used. Garrod recommends iodide of iron, and Luff potassium iodide or carbonate of guaiacol gr. v-x (grm. 0.3-0.6) thrice daily. Massage is often of great service. At first light general massage only should be employed, but after a few days frictions may be made over the affected joints. Passive movement is also useful, and the thermo-cautery helps to relieve pain and lessen thickening. Any local infection which may cause a toxæmia should be treated, and of late vaccines made from the organisms found in such local conditions as pyorrhœa alveolaris, leucorrhœa, and colitis have been used with considerable success. Even in the absence of such conditions a polyvalent streptococcus vaccine has sometimes given good results, and the recently introduced phylacogens would seem to have done good.

## DIABETES MELLITUS

A nutritional disorder characterised by an excess of sugar in the blood and its excretion in the urine, and attended by polyuria and progressive emaciation. To constitute diabetes the sugar must be *permanently* present. A temporary glycosuria is not diabetes.



**Etiology.**—The disease most frequently begins between the ages of forty and sixty, but is not uncommon throughout adult life, and may affect the aged. It is least common but most fatal in childhood. It attacks men oftener than women, the rich oftener than the poor, and of the various races the Jews are specially liable. Heredity is sometimes a factor. Those of a nervous temperament are prone to it, and worry, nervous shock, and injury or disease of the cord or brain sometimes precede the onset. Obesity is often associated with it, and it occasionally follows the infectious fevers. Gout may also predispose to it.

**Morbid Anatomy.**—1. *The nervous system* shows no constant changes. In some cases, tumours or degenerative changes in the floor of the fourth ventricle, in the medulla, or at the base of the brain, and sclerosis of sympathetic ganglia have been found.

2. *The pancreas* is sometimes atrophied and fibrosed, and on section may show extensive areas of fatty degeneration, and fat necrosis. The islands of Langerhans, which furnish the internal secretion of the pancreas, have been found in a state of hyaline degeneration.

3. *The liver* is sometimes enlarged, sclerosed, and peculiarly pigmented (*diabetic cirrhosis*); and it may be fatty. The changes are not constant.

4. In *the kidneys* there are often hyaline degeneration of the tubular epithelium, and some degree of interstitial nephritis.

5. *The blood* shows—

- (1) The presence of an excess of sugar (from two to four times the normal amount) in the plasma and corpuscles.
- (2) In some cases a reduction of its alkalinity: oxy-butyric acid has been found in it.
- (3) In some instances a black and tarry appearance; in others the presence of fat (*lipæmia*), which may form a creamy layer on standing. In many cases the appearance of the blood is normal, and the blood-count shows no anæmia. Lipæmia is most common where there is acidosis.

*The urine* is pale in colour, clear, and acid in reaction. Both quantity and specific gravity are increased, the former to the



extent of ten or even twenty pints per diem, the latter to between 1025 and 1045 or more. The quantity of sugar ranges from *two to forty grains per oz.* ( $\frac{1}{2}$ –10%), or as much as twenty ozs. (grm. 580) and upwards daily. Occasionally, when the amount of sugar is small, the specific gravity may be low. Uric acid is not increased, but both urea and phosphates are. Albuminuria is often present in the late stages. Acetone, diacetic acid, and  $\beta$ -oxybutyric acid are to be found in most of the severe cases. These acetone bodies are variously supposed to be formed from the breaking down of the tissue albumins, or from defective oxidation of the fats. Acetone and diacetic acid are derivative products of  $\beta$ -oxybutyric acid, to the presence of which in large quantities in the blood diabetic coma is ultimately due.

**Pathology.**—The theoretic interpretation of diabetes varies with the view which may happen to be held of the glycogenic function of the liver. Normally about 0.1 per cent. of sugar is present in the blood, and a minute trace, which the clinical tests cannot detect, in the urine. When the amount in the blood is more than 0.2 per cent. an appreciable glycosuria occurs, and even in health a temporary glycosuria may be produced by an ingestion of sugar beyond the physiological limit. The appearance of sugar in the urine is thus due to an excess of sugar in the blood. The usual view of the glycogenic function is that the liver converts the carbohydrates brought to it from the intestines into glycogen, which it stores up as a reserve, and gradually reconverts into sugar and delivers *via* the blood to the tissues according to their needs. On this view the excess of sugar in the blood is due either to excessive production of sugar in the liver, or to diminished oxidation by the tissues. On the other hand, Pavy held that part of the ingested carbohydrates is converted by the intestinal villi into fat, and another part is synthetically built up into proteids, in which forms it reaches the blood, and that only a portion reaches the liver, where it is stored up as glycogen, and prevented from entering the general circulation except in synthetic combination with proteid bodies. A temporary glycosuria would therefore be due to a defect in the sugar-transforming mechanism, and diabetes to its arrest, permitting the passage of unaltered glucose.

In mild forms of the disease, a diet free from carbohydrates stops the glycosuria, the excess of sugar being thus derived from the carbohydrates of the food; but in severer cases, glycosuria persists though carbohydrates are withheld, and sometimes even though no food is taken. In these instances, sugar is formed by



disintegration of the proteids of the food, and in the gravest cases by disintegration of the body proteids.

It remains to explain why the excess of sugar is not utilised by the tissues—is not, that is, oxidised in the ordinary manner by the muscles. There is no uniformity of opinion on this subject. Recent work upon the internal secretions of the ductless glands has shown that they exert an important influence upon the glycogenic function of the liver, and hence on carbohydrate metabolism, the suprarenal bodies in the direction of increasing, and the pancreas in that of limiting, the consumption of sugar. The pancreas is in turn controlled by the thyroid gland and parathyroids, and by the pituitary body; and the suprarenals by the central nervous system. In diabetes the quantity of sugar in the blood is increased, in the view of von Noorden's school, beyond the power of the tissues to deal with it, either by an increase in the augmenting power of the suprarenals, or by a deficiency in the inhibiting power of the pancreas. Glycosuria due to the "diabetic puncture" of Claude Bernard (puncture of the floor of the fourth ventricle), for example, is the result of central stimulation of the suprarenals and the chromaffin system generally. On the other hand, there is little real evidence for the over-production on which von Noorden lays stress. Other workers maintain that in severe diabetes there is a failure to store sugar, and the failure to utilise the excess thus present in the blood, since there is nothing to indicate diminished oxidation, may be accounted for by the absence of some substance in the pancreatic secretion which prepares the sugar for cleavage or acts as an amboceptor. Extensive disease of the pancreas would destroy this substance, and render the sugar unavailable for combustion in the muscles. There is little to show that the substance is more the property of the islands of Langerhans than of the pancreatic acini.

It must be added that, in some cases, no disease of the pancreas or liver has been found. In some of these there has been disease in the region of the "diabetic puncture" (floor of the fourth ventricle), or in that of the optic chiasma (pituitary body).

**Symptoms.**—*Two* forms of diabetes exist—viz., the acute and the chronic forms. Those attacked by the acute form are usually comparatively young, the symptoms assume a grave type very rapidly, and *post mortem* the pancreas is frequently found to be extensively diseased. In the chronic form, on the other hand, the symptoms are often obscure, and but for the peculiar char-



acters of the urine and attacks of dyspepsia, the patient may for a long time maintain a fair standard of health.

Taking a typical case of diabetes, the characteristic symptoms are, the passage of large quantities of pale urine, great thirst, voracious appetite, progressive muscular weakness, a dry, parchment-like sallow skin, emaciation, and the development of some of the complications tabulated below. Caries of the teeth is common, and males often become impotent. In the later stages the pulse becomes very feeble, severe attacks of diabetic dyspnœa, or "air-hunger," add to the general misery, the breath gives out a peculiar, sweetish, apple-like odour, due to acetone, and the sudden onset of coma frequently ends the scene. Often, however, the patient is carried off by a critical diarrhœa, pneumonia, etc.

### Complications.—

1. *Cutaneous Lesions*.—Boils, carbuncle, and in women eczema or pruritus of the vulva, accompanied by intolerable itching.

2. *Visual Changes*.—Retinitis, soft cataract, and optic atrophy. Retinitis and atrophy are occasional complications; cataract is common.

3. *Nervous Changes*.—The knee-jerk is often absent, especially in severe cases. This is due to peripheral neuritis, which is further manifested by cramps in the legs, tingling, numbness, or neuralgic pains. There is loss of sexual desire. The nervous changes may lead to trophic alterations such as—

4. Gangrene, perforating ulcer of the foot, brittleness of the nails, etc.

5. *Renal Changes*.—Albuminuria, at first without Bright's disease, later with more or less cirrhosis of the kidney and symptoms arising from such a condition.

6. Tubercular affections of the lungs are very common.

*Diabetic coma* may arise suddenly, or may be preceded by headache, restlessness, and a feeble, rapid pulse. The amount of urine and the percentage of sugar often fall before its onset. It is favoured by excitement or fatigue. The temperature is subnormal, there is drowsiness, and the respirations are prolonged and sighing ("air-hunger"). There is an odour like that of apples in the breath and urine. The urine may contain acetone, diacetic acid, and  $\beta$ -oxybutyric acid. The drowsiness deepens to complete coma, and ends in death, usually without convulsions.

Carbohydrate deprivation, whether as a consequence of failure of the tissues to use the excess of sugar in the blood, or of a severe diabetic diet, leads to an incomplete catabolism of the



higher fatty acids and of the amino-acids. These bodies break down in such circumstances into diacetic acid and  $\beta$ -oxybutyric acid, the transformation of diacetic acid into acetone taking place after the diacetic acid has left the blood. In severe diabetes they accumulate in the blood to an enormous extent, and cause an "acidosis," to which the coma is due. It may be explained either by a specific toxic action of the acids themselves, or by the combination of the soda and potash bases with the  $\beta$ -oxybutyric acid, which thus prevents their union with carbonic acid and leaves it to circulate in poisonous quantities in the blood.

In addition to the above common complications, there are often grave mental changes. *Diabetic tabes*, a peculiar form of high-stepping ataxia, is due to a neuritis of the lower limbs. It should not be forgotten that in diabetes diarrhoea may be easily provoked, and death from this cause may follow the administration of a simple purge.

*The diagnosis* depends upon careful and repeated testing of the urine (for tests, see Examination of Urine). Remember the wasting of muscle may be concealed by obesity.

**Prognosis.**—In confirmed cases the outlook is bad. In the more chronic varieties the disease may be arrested for a considerable time. Some patients have had diabetes for twenty years, and cases occurring in later life are apt to run a chronic course. The younger the patient the more likely and the more rapid is a fatal issue.

**Treatment.**—The general health of the diabetic must be carefully watched. He should lead a quiet life, avoiding worry, and take daily gentle exercise. He should bathe daily in lukewarm water, and wear flannel underclothing. After the average output of sugar in the twenty-four hours upon ordinary diet has been ascertained, carbohydrates should be gradually removed from the dietary until the urine is free from sugar, or until a non-carbohydrate diet is attained. The weight should be noted once a week. When the excretion of sugar has ceased for some time, carbohydrates may again be very carefully added, any reappearance of sugar causing a return to the strict regimen. A diet too exclusively nitrogenous is apt to throw an excessive strain upon the liver and kidneys, and to increase the risk of acidosis; and in practice many patients will be found to do better on a moderate amount of carbohydrates than on none at all. The general principle is *to restrict the amount of carbohydrates*



*within the limits of the patient's tolerance*, and each case must be studied separately. Sir William Roberts's dietary is appended as a guide.

### ALLOW

Butcher's meat, poultry, game and fish. Cheese, eggs, butter, fat and oil. Broths, animal soups, and jellies made without sugar. Cabbage, endive, spinach, broccoli, lettuce, spring-onions, watercress, celery. Dry sherry, claret, brandy, and whisky. Tea, coffee (without sugar), soda-water, bitartrate of potash water.

### FORBID

All saccharine and farinaceous food, *bread*, potatoes, rice, tapioca, sago, arrowroot, macaroni; turnips, carrots, parsnips, beans and peas.

Liver contains much sugar-forming substance, and is therefore forbidden. So also are oysters, cockles, and mussels, which contain enormous livers.

All sweet fruits. All sweet wines.

Milk, if allowed at all, should be given very sparingly.

It will be seen from the above table that there is a pretty extensive range or choice of food, but unfortunately the two great drawbacks are the non-allowance of bread and potatoes. The substitutes for bread are either nauseous or too expensive for general use, and some of them are not free from carbohydrates. The principal substitutes are almond biscuits and gluten or bran bread.

**Medicinal Treatment.**—Codeine and morphine are the most beneficial drugs. Codeine may be given in doses of gr.  $\frac{1}{2}$  (grm. 0.03) thrice daily, increasing if necessary up to gr. 2 or gr. 3 (grm. 0.13 to 0.2). Antipyrin gr. x (grm. 0.6) thrice daily is sometimes useful in neurotic cases. Arsenic and strychnine are serviceable tonics, and saccharin or glycerin may be used for sweetening purposes. Massage, electricity, cod-liver oil, etc., all are useful in various cases. The great thirst may be relieved by lemon-juice or citrate of potassium freely diluted. If diacetic acid is present, the dietary must be made less rigid, bicarbonate of soda may be given in large doses (up to  $\bar{3}$ i ss. (grm. 45.0) in twenty-four hours), and in coma it or the carbonate should be given intravenously. Naunyn recommends the carbonate (grm. 35–40 to the litre of normal salt solution). This must not be



given subcutaneously, lest it cause gangrene, but intravenously it is apparently well borne. In less severe cases, alkalies may be given by the mouth or rectum, but however they are administered, the improvement is almost always only temporary.

## DIABETES INSIPIDUS

A rare condition characterised by the passage of an excessive quantity of pale limpid urine, free from sugar or albumin; and accompanied by insatiable thirst.

**Etiology.**—The disease is commonest in the young, and attacks males oftener than females. It may be hereditary. Bernard produced polyuria by puncturing the fourth ventricle higher than the centre for producing saccharine urine; and it has been suggested that diabetes insipidus is due to some disturbance of this centre. Extract of the infundibular lobe of the pituitary body has a profound diuretic influence, and in many cases of diabetes insipidus there has been evidence of a lesion of the base of the brain, *e.g.* gummatous meningitis of the middle cerebral fossa. The condition is probably due to a vasomotor paralysis of the renal blood-vessels, produced either by local causes in the abdomen, or by central disturbance, particularly of the posterior lobe of the pituitary body. It has sometimes been produced by severe mental shock. The temporary polyuria after a hysterical fit is well known.

Cases may be *primary* or *idiopathic*, when there is no organic cause; or *secondary* to disease in the brain or elsewhere.

**Morbid Anatomy.**—Various conditions have been found, of which the most frequent are the natural results of polyuria—hypertrophy of the bladder and dilatation of the ureters and renal pelvis. None are constant.

**Symptoms.**—Polyuria and the consequent thirst are the chief symptoms. Primary optic atrophy is sometimes present. Only the watery constituents of the urine are increased, and as much as fifteen to forty pints of this pale urine may be voided in the twenty-four hours. The specific gravity is very low, from 1008 down even to 1001. Dyspepsia, mental irritability, and muscular weakness are frequently prominent. Though the disease is rarely fatal, the persistent thirst and frequent micturition prevent sound sleep, and may undermine the general health.



**Diagnosis.**—Care must be taken not to mistake this condition for the polyuria of Bright's disease or diabetes mellitus. Note the absence of casts, and the low sp. gr. Hysterical polyuria is transitory, not permanent.

**Treatment.**—Valerianates, iron, strychnine, galvanism, and the bromides have all been used. The valerianates must be given in large doses. Specific treatment should be tried when there is evidence of syphilis. It is wrong to try to limit the amount of fluid swallowed. There is no evidence that any drug materially affects the course of the disease.



# THE INTOXICATIONS

## ALCOHOLISM

ALCOHOLISM may exist in two forms, acute and chronic. Of acute alcoholism little need here be said. The diagnosis of ordinary intoxication is obvious; but when a large overdose has been taken, the victim is frequently found comatose, and it becomes necessary to decide between alcoholism, organic disease, and opium poisoning. The diagnosis is discussed under Uræmia (*see* p. 392), but it must be emphasized here that the smell of the breath is an entirely fallacious indication, and that as a mistake may lead to the patient's being left alone in a police-cell to "sleep it off," its consequences may be fatal.

Of **chronic alcoholism** the predisposing causes are heredity, occupation, and "good company." Hereditary influence is often strongly marked, but it is apt to be confused with parental example; and there is no more false or disastrous idea for a young alcoholic to entertain than that, because an ancestor has suffered, it is useless for him to fight. The influence of occupation is shown in the prevalence of the disease in publicans, barmen, draymen, and those whose trade exposes them much to heat (puddlers, furnace-men, riveters, etc.).

The *morbid changes* are numerous. The digestive tract suffers early, chronic œsophageal, gastric, or gastro-intestinal catarrh being frequent. The liver is often fatty; cirrhosis is most common in spirit-drinkers. Arterio-sclerosis is very constant, and dilatation of the heart is frequent. The interstitial nephritis (granular kidney) which is often present, is probably secondary to the arterio-sclerosis, and not a direct effect of the alcohol. The nervous system is especially liable to suffer, peripheral neuritis being the most constant manifestation, while pachymeningitis is not uncommon, and myelitis may occur.

The *symptoms* generally begin with digestive disturbances. Nausea, or a feeling of sinking in the morning, is followed by



morning vomiting. The tongue is furred and tremulous, appetite fails, and the bowels, at first constipated, may afterwards become loose. As time goes on, the hands become tremulous, muscular power is materially diminished, the gait may be ataxic, and the knee-jerks are lost (Peripheral Neuritis, which see). Albuminuria is present if the kidneys are diseased; and glycosuria may occur. Insomnia or disturbed sleep is common; the memory fails; the moral sense becomes defective, and lying is a constant characteristic. Hallucinations of sight or hearing may occur, and some cases end in dementia. Others terminate in cirrhosis of the liver or kidney, in cardiac failure, or in meningitis. The course of the disease may be broken by attacks of—

*Delirium tremens.* Very rare as the result of a single indulgence, this condition is frequent in habitual drinkers. It may follow upon injury, shock, or exposure, or may develop in the course of acute disease. It is to be distinguished from *mania a potu*, a transitory state of acute and often homicidal insanity which occasionally replaces ordinary intoxication in those of nervous heredity. The onset of delirium tremens is accompanied by irritability, restlessness, and disturbed sleep. Tremor is marked. The patient talks to himself, or answers imaginary voices. In a day or two visual hallucinations of moving animals (rats, snakes, or insects) appear, from which he tries to escape, often by springing out of bed; noisy delirium is present; perspiration is abundant; the temperature is somewhat elevated, and the pulse rapid and soft; and there is complete insomnia. Sleep returns as a rule about the third day, but in severe cases it may not do so till the fifth. In fatal cases, death may be due to exhaustion, or the patient may die comatose or convulsed.

**Treatment.**—In profound alcoholic coma the stomach should be washed out. Hot coffee may be given by the stomach-tube and alternate hot and cold applications should be made to the skin. If the diagnosis is doubtful after emptying the stomach, the case should be watched for further indications. In *mania a potu* apomorphine hypodermically produces vomiting and removes both cause and symptoms. *Delirium tremens* being due to a toxæmia, the first object of treatment is rapid elimination of the poison. Alcohol must be stopped at once, the popular idea that it is required to prevent collapse being altogether fallacious. Treatment should begin with a brisk mercurial purge. Many authorities advise sedatives and narcotics, and push them from the outset. But they do not act unless in large and repeated



doses, and these may have a dangerous cumulative effect. Sleep usually returns about the third day; if it does not, sedatives may then be carefully used. Chloral is safer than morphine, and hyoscine, gr.  $\frac{1}{150}$ – $\frac{1}{100}$  (grm. 0.0004–0.0006), is often effectual. Norman Kerr almost entirely dispensed with hypnotics, and got better results from promoting diaphoresis by Liq. Ammon. Acet., combined if necessary with an emetic dose of ipecacuan. The diet must be fluid and bland, and vigilant nursing is essential. The strait-jacket should be used only when absolutely necessary.

The treatment of *chronic alcoholism* is moral as much as medicinal. Complete abstinence must be practised from the outset and "tapering-off" forbidden. To effect this an institution may be necessary. Attention should be first directed to the restoration of the digestive system, and only after this is effected should nervine tonics be employed. The best are nux vomica and strychnine. The number of so-called specific drugs is legion, and their uselessness is as remarkable as their number. Much is often to be accomplished by persevering non-hypnotic suggestion in the building up of the patient's shattered will.

## MORPHINISM AND COCAINISM

The habitual use of opium or morphia leads to a tolerance of the drug, which often results in its employment in increasing quantities. As much as gr. 60 (grm. 4.0) of morphine hydrochlorate have been injected daily,<sup>1</sup> and over a pint of laudanum has been regularly taken. The practice is common among brain-workers, physicians and literary men being specially addicted to it. The habit is usually begun for the relief of pain. In those accustomed to morphia the ordinary narcotic effect is replaced by a sense of euphoria and exaltation, with quickening of the intellectual processes; but this is succeeded by profound depression as the effect passes off. The main symptoms are a sallow, cachectic appearance, a hard and wrinkled skin, and emaciation; variable appetite, occasional colic, alternating constipation and diarrhoea; restlessness, exaggerated sensibility, disturbed sleep; palpitation or dyspnoea; indolence, irresolution, and untruthfulness. The reflexes are at first increased, later abolished; the pupils, contracted just after a dose, are dilated in the intervals. Death may be due to progressive emaciation, to intercurrent disease, or to an accidental or intentional overdose.

<sup>1</sup> Norman Kerr.



*Cocainism*, a less frequent but yet prevalent habit, is also practised among physicians, and by those who, having found pleasure in the medicinal use of nasal or laryngeal sprays containing the drug, continue it after the need is passed. Emaciation, mental disturbance, and moral perversion develop more rapidly than with morphia; and a sensation of grains of sand or small shot under the skin is frequently complained of. If unchecked, cocainism leads to melancholia or mania.

The *treatment* of both conditions is on similar lines. There is no hope but in complete withdrawal of the drug, and treatment in an institution is essential. In morphinism sudden withdrawal is attended by agonising suffering; the reduction must be gradual, and should be spread over a period of several weeks. Narcotics may occasionally be necessary for sleep or pain, but care must be taken lest one habit be merely replaced by another. Warm baths are useful in allaying restlessness, cardiac tonics to support the heart, and the gastric condition must receive attention. Alcohol must be resolutely avoided, and the cured morphinomaniac should be a total abstainer from both opium and alcohol. The process of cure may occupy from three months to a year or more.

### LEAD-POISONING (Plumbism or Saturnism)

There are three ways in which lead may enter the system: by deglutition, inhalation, and absorption through the skin. Poisoning may therefore result from the use of water contaminated by passage through lead pipes, from food stuffs coloured with substances containing lead, or from tinned foods, particularly fruits, the acid of which may dissolve out lead from the solder. It is also common in occupations involving the inhalation of lead fumes (smelters), or dust (compositors, white-lead workers, painters, potters), and in workers in lead who eat with unwashed hands. Lead may be absorbed through the skin when perspiring, and poisoning has thus followed the use of cosmetics or hair-dyes containing it. All ages are liable, and both sexes, but the female is specially susceptible. Lead is eliminated principally by the bowels and kidneys, and to a less extent by the skin.

**Morbid Anatomy.**—1. *Nervous system.* Peripheral neuritis is very frequent, and most commonly affects the musculo-spiral nerve; but sometimes the peroneal is principally involved. There may be secondary changes in the cells of the anterior cornua. Neuro-retinitis



or optic atrophy may occur. Meningo-encephalitis is sometimes present.

2. *The kidneys*.—The glomerular and tubular epithelium is first affected, and interstitial nephritis develops later.

3. *The circulatory system*.—Arterio-sclerosis is almost constant. It leads to cardiac hypertrophy and ultimately to dilatation. Cerebral hæmorrhage may follow.

4. *The blood*.—Anæmia is constantly present, the red corpuscles and hæmoglobin being diminished in the same proportion. The red cells show a basophil granulation (*punctate basophilia*), and nucleated cells are common; the leucocytes are not altered.

**Symptoms**.—Anorexia and constipation, with a metallic taste in the mouth on rising, and a tendency to headache are among the early symptoms. The face becomes increasingly pale and sallow (*saturnine cachexia*); and at this stage a *blue line*, due to the formation of sulphide of lead in the papillæ, is seen at the margins of the gums. It is produced by the sulphuretted hydrogen evolved from the tartar of the teeth, and is absent where teeth have been extracted. The patient is next attacked by severe abdominal pain (*lead colic*), often of sudden onset, and centred about the umbilicus. It may be of two kinds, acute, superficial, accompanied by tenderness, and more severe on one side than the other; or deep-seated and dull. The former is paroxysmal, the latter constant. The abdomen is retracted, the pulse slow, small, of high tension, and sometimes unequal on the two sides, there is usually constipation, and often vomiting. The colic is probably due to irregular spasm of the small intestine. The attack usually passes off in about three days, but may be frequently repeated.

*Lead palsy* is most frequent in the form of bilateral *wrist-drop*. The extensor muscles of the fingers, hands, and wrists are affected, and the hands hang flabbily by the sides. These muscles are supplied by the musculo-spiral nerve; but the supinator longus and the extensor metacarpi pollicis, which have the same nerve-supply, usually escape. Less frequent forms of paralysis are *ankle-drop*, when the muscles supplied by the peroneal nerve are affected; paralysis of the muscles of the upper arm, including the supinator longus; and paralysis of the intrinsic muscles of the hands (*Aran-Duchenne type*). In all these forms muscular



atrophy is rapid, and the reaction of degeneration (*see* p. 424) is present. Pain is slight or absent.

*Saturnine encephalopathy* is fortunately less common, and is most frequent in women. Severe headache is followed by convulsions and coma, and death may take place on the second or third day. In cases that recover from the coma, temporary or permanent blindness may follow. A "toxic hysteria" (Oliver) may precede the onset of the coma, and is apt to mislead the diagnosis. The condition may arise without warning, neither colic nor wrist-drop having preceded it.

In pregnant women abortion or still-birth is the rule, and of the children born alive, most die in early infancy. Hence lead is often used as an ecboic.

The condition of the *kidneys* may result in uræmia. Both in gout and in lead-poisoning there is retention of uric acid, and gout is apt to occur in those lead-workers who are addicted to beer.

**Treatment.**—Mild cases of lead colic require rest in bed, warm applications, and an aperient, or if there is vomiting, an enema. Severe pain may be relieved by a warm bath, but sometimes morphia is necessary. For elimination of the lead a mixture containing magnesium sulphate and a small dose of potassium iodide, gr. iii–v (grm. 0·2–0·3), should be given thrice daily. Large doses of the iodide are apt to liberate too much lead in a soluble form. The paralysed limbs should be kept at rest on splints, and treated by electricity; later, massage and strychnine are useful. The anæmia must be treated by iron.

In saturnine encephalopathy nitrite of amyl may be inhaled during the convulsions. If the urine is scanty, pilocarpine should be given, and lumbar puncture may relieve intracranial pressure.

## ARSENICAL POISONING.

Chronic arsenical poisoning occasionally occurs in workers in arsenic or its salts, in those trades in which arsenical colours are employed, and as a sequel to the prolonged medicinal use of the drug. Until recently wall-papers and artificial flowers were often coloured by arsenic. Beer has been contaminated with it through the use of adulterated glucose; and it is used, sometimes to a dangerous extent, in the curing of furs.

**Symptoms.**—Workers in arsenical pigments are liable to painful ulcers of the hands or scrotum, less frequently to general poisoning. Inhalation of arsenical dust or vapour, or long-continued administration of the drug leads to redness of the



conjunctivæ and eyelids, huskiness of the voice, and digestive disturbances. Erythema of the palms and soles is frequent, and desquamation may follow; the limbs or trunk are liable to various eruptions, particularly herpes; and a brownish discoloration of the skin (*arsenical pigmentation*) is often seen. The most serious result is peripheral neuritis. It differs from that of lead in affecting the legs rather than the arms, and in being accompanied by marked sensory symptoms, viz., numbness, tingling, and acute tenderness of the muscles. Along with these there are loss of power in the legs and a high-stepping or ataxic gait; and in severe cases the arms are also affected. Recovery is slow.

**Treatment.**—Once its ingestion ceases, arsenic is rapidly eliminated, and treatment is therefore symptomatic. Alcohol is obviously to be forbidden in any form of neuritis; rest in bed is usually required, and when the sensory symptoms have abated, electricity and massage. Strychnine or nux vomica is indicated later on.

## FOOD-POISONING

Apart from the poisons accidentally or deliberately conveyed in it, meat may give rise to poisoning in one of three ways. It may be in a state of decomposition when eaten (*ptomaine poisoning*); organisms which gain access to it after slaughter may produce a chemical poison with no evident signs of decomposition, and the food may be swallowed in this state; or the organisms may be swallowed with the food before they have had time to produce their poison, which is then developed in the body. In the two former cases, the symptoms begin very soon after the food has been swallowed, in the last there is a period of incubation. True ptomaine poisoning is the least common of the three cases, for the alkaloidal products of decomposition are seldom abundant enough to cause serious symptoms. The organisms chiefly concerned are two, *bacillus botulinus* and *b. enteritidis*. The former is responsible for the symptoms of sausage poisoning, and sometimes for poisoning due to undercooked meat, but its toxin is destroyed by boiling. The symptoms, arising either at once or after an interval sometimes extending to forty-eight hours, are very similar to those of atropine poisoning. They include dryness of the throat, hoarseness, dysphagia, rapidity of the pulse, and dilatation of the pupils, which do not respond to light. Nausea, vomiting, abdominal pain, diarrhœa, and prostration are also present. Death is not infrequent, and recovery is slow.



The symptoms due to *bacillus enteritidis* and allied organisms are those of acute gastro-enteritis. Vomiting and purging, cramp in the legs, and collapse are the most important. The temperature is at first subnormal, but later it may be raised.

Poisoning may also result from the ingestion of fish or shellfish, or of milk and its products.

The habitual consumption of certain *cereals* may give rise to poisoning. Bread made from rye contaminated with *claviceps purpurea* (the ergot fungus) causes the symptoms of *ergotism*. The chief morbid changes are spasmodic contraction of the arterioles, and sclerosis of the posterior columns of the cord. The early symptoms are those of digestive derangement, and the lesions are expressed in gangrene of the fingers and toes, or in painful spasmodic clenching of the hands, and hyperextension of the feet. Ataxia may also be present.

*Pellagra* (*maidismus*) has been generally attributed to eating diseased maize, but Sambon and, after him, many others regard it as probably protozoal in origin, the unidentified ultra-microscopic organism being probably conveyed either by the sand-fly or the stable-fly (*Stomoxys calcitrans*). Its description is therefore only provisionally retained in this place. The disease is met with in the countries surrounding the Mediterranean Sea, and in the Southern States of America. Of late years, cases have also been recognised in Britain. Digestive disturbances and debility are followed by an erythematous eruption, recurring every spring, affecting the parts exposed to light, and causing very troublesome itching. It lasts about a fortnight, ends in desquamation, and leaves the skin roughened, scaly, and pigmented. After a few attacks mental changes occur, which may end in melancholia or dementia. There is great emaciation. The reflexes are exaggerated, and the gait is spastic. Sclerosis of the lateral and posterior columns of the cord has been found *post mortem*.

*Lathyrism* is due to eating food made from the seeds of the vetch. The symptoms are those of spastic paraplegia.

The first step in the **treatment** of poisoning by animal food is elimination of the poison by evacuation of the bowels and stomach. Afterwards stimulants, morphia, and external warmth are necessary. In grain poisoning an emetic may sometimes be needed, but for the most part the treatment is symptomatic.



SUNSTROKE<sup>1</sup>

Exposure to the direct rays of the sun, or to excessive heat, may produce one of three conditions, grouped under the above title. They are heat-exhaustion, sunstroke proper, and thermic fever. Fatigue, employment in a confined space, a moist atmosphere, ill-health, and alcoholic indulgence are predisposing causes. The excessive external heat is supposed to cause a break-down of the heat-regulating mechanism in the medulla, in consequence of which the internal temperature rises, and coagulation of the myosin results. Sambon, however, holds that thermic fever is due to a specific organism which can develop outside the body only at high temperatures.

Extreme cerebral and visceral congestion is the chief morbid change. *Rigor mortis* is rapid. When death is sudden, there may be no characteristic lesions.

The *syncopal form*, or heat-exhaustion, may arise as the consequence of fatigue or depression during exposure to heat. Faintness or syncope, a cold and damp skin, and a rapid and feeble pulse are the chief symptoms. Death is due to heart failure, but most cases recover.

*Sunstroke proper* (the *asphyxial form*) is due to exposure of the head and spine to the direct rays of the sun in a heated atmosphere. The rays therefore act immediately upon the brain and the cardiac respiratory centres. The onset is generally sudden. Unconsciousness, pallor, feeble pulse, and rapid failure of the heart and respiration are the symptoms. Death is common, and recovery often partial, the brain being permanently injured.

*Thermic fever* (the *hyperpyrexial form*, *siriasis*) arises either by night or by day from exposure either to artificial or solar heat. There are often premonitory symptoms, malaise, diarrhoea, nausea, giddiness, or sleeplessness, after which the temperature rises abruptly to 108° F., 110° F., or even higher. Lividity of the face, dyspnoea, and delirium are rapidly followed by coma and death in a few hours. Recovery is slow and incomplete.

**Treatment.**—In the syncopal form removal to a cool place, loosening the clothing, and stimulation are required. In the asphyxial form cold douching to the head and body is the

<sup>1</sup> Sunstroke is not an intoxication, but is here described for convenience sake. It should properly form one of a group of Diseases due to the Effects of Heat and Cold.



principal remedy; in thermic fever, cold douching or the cold bath, the temperature of which is to be gradually lowered. If unconsciousness continues after the bath, a blister to the scalp may be of use; but such cases are grave. If malaria is suspected, quinine should be given hypodermically. Bleeding must not be practised.

Patients who have recovered from sunstroke or thermic fever should leave the tropics, and should not return.



# DISEASES OF THE ALIMENTARY TRACT

## I. THE MOUTH

THE mouth is liable to a number of diseases, chiefly inflammatory, which may be local or symptomatic of general disease. The local diseases are mainly due to organisms ingested or inhaled, but may also be produced by the coughing up of infected sputum, as in phthisis. Pathogenic organisms may be present even in health, and require a suitably modified soil before they can cause disease. The most frequent are the *Staphylococcus pyogenes aureus* and *albus*, the *Streptococcus pyogenes*, the *Diplococcus pneumoniae*, and the *Micrococcus tetragenus*. Fungi, such as the *oidium albicans*, or *monilia candida*, are also found.

## STOMATITIS

An inflamed condition of the mouth is common during dentition, the infective fevers, and morbid states of the blood. It may be also caused by mechanical irritation, by the internal use of mercury, or by the presence of carious teeth.

Such a condition, however caused, is likely to be attended with the following symptoms :—

1. *Pain and difficulty* in mastication, articulation, and deglutition, in proportion to the severity of the disease.
2. Increased salivation.
3. Fœtor of breath.
4. Constitutional disturbances.

Like all inflammations of mucous membranes, the condition varies much in severity, and accordingly the following types are described :—

**Simple** or *catarrhal* stomatitis, a mild form which readily yields to the local application of glycerin of borax. In acute cases the surface is very red and raw, in milder cases irregular white patches show increased production of epithelium.



**Aphthous** or *vesicular* stomatitis is characterised by the presence of small pseudo-vesicles upon the mucosa of the lips, tongue, and cheeks. The appearance is due to the presence of a fibrinous exudation beneath the epithelium, which after a time is shed, leaving behind small greyish ulcers. Treatment of the constitutional symptoms and the use of a mouth-wash lead to rapid recovery.

**Ulcerative stomatitis**, or putrid sore mouth, occurs in weakly and ill-nourished children, and sometimes in adults. It may be epidemic. The gums become swollen and ulcerated at their margins. The ulcers are covered with a greyish membrane, and in bad cases may extend deeply, loosening the teeth, and exposing the alveolus. The submaxillary glands are enlarged, and constitutional symptoms are often severe. True ulcerative stomatitis occurs most frequently *after* the first dentition.

*Treatment.*—Chlorate of potash should be given internally, gr. x (grm. 0·6) thrice daily for a child, gr. xx (grm. 1·2) for an adult, and also used as a mouth-wash. The ulcers may be touched with nitrate of silver.

**Parasitic stomatitis** (*thrush*) is a *specific* disease dependent upon a fungus termed the *saccharomyces* or *oidium albicans*; it is frequently met with in weakly children and infants.

The fungus is probably identical with the *Mycoderma vini*, and is therefore a mould. It causes the formation of milk-white or greyish adherent patches on the mucous membranes of the mouth and pharynx of the child attacked.

Microscopically, these patches consist of epithelium united into a membrane by twisted filaments (the fungus), which are often branched and composed of long cells joined end to end, and constricted at the joints. Both hyphæ and spores are to be seen.

The patches may extend, though rarely, to the œsophagus, stomach, and cæcum.

*Symptoms.*—The mouth is *dry*, and tender or painful; there is usually much debility and gastric disturbance. Small white roundish patches form, and may coalesce, producing larger areas. The patches, at first adherent, are later easily detached, leaving behind them little ulcerations.

*Treatment.*—Absolute cleanliness of the feeding bottles, the avoidance of stale milk, and the frequent swabbing out of the mouth with a solution of boracic acid or permanganate of



potash, are the principal indications for successful treatment. Everything must be done to improve the debilitated condition generally.

**Gangrenous stomatitis** (*cancrum oris* or *noma*) is sometimes seen in debilitated children between the ages of two and five years. It follows upon defective nutrition and bad hygiene, and may be a sequela of the acute infections of childhood, especially measles. It is very probably of spirillary origin.

**Symptoms.**—At first there appears a small diphtheroid patch of necrosed tissue with a general inflammation around it. This inflammatory zone extends and becomes brawny in character, the slough separates, and the ulceration goes on until the cheek is perforated. The disease frequently kills the patient before necrosis is well marked, but sometimes the gangrene extends to the jaw, malar bones, tongue, etc., before a fatal termination is brought about. The constitutional disturbance is very great, although fever is moderate, and usually the typical typhoid state rapidly ushers in a fatal ending.

**Treatment** is unsatisfactory. Complete excision of the diseased area by the knife, with the administration of quinine and stimulants, are the best measures. To parts which cannot be excised, nitric acid or pure phenol should be freely applied. Should the child recover, a plastic operation will be necessary later on.

## ACUTE TONSILLITIS

Inflammation of the tonsils is not only a common complication or symptom of many fevers, but is also a common primary affection. It may affect chiefly the follicles (*follicular tonsillitis*), or the whole stroma may be involved (*parenchymatous tonsillitis*). When in parenchymatous tonsillitis the inflammatory process goes on to suppuration (as it frequently does), it is popularly termed *quinsy*.

**Etiology.**—Acute tonsillitis is most frequent in young people, but may occur at any age. It is often associated with, or precedes, acute rheumatism. Primary tonsillitis is a direct infection. In either case the organisms found are mainly the pus-forming cocci.

**Symptoms.**—The onset is sudden. The throat is hot and dry, and the temperature runs rapidly to 104° F., or more,



accompanied with severe frontal headache. The tongue is foul and the breath foetid. The glands below the jaw are enlarged, and surrounded by diffuse tender swelling. There is a great pain on movement of the jaw or in swallowing. The tonsils are enlarged and congested, as are the fauces and soft palate. Yellowish patches of secretion may protrude from the mouths of the follicles, or thin dirty-yellow exudate may cover the surface of the tonsils. In some cases the exudate may be thicker, patchy, and semi-membranous in appearance, but it differs from diphtheritic membrane in that it strips off readily on brushing with 10 per cent. solution of silver nitrate.

If treatment is effective, the acute symptoms subside in four or five days, and the patient is well in about a fortnight. Sometimes, however, suppuration occurs in the peritonsillar connective tissue, and an abscess forms on one or both sides (*quinsy*). In such a case the fever does not subside, the swelling becomes so great that the mouth can hardly be opened, and fluctuation can be felt by the finger on one or other side. The abscess may rupture spontaneously into the mouth, and the rupture occasionally leads to profuse hæmorrhage.

An *epidemic* form of tonsillitis, with early and pronounced involvement of the cervical glands and cardiac complications, has been observed in England in recent years.

**Diagnosis.**—The difficulty lies chiefly in distinguishing between acute tonsillitis and diphtheria. There may be membrane in the former; it may be absent in mild cases of the latter. It is often impossible to decide on a first examination whether diphtheria is present. Even a film prepared direct from a swab may not show the characteristic bacilli. Cultures made upon blood-serum clinch the diagnosis of diphtheria, but time is lost in this examination, as the colonies require about eighteen hours for their development. It is always safest in doubtful cases to inject antitoxin at once. If the case is afterwards proved not to be diphtheria, no harm is done.

**Treatment.**—Begin by opening the bowels freely with calomel followed by a saline, and keep them freely open. Subdue fever and relieve headache by antipyretics or salicylates. The salicylates, gr. x-xv (grm. 0·6-1·0) every three hours, have also an excellent effect on the general course of the disease. Locally use glycerin of belladonna and hot fomentations externally, and paint the throat before food with a two per cent. solution of cocaine, or in mild cases gargle with Potass. Chlor. gr. lxxx (grm. 5·3), Acid. Hydrochlor. dil.  $\bar{3}$ i ss (cc. 5·3), Glycerini  $\bar{3}$ iv (cc. 15·0), Aq.



ad ̄viii (cc. 230·0). Antiseptic sprays are more useful in acute cases, in which gargling is attended with much pain.

The abscess of quinsy should be opened with a guarded knife. In the *low, subacute* forms, frequently seen in overworked patients, astringent applications, such as iron and glycerin, are useful. Nourishing diet and tonics are required during convalescence.

## CHRONIC TONSILLITIS

This condition may follow repeated attacks of the acute disease, or may be chronic from the outset. It is frequent in children before puberty, and is very often associated with adenoids. Both tonsils are enlarged, and either the lymphoid tissues or the stroma may be principally affected.

**Symptoms.**—In conjunction with post-nasal adenoids, chronic tonsillitis sets up the habit of *mouth-breathing*, at first most noticeable at night, when snoring is common, and the *alæ nasi* are often indrawn during inspiration. A short, dry cough, which may cause small hæmorrhages from the dilated tonsillar vessels, is not uncommon. The expression of a mouth-breather, as he stands with parted lips, is heavy and stupid; and the condition leads in time to changes in the shape of the chest, of which pigeon-breast is the most frequent. There is a great tendency to recurrent bronchitis, which may aid in producing the deformity. The general health suffers, and the child becomes thin and anæmic. Hearing, taste, and smell are often impaired. Enlarged tonsils increase the liability to specific infections, and make the prognosis of diphtheria and scarlet fever less favourable.

**Treatment.**—If the condition does not yield to astringent applications, tonics, and an open-air life, the tonsils should be removed. Adenoid vegetations must be dealt with at the same time. Spontaneous recession of the hypertrophy often occurs at puberty, but it is a mistake to wait for this if the organs are very large.

## II. THE ŒSOPHAGUS

### ŒSOPHAGITIS

Inflammation of the œsophagus may be acute or chronic. The acute form arises (1) from scalds or burns, or the swallowing of corrosive poisons; (2) as a complication of the specific fevers;



(3) towards the end of wasting diseases; (4) in infants, as a purely catarrhal type, often without apparent cause. Ulceration or cicatricial stricture may follow upon acute œsophagitis.

The *symptoms* are, mainly, pain in swallowing, tenderness over the cervical portion, and spitting of mucus.

**Treatment** consists in the administration of bland fluid food. Where corrosive poisons have been swallowed, rectal feeding must be instituted.

CHRONIC ŒSOPHAGITIS may follow the acute affection, may arise above a stricture, or may be the result of chronic alcoholism.

It tends to lead to ulceration and perforation. In alcoholics, morning vomiting, usually of œsophageal mucus, but sometimes mixed with the contents of the stomach, is the principal sign. Where the vomit comes only from the œsophagus, its reaction is alkaline; if the gastric contents are also expelled, the reaction is acid.

## STRICTURE

Simple stricture of the œsophagus may be (1) cicatricial, (2) spasmodic (*œsophagismus*).

CICATRICIAL STRICTURE is usually due to the after effects of burns, scalds, or corrosive poisons; but it may follow upon cicatrization of ulcers of any kind. Narrowing of the œsophagus may also result from external pressure, as in aneurysm or mediastinal tumour.

Obviously, the stricture must vary in its length with the cause that has produced it. In different cases the gullet may be slightly narrowed, or so much that even fluids can scarcely pass.

The **symptoms** depend to some extent on the position of the stricture. Dysphagia is common to all sites. If the stricture is high in the tube, the return of food may be immediate, if low, after a slightly longer interval. Emaciation follows when the narrowing is great.

The position of the stricture, and the degree of stricture, must be determined by the passage of an œsophageal bougie, after all serious intrathoracic diseases have been carefully excluded. Aneurysm, mediastinal tumour, valvular affections of the heart, etc., preclude such an examination.

**Treatment.**—Progressive dilatation, with specific treatment in syphilitic cases; if the stricture is impassable, gastrostomy.

SPASMODIC STRICTURE occurs in neurotic individuals, especially young women. It commences suddenly, usually during a



meal, the food sticking for some time, and then either passing on to the stomach or being returned. It is attended by severe burning pain and retching. There is little emaciation.

On passing the sound, the stricture will be found to occupy various positions at different examinations.

**Treatment.**—Pass a full-sized bougie, twice or thrice weekly. This is often sufficient to cure the condition. At the same time treat any nervous disturbance that may be present.

## TUMOURS

Innocent tumours occasionally occur, but are of small importance. Sarcoma is extremely rare.

**CANCER**, of the type of squamous-celled epithelioma, is common between the ages of forty-five and sixty, and most frequent in the male. The growth is annular, infiltrating all the coats, and ulcerates rapidly from the passage of food over it.

The **symptoms** are increasing dysphagia, regurgitation of food mixed with mucus and blood, and rapid emaciation. The passage of a bougie locates the site of the stricture. This must be carefully done, as the diseased wall is very liable to perforation.

Death occurs from six months to a year after the onset of symptoms. It may be due to starvation, or to complications due to direct extension of the growth (septic broncho-pneumonia, perforation of the aorta, etc.). Metastases are uncommon, except in the thoracic and cervical glands. In the latter situation they may aid the diagnosis; and an X-ray examination after a bismuth meal will reveal the site of the stricture.

**Treatment.**—Where the cervical part is affected, excision of the tumour. In other cases permanent intubation, or if this is not tolerated, gastrostomy.

## III. THE STOMACH

### EXAMINATION OF THE STOMACH CONTENTS

To gain a clear idea of the various disturbances of gastric function, it is necessary to recall the main facts relative to gastric digestion. The action upon the starches of the ptyalin derived from the saliva continues for twenty or thirty minutes, until it is inhibited by acids; and during that time starch is converted into maltose, with an intermediate stage of dextrin. The



direct action of the gastric juice converts proteids into peptones, with an intermediate stage of proteoses (albumoses); and fats are liberated by the dissolution of their proteid cellular envelopes. The caseinogen of milk is at the same time converted into coagulated casein by the action of rennin. The churning movement of the stomach is necessary for the complete mixture of the food and gastric juice; and its propulsive movement sends the elaborated chyme onwards into the duodenum. It is evident that the digestive processes can be retarded by—

1. Deficiency either in quantity or quality of gastric secretion.
2. Deficient movements of the stomach, through muscular atony.

It is therefore often desirable to know the exact state of the secretion, and the amount of motor or peristaltic power.

The *reaction* of the stomach contents varies according to the stage of digestion—

1. Immediately after food is taken it is faintly alkaline from the saliva swallowed.
2. Within the next half hour it becomes slightly acid from lactic acid.
3. After this time it gradually becomes more distinctly acid from the presence of free HCl.

Lactic acid is derived from the food, and is normally present during the first hour or so of digestion: after that time *there should only be a trace*. Its presence in any quantity tends to inhibit the secretion of HCl; consequently, if lactic acid is found in excess two hours after a standard meal, we may infer that HCl is deficient.

When it is desired to ascertain whether gastric digestion is normal, a test meal is first given; then, after a definite interval, a portion of the gastric contents is withdrawn by the stomach tube, filtered, and chemically examined.

Examination by this means is chiefly useful in obscure cases of gastric neurosis and functional disorder where there is no vomiting; also where, in an early stage, carcinoma is suspected, but the symptoms are not diagnostic.

Conditions contra-indicating the passage of the stomach tube are the same as those that contra-indicate sounding the œsophagus, with in addition the presence of acute gastritis, gastric ulcer, or a late stage of carcinoma. Where vomiting is present, examination of the vomited matters may suffice.



The first step is to determine the *total acidity* of the gastric juice by a decinormal solution of NaHO, of which 1 cc. = 0.00365 grm. HCl. 10 cc. of the juice are placed in a porcelain capsule with a few drops of a one per cent. solution of phenolphthalein, and the NaHO is added drop by drop. A pink colour forms, which disappears on shaking, but becomes permanent when neutralisation is complete. The percentage acidity can be calculated from the number of cc. of NaHO employed, but it is usual to express the acidity simply in terms of NaHO. If 6.5 cc. are required for 10 cc. of juice, 65 would be required for 100, and the acidity of the juice is spoken of as 65.

We next determine whether the acid is present as free acid or in combination, by adding to the filtered juice a solution of Congo red. In the presence of free acid a dark-blue colour is produced.

To determine that the free acid is HCl, a series of colour tests is used.

1. Dimethyl-amido-azo-benzol (0.5 per cent. alcoholic solution) gives a cherry-red colour with the unfiltered juice in the presence of free HCl.
2. Günzberg's test (alcoholic solution of phloroglucin and vanillin). If this be evaporated with a few drops of the juice, a rosy tint appears in the residue.
3. Boas' test (alcoholic solution of resorcin) is carried out in the same way.

The Congo red and dimethyl-amido-azo-benzol tests can also be carried out by means of test-papers dipped in the respective solutions and allowed to dry. The test-papers keep indefinitely, and are much more convenient than the solutions.

*Töpfer's method of determining the total amount of free HCl* present is simple and fairly accurate. A few drops of dimethyl-amido-azo-benzol solution are added to 10 cc. of the filtered gastric juice, and decinormal NaHO is cautiously added until the cherry-red colour has just disappeared. This indicates the moment when all the free HCl is neutralised, and its amount can be calculated from the number of ccs. of NaHO employed.

*Lactic acid* may be recognised by *Uffelmann's test*. The reagent is made by adding two drops of Liq. Ferri Perchlor. to 30 cc. of a one per cent. solution of carbolic acid. The stomach contents are extracted with ether, which is then evaporated off, and the residue is dissolved in water. A drop or two of this



solution is added to the amethyst-blue reagent, which becomes canary-yellow in the presence of lactic acid.

*Acetic and butyric acids* may be recognised by their odour, vinegary in the one case, like that of rancid butter in the other.

*Proteoses* may be tested by the *biuret reaction*. To one drop or less of a ten per cent. solution of sulphate of copper in a test tube, which is rolled round so as to distribute the solution over its sides, add equal quantities of neutralised and filtered stomach contents, and of ten per cent. solution of NaHO. A pink colour shows the presence of proteoses, from which that of *pepsin* may be inferred. If proteoses are absent, pepsin can be recognised by the digestive action of the gastric contents on hard-boiled white of egg.

The presence of *rennin* may be recognised by adding 2 cc. of the neutralised and filtered stomach contents to 10 cc. of milk, the whole being kept at body heat for fifteen minutes, at the end of which time a solid curd should be formed.

*The motor power of the stomach* is estimated (1) by testing with ferric chloride for salicyluric acid in the urine at various intervals after a dose of salol has been taken by the mouth. Salol is not decomposed by the stomach contents, which are acid, but only when it meets with an alkaline fluid—*i.e.* in the small intestine. If the urinary reaction (a violet colour) is delayed beyond an hour and a half, the motor power of the stomach is held to be deficient. (2) The stomach should be completely empty seven hours after even the largest meal.

## ACUTE GASTRITIS

Acute gastritis may be catarrhal, phlegmonous, toxic, or membranous. The *catarrhal form* is due to the severe irritation set up by the ingestion of unripe fruits, decomposed tin meats, shell-fish, etc. It may also follow upon the abuse of alcohol or even of tea. Persistent hyperacidity (especially when due to organic acids), and delay of food in the stomach, favour its production. It is frequently a complication of convalescence from febrile diseases, low inflammation, etc.

**Pathology.**—The mucous membranes show the usual hyperæmia, exudation, and increased mucous secretion observed when mucous membranes are inflamed. The various epithelial cells of the numerous glands may become highly granular, undergo mucoid degeneration, or desquamate. There may be minute



extravasations of blood, hæmorrhagic erosions, pustules or aphthous patches.

The submucous coat is infiltrated, and the whole stomach wall may be congested. The pyloric region is principally affected.

**Symptoms.**—In severe cases the onset is sudden, with severe epigastric pain passing through to the back, and accompanied by deep *diffuse* tenderness; there is vomiting of food mixed with abundant mucus or streaked with blood; free HCl is absent; there are slight fever and marked prostration. In mild cases abdominal discomfort, nausea, and eructation end in vomiting, which brings relief.

**Treatment.**—The first point is to secure absolute rest for the stomach. It should be washed out once or twice at the outset with a dilute alkaline solution ( $\text{NaHO}$  1—1000). This removes irritants, and often stops vomiting. If it can be retained, a mercurial purge should be given. If retching continues give morphia hypodermically, and for pain apply belladonna fomentations. For thirst small pieces of ice may be sucked. Feed for the first twenty-four to thirty-six hours by nutrient enemata (see p. 167), and return to gastric feeding very gradually and carefully.

*Phlegmonous Gastritis* is very rare, and its causes are obscure. The inflammation goes on to suppuration, either diffuse or localised, and the resultant abscess may burst into the stomach or peritoneal cavity. The symptoms are those of intense gastritis, and in addition those of sepsis. The disease is usually fatal.

*Toxic Gastritis* arises from the ingestion of corrosive or irritant poisons. Intense gastritis is accompanied by symptoms of prostration and rapid collapse. In those who recover, chronic catarrh is apt to be set up.

*Membranous Gastritis* is secondary to a general infection. False membrane may be found in diphtheria, pneumonia, typhus, etc., pustules in small-pox, multiple abscesses in pyæmia. The condition is generally diagnosed *post mortem*.

## CHRONIC GASTRITIS (CHRONIC CATARRH)

This condition may follow acute catarrh or more commonly prolonged gastric irritation due to irritating or indigestible food, or to the abuse of alcohol. It is constantly present in cancer



and dilatation of the stomach, and it accompanies the passive congestion of the organ due to chronic heart disease or cirrhosis of the liver. Except in the earliest stages, the secretion of HCl is defective (*hypochlorhydria*). There is lessened motor activity, often leading to dilatation and bacterial fermentation. Degenerative changes, especially fatty, affect the glandular cells. Mucus is at first continuously secreted; later, if the mucosa is widely destroyed, the secretion may be lessened. Fibrosis occurs between the glands, and the contracting fibrous tissue narrows their necks, and thus causes intra-glandular cysts. These can be seen chiefly in the pyloric region, forming little pinhead-like projections on the surface of the mucosa (*état mamellonné*). Small erosions or superficial ulcers are sometimes present.

In some instances the interstitial changes result in great contraction, causing complete atrophy of the mucous membrane and shrinkage of the whole organ (*cirrhosis* of the stomach).

**Symptoms.**—The course is protracted and afebrile, but marked by subacute exacerbations, in which slight fever may occur. Pain comes on at once or soon after food, with diffuse epigastric tenderness greater in certain spots (by some supposed to mark the site of erosions) than elsewhere. Nausea is frequent, vomiting less so, though there is always vomiting at some periods of the course. It may be related to food, or may occur in the morning. The vomit contains much mucus, little HCl or pepsin, and partly digested food; organic acids (lactic, butyric) are also to be found. Constipation and flatulence are present, the tongue is thickly furred, and there is some wasting.

**Treatment.**—Rest to the whole organism, and especially to the stomach. Begin by washing it out. Give at first only fluids in small quantities and often, and increase the diet very carefully. Correct constipation. Of drugs, dilute hydrochloric acid after food, combined, if there is much pain, with Acid. Hydrocyan. dil., and the tonic bitters are the most valuable. Carbonate of bismuth or Liq. Bismuthi may also be given for the relief of pain. It is only in the earlier stages that cure can be expected, and then only if faulty dietetic habits are permanently abandoned.

## GASTRIC AND DUODENAL ULCER

Perforating or peptic ulcer is a lesion peculiar to the stomach, the first part of the duodenum, and the lower end of the œsophagus, *i.e.* to parts exposed to the gastric juice. The ulcer is usually single, but there may be more than one.



**Etiology.**—Gastric ulcer is most commonly met with in young women, especially those who lead a sedentary life, who live in areas shut out from the sunlight, or who are the subjects of chlorosis or anæmia. The evidence of surgery tends to show that the disproportion between the sexes is less than has been hitherto supposed, and *duodenal* ulcer is more frequent in the male. In women the disease is one of early adult life, in men it becomes commoner towards middle age. Occupation has no very marked effect in predisposing to it. Extensive burns may be followed by gastric or duodenal ulcer. Multiple ulcers are sometimes met with in septic conditions.

**Site.**—Solitary ulcers are most frequently situated on the posterior wall, near to, or involving the lesser curvature, and in the neighbourhood of the pylorus. They rarely attack the greater curvature. Acute ulcers may also be found in the middle portion, or at the cardiac end of the stomach. The cardiac *orifice* is very rarely implicated. Ulcers are occasionally situated on the anterior wall, and then are very liable to perforate.

**Characters.**—The ulcer may be acute or chronic, the acute form being small and sharply punched out, the chronic larger, funnel-shaped (broader at the level of the mucosa than in the deeper coats), and with thickened edges. The shape is circular or oval. The edges are *not* undermined, and there is an absence of vascularity in the margins and base. The edge of the acute ulcer is soft, that of the chronic ulcer firm and thickened. The floor is formed by the submucosa, the muscular coat, or the serous coat; the latter is not infrequently thickened, and adherent to the neighbouring organs. These adhesions are of a conservative nature, and explain the relative infrequency of perforation. As the ulcer deepens, its floor or base becomes narrower, so that the walls come to have a terraced appearance, and an oblique direction. If perforation occurs, the aperture in the serous coat is of small size, but presents the same clean-cut, punched-out appearance, that distinguishes the margins. Perforation is frequent in acute ulcer, but may occur in either form. In the healing of the ulcer, if the mucosa be alone involved, the granulation tissue develops from the edges and the floor, and the newly-formed tissue gradually contracts and unites the margins, leaving a smooth scar. In large ulcers which have become deep, and involved the muscular coat, the cicatricial contraction may cause serious changes, the most important of which is narrowing of the pyloric orifice and *dilatation of the stomach*. *Hour-glass contraction* of the organ may also occur.



Another result of the ulcerative process is *perforation* of the gastric walls, and consequent fatal peritonitis. On the other hand, it is not uncommon for adhesions to form between the walls of the stomach and neighbouring organs, and the ulcer, after perforating the gastric walls, burrows into the pancreas, spleen, or liver. Large vessels may be eaten into in a similar manner, and bring about a fatal issue from *hæmorrhage*. Even quite small erosions may sometimes prove fatal in this manner. Other complications through the ulcerating process are—

1. Perforation into the pleura.
2. Gastro-duodenal fistula.
3. Perforation into the lesser peritoneum, giving rise to subphrenic abscess.

**Pathology.**—The ulcer is due to two immediate factors—

1. A devitalisation of certain areas of the gastric mucous membrane.
2. Erosion of these areas through the digestive action of the gastric juice.

It is not yet known how the devitalisation of the mucous membrane is brought about, though its proximate cause is generally considered to be a local interference with the blood-supply. This is variously attributed to spasm of the arteries, to embolism, and to thrombosis. The theory of thrombosis is due to Virchow, but it does not explain all the cases, and, as Osler points out, the gastric vessels are not end-arteries. Ulcer is very common in chlorosis, and thrombosis in other parts is not common in that disease. Where ulcer is connected with septic states, embolism is no doubt the cause. In other cases Sidney Martin suggests bacterial invasion. If the gastric activity is lessened, bacteria may persist, enter the pyloric glands (where HCl is not secreted), and grow there and in the submucosa. Bolton leads evidence to show that the initial lesion in acute ulcer may be a local necrosis of the mucosa, local hæmorrhage, or local inflammation of the lymphatic follicles, these conditions being due to bacterial infection, irritants introduced by the mouth (as in toxic gastritis), venous congestion, and endogenous poisons of metabolic origin (*gastrotoxins*). Most acute ulcers tend to rapid healing; others extend in depth or superficially, or in both directions. The slower the extension the greater is the thickening; and thus arise chronic ulcers, always, he thinks, out of a previous acute condition. Trophic changes have also been invoked to account for the phenomena.



**Symptoms.**—No disease or condition may have on the one hand more characteristic, or on the other hand more ill-defined symptoms than gastric ulcer. Taking a typical case, we may expect—

1. Pain and tenderness over the epigastric region. The pain is severe and shoots through to the back (xiphi-vertebral), and is rendered worse by eating, or by firm pressure. Mackenzie states that the position of the pain varies with that of the ulcer. When the ulcer is near the cardiac end, the pain is high up in the epigastrium. When in the middle of the stomach, the pain is in mid-epigastrium, and when at the pylorus, the pain is low down in the epigastric area. This pain must be distinguished from *cutaneous* or *muscular hyperalgesia*, which sometimes accompanies it, and like the pain is *very localised*. Both are generally situated in the middle line, but the hyperalgesia may extend further to the left. The upper belly of the left rectus muscle is frequently contracted. When tenderness is severe and persistent, it may indicate a localised acute peritonitis around the ulcer.

2. Vomiting after meals; this may occur soon after food has been swallowed, but more frequently after an hour or longer, the interval being longest (two to three hours) in cases of ulcer at the pylorus. It usually gives temporary relief.

3. Hæmatemesis (vomiting of blood) may be very copious, and occurs in probably more than half the cases. It may be directly fatal, or comparatively slight. Melæna (passage of blood by the bowel) is present in about ten per cent. of cases. The quantity of blood may be so small that it is recognisable only by chemical tests (*occult* bleeding).

4. The appetite remains good, but the patient is afraid to eat, lest pain is set up.

5. The tongue is clean, and may be pale and flabby. There is little wasting.

6. There is generally excess of free HCl in the stomach contents (hyperchlorhydria). Increase of the organic acids is rare, but may occur in old-standing cases with dilatation or hour-glass constriction.

These symptoms in combination with the history, anæmia, and the absence of a tumour, point strongly to the presence of an ulcer. Beginners must, however, be warned not always to expect such typical signs. Often, indeed, the symptoms are very slight, and a copious or even fatal hæmorrhage, or the occurrence of perforation, may be the first indication of such a



condition. In doubtful cases examination by the X-rays after a bismuth meal may aid the diagnosis, and the use of the tests for occult blood (*v. infra*) is of great service.

**Duodenal ulcer**, now recognised as a common condition, is much more frequent in men than in women. The peptic ulcer occurs in the first part of the duodenum, above the point at which the acid chyme is neutralised by the pancreatic juice, and it is stated that its preponderance in the male is accounted for by the comparative fixity of the male duodenum. This predisposes to kinking at the first duodenal angle, and hinders the regurgitation of bile or pancreatic juice by which the acid chyme might be diluted. Acute ulcers may possibly be in the first instance follicular, and due to the breaking down of inflamed lymph-follicles. Whatever the primary cause, the ulcer tends to become chronic through spasm of the muscular coats of the viscus.

A duodenal ulcer is not uncommonly "silent," or attended only by slight dyspeptic disturbances, it may be even until perforation occurs. In most cases it causes distinctive **symptoms**. These are :—

1. "Hunger pain," *i.e.* pain coming on from two to four hours after food, and often at night, and relieved by taking a meal. It is due to the escape of the acid gastric juice into the duodenum, and is relieved when the pylorus is closed. It is situated in the right hypochondrium.

2. Tenderness in the right hypochondrium, with rigidity of the right rectus.

3. Repeated attacks of melæna, *sometimes accompanied, but more usually unaccompanied, by hæmatemesis*. Blood coming from a point high up in the small intestine is altered in its passage to the anus, being mixed with the fæces. The stools are black and tarry.

The pain of duodenal ulcer may be confused with that of *gall-stones*, but the latter is not relieved by food, nor does it come on after every meal.

When the amount of hæmorrhage in gastric or duodenal ulcer is small, and not to be recognised by the appearance of the vomited matter or fæces, the *tests for occult blood* must be used. A portion of the suspected matter is treated with glacial acetic acid, and the blood pigment is then extracted by ether. The addition of a drop or two of tincture of guaiacum and 2 cc. of ozonic ether causes the appearance of a bright blue colour. The benzidine test, which is also used, is much too delicate,



revealing one part of blood in 800,000. Before any test is employed, the patient must have abstained from any blood-containing food such as meat for at least forty-eight hours.

PERFORATION is indicated by the *sudden* occurrence after a meal, or severe exertion, or during vomiting, of intense pain in the upper part of the abdomen, with rigidity of its walls, faintness, rapid wiry pulse, pinched and anxious face. The pain may radiate into the back and shoulders. The abdomen is much distended, and the hepatic and splenic dulness are often absent. Later there may be fluid in the flanks.

SUBPHRENIC ABSCESS forms between the diaphragm and the liver, stomach, and spleen. After the initial symptoms of perforation, the physical signs are displacement of the cardiac apex, slight bulging of the affected side, cessation of abdominal breathing, and displacement downwards of hepatic dulness. Pulmonary complications are common.

**Treatment.**—The indications for treatment are (1) rest, (2) appropriate diet, (3) improvement of the condition of the blood.

(1) Absolute rest in bed must be insisted on for at least a month.

(2) Diet must at first be very restricted. If there has been a hæmorrhage within forty-eight hours, treatment must be begun by rectal feeding, which should be kept up for from three to ten days, according to circumstances. Proteids are not absorbed from the rectum in the form either of albumin or peptone, but glucose is readily absorbed, and so to some extent is nitrogen in the form of amino-acids. An appropriate enema consists of milk pancreatised for twenty-four hours, to separate the amino-acids, and five per cent. of pure dextrose. This may be given to the extent of ten to fifteen ounces four times daily, the rectum being washed out once daily with normal saline. In the absence of hæmorrhage, two ounces of milk and lime-water may be given orally every hour, and the quantity may be increased to four ounces by the end of the first week. If there is no pain, the diet may then be very gradually added to, the intervals between the meals being lengthened as they are increased in quantity. When milk does not agree, egg albumin may be substituted. Normal diet must not be resumed for several months. Lenhartz's dietary, which he gives, it is claimed with safety, even after a hæmorrhage, is composed of eggs beaten up with milk and sugar. One egg with eight ounces of milk is given on the first day, and



one egg and four ounces of milk are added daily until the patient takes six eggs and two pints of milk in the twenty-four hours. At the end of the first week an ounce of raw minced meat is added, and thereafter the diet is carefully extended, the number of eggs being gradually and proportionately reduced.

(3) Medicinal treatment. A combination of iron and sulphate of magnesia or Carlsbad salts will meet the anæmia and constipation. The pain may be relieved by a mixture of morphia, bismuth, and dilute hydrocyanic acid, or by the following powder: Bismuthi Carb., Sodii Bicarb., aa gr. x (grm. 0.6), Pulv. Opii gr.  $\frac{1}{4}$  (grm. 0.016). Larger doses of bismuth are needed if vomiting is prominent. In active hæmorrhage absolute rest, morphia and ergotin hypodermically, ice to suck, and rectal feeding are the measures to be taken. Adrenalin, ℥. xx-xxx (cc. 1.2-1.8), calcium chloride or lactate by the mouth or rectum, or horse serum (10 cc.) may be given in severe cases. Subcutaneous infusion of saline solution may be required.

Surgical measures are called for in the event of perforation, subphrenic abscess, repeated or very severe hæmorrhage, in cases attended with pyloric stenosis (gastro-enterostomy), and in duodenal ulcer. Operation is also justified in old-standing cases of gastric ulcer, which have not yielded to medical treatment.

## CANCER OF THE STOMACH

Primary cancer of the stomach is by no means a rare condition. The principal varieties are—

1. Adenocarcinoma, originating from the glandular epithelium, and hence cylinder-celled or spheroidal-celled. There are two forms:—(a) medullary, (b) scirrhus. The former is highly cellular, in the latter the stroma predominates. Scirrhus is the common pyloric growth.
2. Colloid cancer, in which the cells have undergone a mucinoid transformation. The growth is of the infiltrating type.
3. Squamous-celled epithelioma at the cardiac orifice is due to extension of an œsophageal cancer.

**Etiology.**—The majority of cases occur between the ages of forty and sixty years. The disease is somewhat more frequent



in males than females. In a certain proportion of cases of simple ulcer, cancer develops subsequently, but the percentage is apt to be overstated. There is no definite relationship to other diseases of the stomach.

**Morbid Anatomy.**—The pylorus is much more frequently affected, then the lesser curvature, and after these, the cardia and the greater curvature. The disease begins in the deeper layers of the mucosa, and extends in the submucous and afterwards in the muscular coat, which is often thickened. The serous coat becomes implicated, and cancerous nodules in the course of the lymphatics may be seen externally. In many instances the disease spreads far beyond its point of origin, infiltrating the coats in a horizontal direction. This is especially true of colloid cancer and of scirrhus, in the latter of which contraction of the stomach is not uncommon, while in colloid cancer almost the whole organ may be infiltrated. In other cases growth is more prominently vertical, the tumour bulging into the lumen of the stomach, and ulceration is then frequent, as in the medullary or encephaloid type of adenocarcinoma. The contraction of a scirrhus is apt to cause stenosis of the pyloric orifice, leading to dilatation of the organ. At the cardiac end it may prevent the entrance of food, and produce dilatation of the œsophagus. Ulceration frequently leads to erosion of vessels, and hence to hæmorrhage. Sometimes it causes perforation, followed by sub-phrenic abscess, but more commonly adhesions to the liver, pancreas, or intestine take place, and the floor of the ulcer may be formed by these parts. Perforation may occur into the bowel, the pleura, or the pericardium. Metastases are frequent, and involve especially the liver, lymphatic glands, and peritoneum.

**Symptoms.**—They are for a time most insidious, but sooner or later the persistent vomiting, hæmatemesis, constant pain, emaciation, anæmia, and cachexia, with the presence of a local tumour, declare the true condition. The symptoms are considered in detail in the diagnostic table appended. Remember that *persistent* gastric symptoms, appearing for the first time in a patient over the age of forty, who has previously digested everything easily, are in themselves presumptive evidence of cancer.

Free HCl is present in the gastric contents in diminished amount, or may be altogether absent in the later stages. This is by no means characteristic of cancer, as a similar condition may exist in gastric catarrh, for example; but in any case where the symptoms suggest cancer, the presence of free HCl in



normal quantity speaks for the non-malignancy of the affection. Lactic acid is often persistently present, even after a test meal from which no lactic acid could be derived. A long non-mobile bacillus—the Oppler-Boas bacillus—is also often found.

A further test—the *Wolff-Junghaus* test—is of much value, and depends upon the albuminous character of the cancer juice. Successive dilutions (1-10, 1-20, 1-40, 1-100, 1-200, 1-400) of the gastric juice are made, and placed in a series of test-tubes. The reagent consists of phosphotungstic acid, 0.3, hydrochloric acid, 1.0, alcohol (96 per cent.), 20, and water to 200 parts. This is floated on to the surface of the diluted juice, and when albumin is present a white ring forms at the line of contact. The test is positive in cancer even up to the last dilution. Blood must be absent from the suspected gastric juice, and the tests for *occult blood* must first be carried out.

On *physical examination*, a tumour may be discovered anywhere within an area formed by joining the ensiform cartilage with the umbilicus, and the umbilicus with the ninth costal cartilage. It is nodular and painful on handling; it is generally mobile, either with respiration or on palpation, and may receive an impulse from the abdominal aorta. Later, it may become fixed by adhesions. In about twenty per cent. of cases no tumour is recognisable from first to last.

Physical examination also reveals the dilated condition of the stomach; but when the pylorus is not affected, there may be no dilatation, and when the cardiac orifice suffers, the stomach may be atrophied. An X-ray examination after a bismuth meal is of great value. Enlargement of the cervical or inguinal glands may help the diagnosis, and in the late stages there may be an enlarged and tender liver, or signs of chronic peritonitis.

Cancer of the stomach generally proves fatal in from six months to two years.

### Diagnosis.—

#### CANCER

1. Is less common under than over forty years of age.
2. Epigastric pain is more or less continuous, and not much relieved by vomiting.
3. Anorexia is marked, *and early*.

#### ULCER OF STOMACH

1. Usually in young adults, especially women.
2. The pain is rendered worse by food, and vomiting gives much relief. The pain is also more localised and xiphi-vertebral in character.
3. Appetite is good, but the patient is afraid to eat.



## CANCER.

4. Vomiting is not frequent, but is copious; and in the vomited matter the Oppler-Boas bacillus may be found. Cancerous fragments are rare. Free HCl is diminished or absent, and lactic acid is often present. The Wolff-Junghaus test is positive.

5. Hæmorrhage is seldom copious, but may be frequent ("coffee-ground" vomiting); most common in the later stages. "Occult blood" is often present.

6. Loss of flesh; the development of cachexia is rapid.

7. Epigastric tumour is usually easily detected.

## ULCER OF STOMACH.

4. Vomiting is frequent, and the vomited matter contains free HCl often in excess.

5. Hæmorrhage is not so frequent, but is copious at times. May be the first symptom.

6. When the gastric symptoms are severe and prolonged, a cachectic appearance may develop, but never to the same extent as in cancer.

7. No epigastric tumour, though there may be some rigidity of the rectus.

The appearance and the general symptoms may suggest *pernicious anæmia* rather than cancer. In doubtful cases the blood should be examined.

The fact that a patient is under forty must not be allowed to exclude the diagnosis of cancer, where other circumstances point to it.

**Treatment.**—Must be mainly palliative. Easily digestible food and hypodermic injections of morphia help to make the patient's life tolerable. Bacterial fermentation should be prevented by washing out the stomach once daily with a solution of bicarbonate of soda,  $\text{ʒiij}$  to the pint (grm. 20 to the litre), and the administration of the anti-fermentatives, such as hyposulphite of soda, creosote, or glycerin of carbolic acid. Surgical procedures offer some chance of prolonging life, but to be of any use the radical operation (pylorectomy) must be performed early. In the later stages, if there is stricture of the pylorus, a gastro-enterostomy often affords much temporary relief, but though the direct mortality from the operation has been reduced, it is a formidable drain on the strength of an exhausted patient. If it is to be done, it should not be left too late.

## HÆMATEMESIS

*Gastrorrhagia* is the name given to hæmorrhage into the stomach; *hæmatemesis*, which may be an important symptom in many diseases, is vomiting of blood.



The source of the hæmorrhage may be either congested and distended venules or capillaries in the gastric mucous membrane, or the bursting of a large vessel; for example, the splenic artery in gastric ulcer.

**Causes.**—The more common are—

1. Erosion of vessels by gastric ulcer or cancer; and occasionally in chronic gastritis.

2. Congestion of the portal circulation from any cause, but especially cirrhosis of the liver; less often hepatic tumours pressing on the portal vein, or extreme backward pressure from cardiac disease.

3. The action of irritant poisons.

4. Toxic alterations in the blood and blood-vessels, leading to oozing of blood, as seen in malignant fevers, purpura, acute yellow atrophy of the liver, etc.

5. Rarely, severe hæmorrhage occurs in young girls without visible lesion of the mucosa (*gastrostaxis*); but even in these cases it is possible that a minute acute ulcer has been overlooked.

6. Blood that has been previously swallowed may be afterwards vomited (epistaxis, pulmonary hæmorrhage, etc.).

**Symptoms** of course depend on the extent of the hæmorrhage; when it is severe the symptoms of collapse are marked. The attack is usually sudden, and accompanied by a feeling of intense nausea and of weight in the stomach. In ulcer, it frequently follows a meal or sudden exertion. After the collapse has passed off, a stage of reaction occurs.

**Diagnosis.**—When the hæmorrhage is not profuse it is sometimes difficult to distinguish this condition from bleeding from the lungs.

A table showing the chief differences is appended :—

#### HÆMOPTYSIS

1. Previous history of pulmonary troubles.
2. The blood is coughed up.
3. The blood is frothy, bright red, and alkaline in reaction.
4. The blood may be mixed with sputum.
5. Dyspnœa and pains in the chest.
6. Is not usually succeeded by melæna.

#### HÆMATEMESIS

1. Previous history of gastric disturbance.
2. The blood is vomited.
3. The blood is clotted, not frothy, and acid in reaction. It may be dark.
4. The blood may be mixed with food.
5. Nausea, and weight in the epigastrium.
6. Often followed by melæna.



Whilst the above differences are of much value, they are not always conclusive. For instance, in cases of hæmoptysis the blood is occasionally *not* frothy, or some of the pulmonary blood may be swallowed and *vomited afterwards*. Again, the source of a sudden severe hæmorrhage may be very obscure, and ascertainable only by a thorough examination such as it is not desirable to make in a moment of urgency.

**Treatment.**—The first thing to be decided if possible is the source of the bleeding; but in severe hæmorrhage it is best at first to avoid excessive manipulation. Absolute quiet, mentally and bodily, must be obtained, and no treatment is more efficacious than a full hypodermic injection of morphia, combined with a little ice to suck. Ergotin may also be used hypodermically, or if necessary the other remedies referred to under Gastric Ulcer (p. 168). Feed by the rectum for the first few days, and afterwards give all food *cold*. If reaction does not follow, and the state of collapse persists, subcutaneous transfusion of warm normal saline solution may be performed. One or two pints may be injected. In very severe and repeated hæmorrhages operation with ligature of the bleeding vessel may save life.

## DILATATION OF THE STOMACH

This condition may be non-obstructive or obstructive. Non-obstructive dilatation is due to atony of the muscular coat, whether this be the result of repeated over-distension by excessive meals, of constitutional diseases such as anæmia and the fevers, or of defective innervation. It is a frequent occurrence in chronic gastric catarrh.

*Obstructive dilatation* is caused by (1) stenosis of the pylorus (cancer, cicatrising ulcer), (2) pressure on the duodenum, or contraction after duodenal ulcer, (3) constriction of the pylorus by adhesions in chronic peritonitis.

In great dilatation there is bacterial fermentation, owing to delay of food in the stomach. In the lesser degrees there may be hyperchlorhydria, but more frequently there is hypochlorhydria, as in gastric catarrh. If the obstruction lie beyond the pylorus there is hyperchlorhydria, and bile is present in the vomited matters.

**Symptoms** occur at irregular intervals; it may be several hours after food, or at the end of the day, or several days may elapse between the attacks. There is diffuse burning epigastric



pain (*pyrosis*), relieved by vomiting, which patients often excite. Flatulence and constipation are marked. Wasting is present. Both respiration and circulation may be affected. Where dilatation is marked, the outline of the greater curvature, and, if the stomach is displaced (*gastroptosis*), of the lesser also, may be visible. By forcibly stroking the epigastrium, peristaltic movements of the stomach may be set up. On shaking the patient's body from side to side, a splashing sound (*succussion*) can often be heard, due to the mixture of gas and fluid in the stomach. It is not of diagnostic value unless heard after the time at which food should have normally passed into the intestine (five to six hours after the last food or drink). To determine the size of the stomach, it may be artificially distended by fluid or by gas. Air may be pumped in, or 30 grs. (grm. 2.0) of tartaric acid may be given in half a tumbler of water, followed by 30 grs. of bicarbonate of soda in the same quantity of water.  $\text{CO}_2$  is thus developed in the stomach. The greater curvature can then be percussed out, giving a dull note if fluid has been used, if gas a tympanitic note. Auscultatory percussion may also be employed.

The vomited matters are characteristic. The quantity is larger than the normal content of the stomach, and the taste is excessively sour (in the early stages sometimes from hyperchlorhydria, later from the defect of HCl and excess of organic acids). Fragments of partly digested food are found, and under the microscope the bacillus acidi lactici, bacillus butyricus, sarcina ventriculi, and yeast-cells.

Examination by the X-rays after a bismuth meal reveals the size of the organ, the possible presence of gastroptosis, and the delay in the passage of food into the bowel.

**Prognosis** is that of the causative disease, though appropriate treatment always relieves symptoms.

**Treatment.**—Careful regulation of the diet, avoiding carbohydrates where there is much bacterial fermentation. Meals should be small and dry. In non-obstructive cases, daily lavage of the stomach with antifermentative or alkaline solutions (boric acid, permanganate of potash, bicarbonate of soda) continued till the returning fluid is clear, then administration of antifermentatives, such as creosote or hyposulphite of soda. Massage and douching of the abdomen may be of value.

In obstructive cases washing out is only palliative. Gastroenterostomy or pylorotomy should be done as soon as the general health permits.



## HYPERTROPHIC STENOSIS OF THE PYLORUS

This condition, which may occur in adults, is more frequent as a congenital affection. Marked hypertrophy is associated with spastic contraction of the pyloric muscular fibres.

**The symptoms** are characteristic. A few days after birth the child begins to vomit whatever food is swallowed, constipation is obstinate, and emaciation is rapid and extreme. The peristaltic movements of the stomach are visible, and a tumour is palpable in the pyloric area.

Under very careful dieting with lavage of the stomach many cases recover; in others operation is necessary. If it is to be done, it should not be delayed till emaciation is advanced.

## NERVOUS DYSPEPSIA

As the diagnosis of gastric disease becomes more accurate, the term dyspepsia acquires a more limited significance. Many cases, formerly grouped as acid or atonic dyspepsia, are now recognised as early stages of gastric ulcer or catarrh, as mild cases of acute gastritis, or as symptomatic of general affections such as anæmia or renal disease. The most definite group of cases, that of nervous dyspepsia, comprises a host of neuroses of the stomach, of which only the most prominent can here be discussed.

The subjects of these functional disorders not infrequently manifest their nervous tendency in other ways, being often of nervous heredity, or presenting symptoms of neurasthenia or other nervous disease. In many instances, however, the gastric affection is much the most prominent element.

Besides the underlying nervous predisposition, causes contributory to the development of one or other of these forms of dyspepsia are the following:—

Intemperance in eating or drinking; imperfect mastication, often due to bad teeth; excessive use of tea, coffee, or tobacco; and mental overwork or worry.

Neuroses of the stomach are of three classes, motor, sensory, and secretory. In each class the error may be one either of excess or defect. Thus among *motor neuroses* there are on the one hand (1) *peristaltic unrest*, with noisy gurgling sounds (*borborygmi*) audible at a distance and distressing to the patient, (2) nervous *eructation* or *vomiting*, unattended by



nausea and most frequent in women, (3) painful *spasm* of the cardia; and on the other hand *atony*, which as we have seen (p. 173) is occasionally due to defective innervation, and leads to moderate dilatation.

*Gastralgia* or *gastrodynia* is the most important of the *sensory neuroses*. It occurs chiefly in neurasthenic women, and is usually associated with anæmia and constipation; but it may be met with in the male. It is marked by severe attacks of epigastric pain, which may radiate towards the back, and is often periodic. The pain arises independently of meals, and is usually relieved by food and by pressure. Vomiting is exceptional. The diagnosis should only be made after careful exclusion of organic disease of the stomach, locomotor ataxy, and gall-stones.

An example of sensory defect is to be found in the rare condition of *anorexia nervosa*, an occasional manifestation of hysteria, in which extreme emaciation accompanies annihilation of the sense of appetite.

*Secretory neuroses* are often associated with sensory or motor disturbances, but may occur alone, in which case symptoms may be long delayed. Error of excess is manifested in two forms, hyperacidity and excessive secretion. In hyperacidity (*hyperchlorhydria*) an abnormally acid gastric juice is secreted during the process of digestion. The symptoms commence an hour or two after meals, and are most marked after the chief meal of the day. They are a sense of weight and burning in the epigastrium, often with eructations of sour fluid. Vomiting occurs occasionally, and is followed by relief of pain. The vomited material is highly acid. Appetite is at first normal, and there is often a "sinking feeling" before meals. Such cases are common in young and nervous women, but may be met with at all ages and in all classes. Hyperchlorhydria is frequently associated with gastric ulcer.

Excessive secretion (*gastrosuccorrhœa*) may be periodic or continuous. Periodic attacks (*gastroxynsis*) occur at night when the stomach is empty. They are attended by severe spasmodic pain, and vomiting of large quantities of very acid gastric juice, quite unmixed with food. The condition is rare. Continuous over-secretion leads to an irritative spasm of the pylorus and hence to dilatation.

Error of defect may take the form of subacidity or absence of secretion. Subacidity (*hypochlorhydria*) is met with, apart from organic or general disease, in those who follow sedentary mental occupations, especially towards middle age, in women about the



menopause, in neurasthenia, and in hysteria. It leads to a sense of fulness and oppression after meals, lasting sometimes all day long, with flatulence, headache, drowsiness, and constipation. Vomiting is not a symptom. The tongue is pale, flabby, broad, and indented by the teeth. The condition is often associated with motor defect, and may then end in dilatation.

Absence of secretion (*achylia gastrica*) occurs in catarrh of the stomach when the mucosa is entirely atrophied, but also as a neurosis. Food withdrawn in the usual manner after a test meal is found to be unaltered. Symptoms may be absent for years, intestinal replacing gastric digestion, but ultimately dyspeptic symptoms make their appearance.

**Treatment.**—Success in treatment is seldom to be looked for if the gastric symptoms alone are considered. Any constitutional condition must be dealt with, anæmia, for example, by iron or arsenic. Worry and mental exhaustion are best met by change of air and scene, and in some neurasthenic cases the Weir-Mitchell treatment (complete isolation, absolute rest in bed, and massage) is of great benefit. An exclusively milk diet is not desirable in the majority of instances; indeed in hyperchlorhydria, Osler recommends a strictly meat diet, the meat being either raw or very lightly cooked. In hypochlorhydria a light, easily-digested, mixed diet is the best, and in hyperæsthesia rectal feeding is sometimes required. In gastric atony the meals should be small and frequent, and fluids should also be limited. Lavage of the stomach is useful in excessive secretion and in atony accompanied by dilatation.

One of the most important points in medicinal treatment is to avoid indiscriminate drugging. Such patients are too apt to fly from one remedy to another, and to add a drug-dyspepsia to the original ailment. Strychnine or nux vomica is in most cases useful to counter the nervous predisposition, but other drugs must be used to meet special indications. Thus the pain of hyperchlorhydria is best treated by large doses of alkalies given at the height of digestion, one or two hours after food; in subacidity, on the other hand, small doses of alkalies before meals may promote secretion, or hydrochloric acid after food may be given to replace the defect. In excessive secretion, alkalies should be given during the attack of pain, *i.e.* when the stomach is empty. The pain of gastralgia may sometimes be relieved by bromides; severer cases require opium, best given in powder along with bismuth, and if possible without the patient's knowledge. Morphia should be used only as a last resort, and the syringe



should never be entrusted to the patient. The accompanying constipation always requires attention (*see* p. 191).

Stimulants and narcotics should as a rule be entirely forbidden, and the use of tobacco either cut off or restricted to a minimum. The habitual use even of strong coffee is undesirable.

#### IV. THE INTESTINES

##### ENTERITIS

The mucous membrane of the intestines is inflamed in very many diseases, especially in the infectious diseases, such as cholera, typhoid fever, dysentery, etc., in portal obstruction and chronic cardiac disease, and as a terminal event in profound anæmias, cancer, and Bright's disease. But for clinical purposes, the term enteritis is much more strictly limited, and means an inflammation of the alimentary tract due to irritation by indigestible food, or a general catarrhal condition following exposure to cold, etc.

As in all other inflammations of mucous membranes, we may have the following varieties:—

1. The ordinary, or catarrhal inflammation.
2. The croupous or fibrinous form, when the inflammatory exudation is unusually rich in fibrin, and tends to coagulate.
3. The phlegmonous or gangrenous, and
4. The ulcerative type.

It should be noted that in some forms of enteritis the actual inflammatory changes are very slight, and the symptoms are mainly due to bacterial intoxication.

##### CATARRHAL ENTERITIS

*Catarrh of the intestine* is most frequent in the hot summer months, probably because of the ingestion of large quantities of unripe or decomposed fruit, contaminated milk, etc. It should be remembered that food-stuffs are at this time open to fly-borne infection, and flies may thus be regarded as an indirect cause of enteritis. It sometimes follows a sudden fall of temperature. Other causes are the prolonged administration of mercury or arsenic, too vigorous purgation, and excess in strong alcoholic drinks.

**Morbid Anatomy.**—In acute enteritis the most marked changes are observable *in the ileum* as hyperæmia of the valvulæ



conniventes and a swollen condition of the solitary glands, which project like small shot, and sometimes present abraded or slightly ulcerated surfaces (follicular ulcers).

In chronic catarrh the most marked changes are to be seen in the *large* gut. The mucous membrane is much thickened, often pigmented, and shows extensive small follicular ulcers, which after healing give a peculiar worm-eaten character to the gut on holding it up to the light. The surface is coated with viscid mucus. The superficial veins are distended, piles are often prominent in the rectum, and polypoid outgrowths may spring from the mucous surface.

**Symptoms of Acute Enteritis.**—The chief symptoms are—

1. Diarrhœa.
2. Abdominal pain, especially around the umbilicus.
3. Nausea, anorexia, and sometimes vomiting, *without* *fæcal odour*.
4. In severe cases, pyrexia.

In a typical case of enteritis, watery diarrhœa is the chief symptom, and owing to decomposition within the intestines, the stools are very offensive. They consist principally of undigested food, epithelial debris, triple phosphates, biliary pigments, and mucus. Various organisms, such as the *proteus vulgaris*, the *bacillus enteritidis*, and the *bacillus coli*, may also be found. The fæces may appear to contain an excessive amount of bile through the haste with which the duodenal contents are hurried on; or on the other hand, the catarrhal secretions may block up the common bile-duct and prevent the bile from entering the bowels, in which case the fæces will be pale and clay-like, and a subsequent jaundice may develop. When undigested food is present, the small intestine is the seat of the catarrh (*lienteric* diarrhœa); if there is much mucus, the colon.

Pain of a colicky type, worst just before the passage of a motion, is usually present. There is no abdominal tenderness, nor is there visible peristalsis.

The general symptoms are marked. The face is pinched, the tongue dry and furred. Fever may be absent, and is seldom high, but the pulse is disproportionately rapid. Thirst and oliguria are present, and in severe attacks, vomiting. In fatal cases collapse, due to toxæmia, and ending in coma, appears, often without warning. But in the great majority of cases recovery takes place in a few days, while some pass into the chronic stage.



In *chronic* enteritis diarrhœa, or diarrhœa alternating with constipation, is the main symptom. Colicky pains may be present or absent. The stools vary in character with the seat of the lesion. Wasting is prominent, and the complexion is muddy and pallid.

**Treatment.**—The patient must be confined to bed, and put upon a milk diet. If the attack is due to improper food, the bowels should be cleared by castor-oil and laudanum, or by a mercurial purge. This is unnecessary where there has already been much diarrhœa. Pain may be relieved by warmth to the abdomen or by turpentine stupes; morphia is sometimes necessary. Bismuth in large doses, or chlorodyne may be used to check the diarrhœa; but it tends to abate spontaneously when the irritant has been evacuated. Starch and laudanum enemata are also useful. In chronic cases the stools should be carefully examined. Bismuth, or the lead and opium pill should be used to check the diarrhœa. Salol is also valuable as an intestinal antiseptic. In protracted cases the colon may be douched out with astringent enemata.

**Catarrhal Enteritis in Children** is due almost entirely to improper feeding, and is rarely met with in breast-fed infants. Its onset is favoured by warm weather, and by debility due to rickets or to teething, and sometimes “chill” may determine an attack. It is most frequent in the first two years of life. Organisms of various kinds, including *b. coli*, *b. proteus*, and the pyogenic cocci are found abundantly in the stools, and in a large proportion of cases organisms allied to the bacillus of Shiga have been discovered.

The intestinal changes are similar to those of enteritis in the adult. The mesenteric glands may be enlarged. In chronic cases there may be extensive ulceration of the colon.

**Symptoms.**—In *mild cases* diarrhœa is the chief symptom. The stools are greenish and very offensive, and resemble chopped spinach. They may contain undigested casein. Abdominal pain causes restlessness and irritability. Fever is slight, and vomiting only occasional. In the *severer forms* (gastro-enteritis), of which milk contaminated by flies is the most frequent cause, the symptoms suggest a bacterial intoxication. Vomiting and diarrhœa are urgent, the stools being more fluid, but still greenish and offensive. The abdomen may be distended or hollow, and is tender. There is sharp fever, reaching 104°–105° F. Wasting is rapid, and collapse and coma may cause death in a few days, or after a longer interval bronchopneumonia may end the scene.



Most cases recover ; some become chronic. The disease may be *chronic* from the first, the stools being large, pale, pasty and offensive, and the abdomen distended. The child gradually wastes, and death is due to asthenia.

*Treatment.*—Where the disease occurs in breast-fed children, it is due to over-requent feeding. A simple purge and proper regulation of the meals usually suffice for a cure. Cows' milk, boiled, and diluted in proper proportion to the child's age, with the addition of a little lime-water or citrate of soda to prevent the formation of large curds, is better than any artificial food for hand-fed infants. But in the presence of enteritis, milk should be stopped for a few days, and albumin water or raw-meat juice given in its stead, with barley-water for thirst. The abdomen must be kept warm. At the onset a dose of castor-oil should be given, or calomel in divided doses, or hydrarg. c. creta, gr.  $\frac{1}{2}$ —1 (grm. 0·03—0·06), twice or thrice daily. Lavage of the stomach where vomiting is urgent, and of the colon in profuse diarrhœa, are valuable measures. Bismuth and, if necessary, Dover's powder may be used to check the diarrhœa, but all forms of opium must be used with great care in young children, and none should be given to an infant under six months old. Alcohol is sometimes necessary to meet the collapse.

*Cholera nostras*, a term now almost obsolete, is simply a severe form of acute gastro-enteritis due to food poisoning, and considered at p. 147. *Cholera infantum* with a similar cause presents similar symptoms ; but as it occurs in infants, its mortality is much greater.

*Croupous* or *diphtheritic enteritis* occurs chiefly in the final stages of chronic constitutional diseases (granular kidney, etc.), and is generally fatal. The treatment is purely symptomatic. *Phlegmonous* or *gangrenous enteritis* is a local affection, due to strangulated hernia, intussusception, or chronic obstruction. It involves all the coats of the bowel, and is therefore complicated with peritonitis, to which its chief symptoms are due.

**Ulcerative Enteritis.**—The intestine is subject to many forms of ulceration, among which the enteric and dysenteric forms, the duodenal ulcer, and the follicular ulcers of catarrh have already been described. Specific ulcers may also occur in syphilis and in tuberculosis. *Syphilitic ulceration* is most common in the rectum, and occurs chiefly in women. It is due to the growth of gummata in the submucous coat, and ends in the gradual



onset of a hard fibrous stricture, easily distinguishable from that of malignant disease.

*Tuberculous ulceration* affects principally the ileum, cæcum, and colon. The ulcers contrast with those of enteric fever in that they are irregular in outline, with their long diameter in the circumference, not the axis, of the bowel, and their edges are infiltrated and undermined. They involve the submucous and muscular coats, and may perforate. Cicatrisation may cause a single or multiple stricture of the bowel. In the ileo-cæcal region tuberculosis may cause a chronic hyperplasia, sometimes involving the appendix. The symptoms are periodic pain, alternating constipation and diarrhœa, and slowly advancing stricture. An elongated, hard, and tender tumour-like mass is found in the right iliac fossa. It is localised, and may be removable by enterectomy.

*Stercoral or fæcal* ulcers are apt to form in the colon in cases of long-standing constipation, or above the stricture in chronic intestinal obstruction.

*Ulcerative colitis* is a non-specific affection occurring in both sexes, most frequently about middle age. The mucosa is widely destroyed, and only islets or polypoid projections may remain. The muscular coat, which is greatly thickened, forms the floor of the ulcers. The colon is often dilated. Watery diarrhœa, the stools being brown, offensive, and frequently blood-stained, griping abdominal pain without tenesmus, slight abdominal distension, wasting, and sometimes fever, are the chief symptoms. Death may be due to asthenia, hæmorrhage, or perforation. The treatment is that of chronic dysentery. In an early case, the colon may be irrigated through an artificial anus above the cæcum.

*Mucous colitis* is a catarrhal colitis accompanied by atony of the bowel, and is most frequent in women of about middle age and of a neurotic disposition. It is characterised by the frequent or occasional passage of membranous casts of the lower bowel, the casts varying from a few inches to several feet in length. They are transparent, gelatinous, and show fæcal staining. Embedded in the mucus of which they consist are intestinal epithelium and crystals of cholesterin and triple phosphate. In the intervals between the attacks constipation, flatulence, and abdominal discomfort are common symptoms; the passage of the casts is preceded by colicky pain. The treatment consists in a bland non-irritating diet, irrigation of the



bowel, and attention to the nervous predisposition ; but although recovery is not uncommon, many cases are very obstinate.

## APPENDICITIS

Under this title are now included the conditions formerly known as typhlitis and perityphlitis, as it is recognised that the cæcum itself is rarely affected.

**Anatomy.**—But for a small part of the upper portion of its posterior wall, which is in immediate contact with the iliac fascia, the cæcum, which extends upwards for about three inches from a point half an inch above Poupart's ligament, is usually entirely surrounded by peritoneum. Its posterior surface may in rare cases be devoid of peritoneum, or be included within two mesenteric folds forming the boundaries of a retro-cæcal fossa. The ileo-cæcal valve, which prevents regurgitation into the small intestine, is situated on the left side of it, and at its upper end. The *vermiform appendix* arises posteriorly from the inner wall of the cæcum, below the ileo-cæcal valve. It, too, is usually entirely surrounded by peritoneum, but its root may be extra-peritoneal. A mesentery is attached to it for the proximal one-third or two-thirds of its length, the tip being free. The appendix may take different directions, running (1) upwards and inwards behind the coils of the ileum, (2) upwards, or upwards and outwards, either in the retro-cæcal fossa, when that exists, or in front of the colon, (3) downwards and inwards into the pelvis ; and (4) it may be coiled upon itself behind the cæcum. Its length varies from half an inch to nine inches, the average being three to four inches. Its root is nearly opposite McBurney's point.

**Etiology.**—The disease is infective, the organism usually at fault being the *bacillus coli*, although others, such as the pyogenic cocci and *bacillus proteus*, are also found. In most cases the organism excites the disease only in presence of some cause of injury to the mucous membrane of the appendix. Thus catarrh of the appendix may cause blocking of its lumen ; fæcal concretions, particularly in those of costive habit, may lodge in it ; and less commonly it may be damaged by foreign bodies, such as pins or grape-seeds. Even without injury due to such causes, virulent bacilli may in some instances cause inflammation. The disease may follow chill or muscular overstrain, and may complicate pneumonia or the other infections. It is most common between the ages of fifteen and thirty.



**Morbid Anatomy.**—The main varieties are (1) catarrhal, (2) ulcerative, often leading to perforation, and (3) gangrenous, in which the tip of the appendix or even the whole organ may slough off. After repeated attacks, the inflammation may become chronic, in which case appendix, cæcum, and small intestine are firmly united by adhesions, which may cause partial or complete obstruction of the bowel. In other instances chronic catarrh leads to (1) partial obliteration of the lumen, resulting in the formation of appendicular cysts, which may rupture into the peritoneal cavity, or to (2) complete obliteration.

In the ulcerative variety, the ulceration may be superficial or deep. Deep ulceration leads to perforation, of which the consequences depend upon the presence or absence of previously formed adhesions, and upon the relations of the appendix to the peritoneum.

1. *Perforation may occur after adhesions have formed, and is followed by circumscribed intra-peritoneal abscess. This localised abscess may after a time open into the peritoneal cavity, and excite an intense general peritonitis.*

2. *If the perforation takes place between the folds of the mesentery, or at the root of the appendix when that is uncovered by peritoneum, the result is a retro-peritoneal abscess; in such cases, the pus may burrow in the cellular tissue, between the gut and the iliac fascia, and may then extend in one of three directions—*

- (1) Upwards, forming a perinephric abscess.
- (2) Downwards to Poupart's ligament, where it is prevented from extending down the thigh by the union of the ligament and fascia, and so bursts externally.
- (3) Into the true pelvis, when it may burst into the rectum or bladder, or through the obturator foramen.

3. *Perforation may occur before adhesions are formed, the appendix hanging free in the peritoneal cavity. In such cases, a speedy death results from violent septic peritonitis.*

**Symptoms.**—In an ordinary attack the initial symptoms are pain of sudden onset, at first over the whole abdomen, but soon localised to the right iliac fossa, local tenderness, greatest at McBurney's point (which bisects the line between the right anterior superior iliac spine and the umbilicus), rigidity of the right rectus, elevation of temperature, furred tongue, constipation, and vomiting, not excessive and not fæcal. There may be frequent micturition. The patient lies on his back, with the



right thigh slightly drawn up. A definite swelling can usually be made out in the iliac fossa. The great majority of these cases recover, fever and tenderness lessening about the third day, and the illness being over in about a week.

Should the case go on to the formation of a local abscess, the fever continues or increases, assuming a septic type, and the swelling becomes larger, harder, and more tender. Fluctuation is very rarely present. There is well-marked leucocytosis, and the general symptoms are severe.

When perforation into the peritoneum occurs, from ulcer or from gangrene, the symptoms are those of collapse, or those described under general peritonitis.

There is a great liability to recurrence. Repeated attacks often result in increased density of the adhesions, obliteration of the appendix, and ultimate recovery ; but in any one of them abscess or perforation may occur.

**Diagnosis.**—The diagnosis is sometimes difficult, and the symptoms may closely simulate obstruction of the bowels ; but in appendicitis the tumour is diffuse, and fæcal vomiting is never present, while in obstruction there is no fever. In children the history, tenesmus, bloody discharge, and the results of rectal examination under chloroform, make a diagnosis of *intussusception* fairly easy, though it sometimes simulates appendicitis. Biliary and renal colic, and pelvic cellulitis in women are also conditions which must be eliminated before arriving at a diagnosis.

Where the appendix extends downwards into the pelvis, the existence of tumour may be made out only on rectal or vaginal examination.

**Treatment.**—In all cases of appendicitis immediate operation is the best practice. Although complete recovery is the rule in ordinary cases, without surgical interference, yet there is no foretelling that a perforation may not at any moment occur, and cause a fatal peritonitis. Operation is of course necessary in the presence of abscess, and where there is generalised peritonitis it affords the only hope of recovery, though the hope is but slender. Medical treatment finds a place only where operation is refused, or in anticipation of an operation. In such cases hot fomentations, enemata to open the bowels, morphia only when necessary for the relief of pain, fluid diet (chiefly milk), and absolute rest in bed are the chief means of treatment.



## INTESTINAL OBSTRUCTION

The causes of intestinal obstruction are numerous. They may be divided into—

1. *Causes originating in the intestine itself.* These are intussusception; volvulus; stricture, malignant or non-malignant; and obstruction of the lumen by gall-stones, hardened fæces, or foreign bodies.

2. *Causes external to the intestine.* Such are bands or adhesions, giving rise to *strangulation*; incarceration by hernial or other openings; and compression by large tumours.

*Strangulation* is the most common cause of acute obstruction. The bands or adhesions causing it are generally the result of old peritonitis, but the bowel may also slip through omental or mesenteric slits, into peritoneal pouches, or under an adherent Meckel's diverticulum or appendix. The small intestine is involved in ninety per cent. of the cases. Males suffer more often than females, and young adults more often than children or elderly people.

*Intussusception* is most common in male infants and children up to the age of ten. In this affection one portion of bowel is invaginated into another, usually the small intestine into the large. There are thus three layers of bowel, an innermost or *entering* layer, a middle or *returning*, and an outermost or *receiving* layer. The entering bowel cannot return because of the swelling produced by the pressure on its veins. It is therefore dragged steadily onwards by the peristaltic action of the receiving layer, and thus the originally small tumour may increase in size till it becomes more than a foot long. The *ileo-cæcal* variety, in which the ileo-cæcal valve enters the colon, is the most frequent. *Enteric* and *colic* forms also occur. In the former, one part of the small bowel is invaginated into another; in the latter one part of the colon into another. The *ileo-colic* form, in which the ileum passes through the ileo-cæcal valve, is the least common.

*Volvulus* is a twisting of the bowel upon itself, or of one loop of bowel upon another. It is commonest in the sigmoid flexure, where the mesocolon is long, and occurs chiefly in males about middle life.

Acute obstruction may also be caused by a gall-stone ulcerating through the gall-bladder into the small intestine, which it blocks.



The other causes mentioned,—stricture, whether due to a malignant tumour or to the cicatrisation of ulcers, impaction of fæces, compression or traction,—usually give rise to chronic obstruction.

**Morbid Anatomy.**—In acute cases the intestine above the obstruction is distended, and filled with a mixture of fluid and gas. Its walls are deeply congested, and may be gangrenous. In intussusception, the opposed peritoneal surfaces may be united by adhesions, and the invaginated part sometimes sloughs. Peristalsis is at first active above the obstruction, but paralysis of the gut succeeds. Below the obstruction the bowel is contracted and empty.

**Symptoms of Acute Obstruction.**—The characteristic symptoms are pain, constipation, and vomiting, with abdominal distension. The *pain* sets in suddenly, and is usually intense. At first paroxysmal, it becomes continuous. Its seat is in the middle line, and in obstruction of the small intestine it does not descend lower than the umbilical region, while if the large intestine is affected, it descends into the hypogastric region. The *constipation* is absolute, not even flatus being passed; but fæces may be passed, or may be removed by an enema, from the bowel below the obstruction. The *vomited matter* consists at first of the stomach contents, then of bile-stained material, and finally of brownish fluid with a fæcal odour (*stercoraceous* vomiting).

There is a *uniform distension* of the abdomen, unless the obstruction is high up, but the flanks do not bulge. The abdomen is *not tender* unless peritonitis has set in. *Paroxysmal peristaltic movement*, visible through the abdominal walls, is an important sign. In obstruction in the small intestine, the peristalsis is mainly in the umbilical region; in obstruction low down in the colon, the waves may pass from right to left along the line of that viscus. A *tumour* may sometimes be felt in malignant stricture, and in *intussusception* a sausage-shaped tumour-like mass may be found in the right iliac fossa or in the line of the colon. Occasionally, in infants, it is to be felt in the rectum, and the ileo-colic valve may be recognised there. Blood is passed by the bowel, and tenesmus is often marked.

The general symptoms are those of collapse, the face being pinched, the skin covered with cold sweat, the pulse small and rapid, the tongue dry, and the urine scanty. The temperature is normal or subnormal. Thirst is great. Death results from asthenia or peritonitis in from three to six days.



In *chronic obstruction* (most commonly due to malignant tumour) there is a history of gradually increasing constipation, alternating, it may be, with attacks of diarrhoea. The fæces are narrowed in calibre. They may assume a pipe-stem shape or be flattened like a tape-worm, or they may be passed in small rounded masses like sheep's dung. Frequently they are smeared on the surface with blood and pus. Portions of the growth are sometimes found. Recurrent threatenings of acute symptoms end in complete occlusion and death.

**Diagnosis.**—The seat of obstruction may sometimes be determined by rectal examination. In females a vaginal examination should also be made. The hernial orifices must be carefully examined, and abdominal palpation may reveal the site of a malignant tumour. In obstruction low down, the colon is distended and peristalsis may be visible over it; in obstruction in the small intestine, distension and peristalsis are more prominent in the umbilical region. In the former, blood and mucus may be passed by the bowel; in the latter, fæcal vomiting occurs early. The position of the pain is also helpful.

The cause of obstruction may sometimes be ascertained from a consideration of the age and the history (peritonitis, pelvic disorders, etc.), in other instances it is not accurately ascertainable.

**Treatment.**—*Purgatives must not be used*, but the lower bowel may be cleared by enema. In fæcal impaction, recognisable by rectal examination, the scybalous masses may be broken up by the finger, and cleared away by a large injection. In an early case of intussusception, return of the bowel is sometimes effected by a large injection of warm water, very slowly administered, the patient lying on the back with the hips raised. During the process, the operator should keep one hand on the abdominal tumour to watch its retrocession, and the method should not be attempted unless he is prepared for immediate laparotomy in the event of failure. The method is now seldom used in this country, and it is better to operate at once. Food must be given by the rectum only, nothing but small quantities of ice for the thirst being swallowed by the mouth. To relieve the urgent vomiting, the stomach may be washed out three or four times daily. For the pain hot fomentations may be tried, but morphia is usually necessary. It is not desirable to give it until the diagnosis is decided, and then only in sufficient quantity to relieve the pain. In all cases of acute obstruction unrelievable by enema, operation, and *early* operation, affords the only chance of success, and the attendant must be firm in regard to



its necessity, even against the opposition of the patient or the relatives.

In chronic obstruction careful regulation of the diet, opium for the relief of pain, and enemata may tide over threatening symptoms. Surgery is called for when the obstruction becomes complete.

## TUMOURS OF THE INTESTINES

Simple tumours are uncommon, but sometimes produce obstruction. Of malignant growths by far the most frequent is—

**CANCER.** This affects the large intestine principally, especially the various flexures of the colon *and the rectum*. It is commonest in the male, between forty and sixty, but it may arise as early as twenty years of age.

**Morbid Anatomy.**—The growth, which is usually a cylinder-celled carcinoma, may be annular, or irregular and nodular. It tends to early ulceration. Metastases may be found in the mesenteric and other intra-abdominal glands, or a generalised peritoneal carcinosis may occur. It may spread by the blood-vessels through the portal vein to the *liver*.

**Symptoms.**—Besides cachexia and emaciation, commencing early when the lesion is high up, late when it is low down, the symptoms are—

1. Pain of two kinds—a permanent, dull, localised pain, often very slight, sometimes absent; and paroxysmal, colicky pain, worst just before defæcation.
2. Obstinate constipation, or constipation alternating with diarrhœa.
3. Reflex vomiting. Stercoraceous vomiting may occur when obstruction is complete.

The *physical signs* are (1) *tumour*, present in about forty per cent. of the cases. The swelling is hard, nodular, tender, and *freely mobile*; (2) *meteorism*, localised or general; (3) in the later stages, where obstruction is becoming complete, *visible peristalsis*; (4) *alterations in the fæces*, which are mixed with blood and pus in large or only microscopical amount, and may be diminished in calibre when the stricture is low down; (5) occasionally ascites, or enlargement of the inguinal glands.

In *rectal cancer*, along with all the above symptoms, *pressure symptoms*, such as unilateral or bilateral sciatica, may occur. The first evidence is usually interference with defæcation. The stools are altered in calibre (“pipe-stem” or “sheep’s-dung”



stools). This is followed by tenesmus, and repeated unsatisfactory efforts at evacuation. Later, mucus, blood, and pus become persistent in the stools. Digital examination reveals the presence of a hard, irregular, nodular tumour. The chief complications are *rupture* into neighbouring viscera, and metastases in the *liver*.

**Diagnosis.**—In all cases in which the existence of an intestinal, or indeed of a gastric tumour is suspected, it is important to have the bowels thoroughly evacuated before the examination. In rectal cancer one must not be satisfied with a mere diagnosis, but must endeavour to determine the possibility of relief by operation. For this, the extent of the tumour, the degree of mobility, and the presence of complications must be considered.

Syphilitic stricture must be distinguished from cancer of the rectum. The ulcers of syphilis do not present the protuberant edges of carcinoma; they are *multiple* and the process is usually diffuse; and syphilis is often accompanied by periproctitis or external abscesses. This form of stricture is more frequent in women, cancer in men.

**Treatment of INTESTINAL CANCER.**—No *cure* results from treatment, even in operable cases, for there is a liability to recurrence at any point of the after history. In inoperable cases treatment must be directed to (1) temporary increase of strength—tonics, and especially dietetic treatment; (2) removal of symptoms of stenosis by mild laxatives or appropriate changes in diet; (3) for the relief of pain, narcotics; (4) operative measures of a palliative nature. These are very various, according to the situation of the tumour, but rarely lead to more than temporary amelioration. The technique of the operation of colostomy has now been much improved, and if it is skilfully done, the after condition of the patient may be fairly comfortable; but a badly-formed artificial anus leads to a state of misery not less than that of the disease.

## DIARRHŒA

The frequent passage of loose motions occurs, as we have seen, in many diseases associated with intestinal lesions; but it may also arise from purely functional causes. Diarrhœa is due either to an increase in the intestinal secretions, or to increased peristalsis, and from one or other of these causes it may be excited by the irritation of unsuitable food, unripe fruit, etc., by drinking cold liquids, by chemical irritants, by putrefactive decomposition in the large intestine, by sudden changes



of temperature, and by certain nervous influences, such as emotion or fear. It is also frequently met with in the late stages of portal congestion, as in cirrhosis of the liver, or of valvular affections of the heart and chronic renal disease. Irritation due to food often leads to diarrhœa occurring on rising in the morning, and may also cause looseness after meals. Vegetable foods, rich in cellulose, or those containing an excess of sugar or fat, are particularly apt to cause it. In various nervous conditions it is liable to occur after meals.

**Diagnosis.**—It is not always easy to say whether the diarrhœa is a mere temporary disturbance, or evidence of an organic disease. In all cases note—

1. How the looseness commenced.
2. The consistency, colour, and odour of the fæces. Look for blood, mucus, undigested food, excess of fat, segments of worms, etc.
3. The presence of pain or abdominal tenderness.
4. The amount of pain at the time and immediately after defæcation.
5. The presence or absence of fever.

**Treatment.**—If the diarrhœa is due to the presence of irritating substances in the bowel, a dose of castor-oil and laudanum may suffice to arrest it. The diet must be carefully regulated, and in acute cases, sedatives such as Dover's powder may be given. In chronic cases, astringents are of more service, catechu and chalk, dilute sulphuric acid, bismuth, and the lead and opium pill being all of them useful. Rest in bed may be necessary at the beginning of the treatment, and warmth to the abdomen is serviceable, an abdominal belt being valuable where the diarrhœa depends upon changes of temperature. In diarrhœa due to intestinal putrefaction, douching the colon is an effective measure, and in the form which comes on immediately after meals, small doses (1-2 minims) of liquor arsenicalis, combined with a carminative, should be given just *before* food.

Diarrhœa in young children or infants, if not associated with other diseases, such as enteritis, tuberculous affections, or rickets, is nearly always due either to improper feeding or to irritation from worms, in which case anthelmintic treatment is indicated.

## CONSTIPATION

To ensure the due evacuation of the bowels, the digestive functions of the stomach and small intestine, and the secretion



of bile and pancreatic juice, must all be in proper working order; the colon must absorb some of the water from the fluid fæces, rendering them of a proper consistency for expulsion; and the intestinal musculature must be in a healthy condition. Evidently, then, constipation may be brought about by interference with any of these functions, and hence may be due to a large variety of causes. The chief are—

1. Causes producing atony of the intestinal musculature: (a) general diseases such as anæmia, the specific fevers, chronic Bright's disease; (b) nervous diseases, such as neurasthenia, paraplegia, chronic brain disease; (c) sedentary habits.

2. A diet which does not sufficiently stimulate the bowel. There may not be enough food, or it may be too dry, or it may not cause sufficient mechanical irritation (defect of vegetable matters). The fæces may become dry through loss of water by other channels (diabetes, granular kidney, vomiting, perspiration).

3. Mechanical obstacles, such as ptosis of the bowel, or "kinks" due to bands stretching between the bowel and the abdominal wall, and giving rise to "intestinal stasis." Whether this condition is responsible for the manifold symptoms attributed to it is open to grave doubt.

4. Inhibition of the reflex action of the bowel owing to painful defæcation or abdominal pain.

5. Neglect of the daily call to defæcation, leading gradually to extinction of the impulse.

**Morbid Anatomy.**—The changes in the gut are few. There may be hypertrophy of the muscular coat of the descending colon, and small ulcers in the cæcum. In extreme cases there may be enormous distension of the whole colon.

**Symptoms** are sometimes practically absent, but usually there are headache, anorexia, depression, and loss of energy. Where the lower bowel is much loaded, there may be *pressure* on the lumbar or sacral nerves, and pain down the back or front of the left thigh. Pressure on the intrapelvic veins may cause hæmorrhoids or varicocele.

Fæcal masses may sometimes be felt by palpation through the abdominal wall, or rectal examination may reveal the presence of hard scybala.

**Treatment.**—A daily attempt at evacuation should be made at a fixed hour, regular exercise must be taken, and any constitutional fault must be corrected. Add laxative vegetable



foods to the diet. Give a tumblerful of water before breakfast, and an occasional laxative. Even in severer cases *avoid purgation*, but use a mild laxative habitually. The following is an excellent pill :—

R Aloin . . . . .	gr. $\frac{1}{4}$ (grm. 0·016).
Strychninæ . . . . .	gr. $\frac{1}{60}$ (grm. 0·001).
Ext. Belladonnæ . . . . .	gr. $\frac{1}{12}$ (grm. 0·004).
Ext. Cascaræ (Ext. Rhamni) . . . . .	gr. ss. (grm. 0·032).

Sig.—One pill thrice daily.

or give aperient waters in the morning. Liquid paraffin,  $\text{Zii}$ – $\text{Ziv}$  (cc. 8·0–15·0) night and morning, is now much used, and suits very well in some cases.

If the rectum is blocked by scybala, these must be removed mechanically, and soap and water enemata given daily for some time afterwards.



## DISEASES DUE TO INTERNAL PARASITES

WITH the exception of the protozoal organisms already described in connection with specific infectious diseases, nearly all the internal parasites of man have their habitat in the intestine during part of their life-history, and are therefore appropriately described in this place. Of some, man is the *host*, of others, the *intermediate host*; the host being that animal in which the parasite passes the mature stage of its existence, the intermediate host the animal in which the larval or embryonic stage is passed. The parasites belong to the order *Vermes*, and are divided into three genera, *Cestoda* (tapeworms), *Nematoda* (round worms), and *Trematoda* (flukes).

### CESTODA

(Κεστός—a girdle)

The Cestoda are all endoparasitic worms, and infest the intestinal canal of vertebrata.

**General Characters.**—The Cestodes are multiple in character. The tapeworm is not a single individual, but a multitude of organisms arranged in a chain, thus forming a compound jointed colony.

1. *Shape.*—The Cestoda are compound, flat, parasitic worms.
2. *Size.*—Varies much; some forms measure a quarter of an inch, others twenty-four feet in length.
3. *Structure.*—The adult worm or Strobila consists of a number of complete sexual individuals arranged in a chain.

We have—

(1) The Head or Nurse, which is usually small in size, pyriform in shape, and has one or two suckers surrounded by a ring of chitinous hooklets, to enable the worm to cling to the intestines of its host. It has neither an alimentary system nor sexual organs.

(2) The Proglottides.—These are a series of segments produced one behind the other by a process of budding from the



head or nurse. Each segment or proglottis resembles its neighbours except in size and degree of maturity. The segments farthest from the head are the oldest, the largest, and most mature; the segments next the head being immature and having no sexual organs. In an ordinary tapeworm there may be as many as 1200 of these segments. Each proglottis has a complete water-vascular system, composed of parallel canals running on each side of the body, and united at the hinder end of each segment by cross branches. The proglottides have no digestive organs of any kind, being nourished by imbibition. They are hermaphrodite, and ova are formed by sexual union within the proglottides. A single proglottis may contain as many as 35,000 eggs.

**Life History.**—As above stated, the ova of the cestoda are produced in the proglottides, which when ripe break off from the rest of the chain and are cast out by the body of their host. Within these ripe proglottides the ova are already partially developed, and when ejected are full of active embryos. These embryos are enclosed in a membrane to protect them from injury, and consist of a head furnished with three pairs of silicious spines or hooklets. By the decomposition of the proglottides the embryo-bearing ova are set free, reach water, and thence find their way into the stomach and intestines of their host, where the membrane enclosing the embryo is digested, and the embryos are liberated. They are called PROSCOLICES (*scolex*, a worm), and consist of a small vesicle with three pairs of silicious spines. By means of these hooklets the proscolex fastens itself to the intestinal wall, bores through it, and makes its way to the liver or other organ of its host. Here it becomes encysted, loses its hooks, and from its hinder end develops a small vesicle full of fluid.

It is now called a SCOLEX, and in some tæniada the scolices are known as *hydatids*, in others as *cysticerci*. When thus encysted, the scolex is composed of a vesicle united by a narrow neck to a head similar to that of the adult tapeworm, being armed with a circlet of hooklets, and having four oscula or suckers. It has no reproductive system, nor, in fact, organs of any kind, and can undergo no further development unless it gains entrance into the intestinal canal of man or other host. This is effected by an animal eating flesh, etc., containing the scolices, when the cysts are digested and the scolices set free. They at once lose their caudal vesicle, attach themselves to the intestinal wall of their host but do not perforate it, and in this situation soon



become the head of the future tapeworm and begin to produce proglottides, which again pass through the cycle of development above described. Thus we have—

1. The *Ova* discharged from the ripe proglottis.
2. The *Prosclex*.—The minute embryo liberated from the ova when taken up from water, etc., by some animal.
3. The *Scolex*.—The more advanced, but still sexually immature embryo into which the prosclex develops when it has become encysted in the tissues.
4. The *Strobila* or adult tapeworm, infesting the alimentary canal of its host, and composed of a head, neck, and proglottides.

**Division.**—The Cestodes which infest man are—

1. *Tæniada*—
  - (1) *Tænia solium*.
  - (2) *Tænia mediocanellata*.
  - (3) *Tænia echinococcus*.
2. *Bothriocephalida*.—*Bothriocephalus latus*.

### I. TÆNIA SOLIUM

*Synonyms*—*Tænia cucurbitina*, *Tænia humana armata*, *Tænia vulgaris*.

*Larva*—Simple scolex, Measle, *Cysticercus cellulosæ*.

**General Characters.**—The adult worm or strobila measures about two to ten feet in length. It has a small head, long narrow neck, and transversely segmented body.

The *Head* is small, rounded, about the size of a pinhead, and consists of a rostellum or beak with twenty-six hooklets, and of a wider part on which are four suckers.

*Body.*—Next the head comes a long, narrow, thread-like neck, followed by a series of larger segments—the proglottides. The first segments are broader than they are long, and are immature. The remaining segments are the reverse, longer than broad, and are sexually mature proglottides. These proglottides are hermaphrodite, the genital orifices being placed alternately on each side of the body, and the male and female organs open by this common genital pore. They have a complete water-vascular system.

The *Uterus* consists of a central stem, with seven to ten lateral branches on either side, each of which again branches freely. The testes appear as clear, white, convoluted tubes, with vesicles.



The *Ova* are nearly spherical in shape, about  $\frac{1}{750}$  inch (0.03 mm.) in diameter, and are surrounded by a dense capsule which encloses the partly-developed, six-hooked embryos. In the flesh of the pig, these embryos give rise to the scolices, which are known as "measles," cysticerci, or bladder-worms.

**Life History.**—As above described.

*Intermediate Host.*—The pig, which gets the embryos from water or from garbage—the scolices forming the "measles" of pork. Within these measles or cysts, the hooklets, which do not decompose, are often found after the scolex has perished. They are short, broad, hook-shaped bodies, with a small knob at their base.

*Host.*—Man; owing to eating imperfectly cooked, measly pork. The parasite infests man, not only as the mature worm, but as cysticerci. The adult worm is found in man only.

*Habitat.*—The *immature* worm is found in the subcutaneous tissues, muscle, brain, eye, and liver of the pig; the *mature* form, in the small intestine of man.

## II. TÆNIA MEDIOCANELLATA

*Synonyms*—*Tænia saginata*, *Tænia dentata*, *Tænia inermis*,  
Beef tapeworm.

*Larva*—*Cysticercus bovis*.

**General Characters.**—This tapeworm is larger than *tænia solium* both in length and breadth, and often measures from fourteen to twenty-four feet. It is commoner in Great Britain than *tænia solium*.

The *Head* has four suckers, but no rostellum nor hooklets. Following the head is a narrow neck, and then the several segments or proglottides.

The *Uterus* has twenty to thirty lateral processes on either side, and these divide only once at their extremities, a character by which the proglottis can be distinguished from that of the *tænia solium*.

*Ova.*—Similar to those of *tænia solium*.

**Life History.**—Similar to that of *tænia solium*.

*Intermediate Host.*—Cattle.

*Host.*—Man.

*Habitat.*—Immature form, in the muscles of cattle; as many



as 300 have been found in a pound of flesh taken from psoas muscles.

The *mature* form occurs in the intestine of man.

### III. BOTHRIOCEPHALUS LATUS

*Synonyms*—Broad tapeworm, *Tænia lata*, *Tænia grisea*,  
*Dibothrium latum*.

**General Characters.**—This is the largest human tape-worm. It measures from sixteen to thirty feet long, and about one inch broad, and consists of three or four thousand segments. It occurs in Switzerland, North-eastern Europe, and Japan.

The *Head* is small, oval, or club-shaped, with a longitudinal groove or slit on each side. It has no proboscis, nor suckers, nor hooklets.

The *Proglottides* are about 4000 in number, the largest being in the middle of the chain. They are each bi-sexual. The uterus consists of a simple, coiled-up tube, and the genital orifices are placed along the middle line of the ventral aspect—not on the sides, as in the last group.

The *Ova* are oval in shape, and about  $\frac{1}{350}$  of an inch (0·07 mm.) long. They have an operculum and a brown-coloured shell.

**Life History.**—The proglottides are passed not singly but in strings, and the ova do not develop embryos in the uterus, but only on reaching fresh water. Thereafter the embryos burst the operculum, and swim about by means of cilia until they are swallowed by a fresh-water fish, the muscles of which they reach by their six hooklets, and there develop into the asexual larval worm.

If the fish be eaten by man, the larva develops into the sexual form above described.

*Intermediate Host.*—Certain fish, as pike, eels, etc.

*Host.*—Man, dog.

*Habitat.*—Intestinal canal.

**Symptoms due to Tapeworm.**—In many instances there are no symptoms, and not one that may occur is diagnostic; but certain reflex disturbances are common, such as itching of the nose or anus, colicky pains, attacks of diarrhoea, voracious appetite, mental trouble such as melancholia, convulsions, and occasionally reflex vomiting. Symptoms suggestive of stone in the bladder have



been known to arise in a case dependent on reflex irritation from tapeworm. If the above symptoms are present without any obvious cause or reason, *always examine the fæces for segments of these worms*. A discharge of the segments is of course conclusive.

The *cysticercus cellulosæ* is rare in man. It has been found in the brain, the muscles, and the eye. In the first, it may or may not cause cerebral symptoms, according to the locality it attacks; in the second, it occasionally produces rheumatoid pains; in the last it may be recognised by the op. thalmoscope.

The symptoms due to bothriocephalus differ from those produced by the tæniæ. While it may cause similar vague disturbances, the characteristic feature is that it almost always gives rise to a severe anæmia, closely resembling pernicious anæmia (*q.v.*) both clinically and in the character of the blood. The diagnosis depends upon the recognition of the cause. This is usually easy, since the segments of bothriocephalus are passed in strings often several feet long.

**Treatment.**—The liquid extract of male fern is the most appropriate anthelmintic, but to be effective it must reach the parasite, and especially the head, which is often protected by mucus or by the valvulæ conniventes. The patient is therefore kept on fluid diet for twenty-four hours, or even for two or three days, and a saline purgative is given every morning.

At the end of this treatment the male fern is administered on an empty stomach; half a drachm (2 cc.) may be safely given to a child, one to two drachms (4–8 cc.) to an adult. It is most easily taken in capsules, each containing 15 minims (1 cc.), one being given every ten minutes till the required dose is attained. The patient should lie quiet for some three hours to avoid nausea. A brisk purge is then given, and the worm is expelled. The dejecta must be carefully examined for the head, since if it remains behind, the worm will grow again.

Other vermifuges are pomegranate root and its alkaloid pelletierin, pumpkin seeds, and turpentine.

#### IV. TÆNIA ECHINOCOCCUS

**General Characters.**—*Tænia echinococcus* is a small worm about an eighth to a quarter of an inch long. It consists of only four segments, including the head. The *Head* is pointed, has four suckers, and a double circlet of hooks. These hooks are about thirty to forty in number, and are shaped like those of *tænia*



solium, but are much smaller. The last proglottis, when mature, is equal in size to the rest of the body, and contains the reproductive organs. The genital pore is placed on the lateral aspect of the body. The ovaries are complicated, and the ova are small but not exceedingly numerous, and in them are developed the six-hooked embryos.

**Life History.**—When ripe the eggs set free from the intestine of the host—the dog or wolf—may be swallowed in drinking water or on vegetable food, and thus reach the stomach of man. There the embryos are liberated, perforate the intestinal walls, and getting into the circulation, are by this or other means carried to the liver or other organ, where they become encysted and develop a spherical vesicle which may reach a great size. They are now called HYDATIDS.

**Hydatid Cysts** are much larger than those of *cysticercus cellulosæ*, and are composed of three parts—

(1) The false cyst—a fibrous capsule formed by the tissues of the part.

(2) The ectocyst—an opaque, chitinous membrane of great thickness, white in colour, smooth, glistening and laminated.

(3) The endocyst—a more opaque, granular layer, composed of nucleated cells, and covered by small white spots—brood capsules.

Inside the cyst there is a colourless watery fluid which contains salts, but no albumin, a point of value in diagnosis. Within these cysts the scolices—*echinococcus* heads—are developed in the following manner:—

On the inner wall of the cyst are formed small vesicles—*brood capsules*—which project into the cavity of the cyst. From the walls of these brood capsules small cup-like buds or hollows are formed, each of which gradually elongates and becomes a *cæcum* with its cavity opening outwards—*i.e.* it communicates with the cavity of the brood capsule. Within these depressions or hollow buds the *echinococcus* head is developed, and this, when mature, is everted, so that the head now projects into the brood capsule. These heads are similar to those of the adult worm, having a double circlet of hooklets and four suckers.

Development cannot proceed further than this in the human body, but if the cysts gain access to the dog, etc., then the adult tapeworm is formed in the intestine.



SECONDARY CYSTS are often found in connection with the primary cyst. This may occur in one of three modes—

1. By a process of budding out of the wall of the ectocyst, thus giving rise to a number of daughter-cysts, side by side—*exogenous cysts*.

2. Again, the daughter-cysts may be formed inside the primary cyst—these are called *endogenous cysts*.

3. Or, the cysts may be *multilocular*, i.e. composed of many separate alveoli divided from each other by dense fibrous tissue. They occur as hard, firm tumours in the liver.

Daughter-cysts may in turn give rise to *grand-daughter-cysts*, and thus the original mother-cyst may come to contain enormous numbers of smaller cysts of varying size.

*Intermediate Host*.—The cystic form alone is found in man.

*Host*.—The adult worm occurs in the dog, wolf, and jackal.

*Habitat*.—The *cystic* form is most common in the liver, but is also found in the kidneys, lungs, brain, heart, and muscle; the *mature* form, in the intestine.

**Diagnosis and Treatment**.—See Hydatid Disease of the Liver.

## NEMATODA

(Νῆμα—a thread)

*Synonym*.—Roundworms.

**General Characters**.—The roundworms are a very large and well-known group of helminths. They are simple, not compound, and do not form colonies. They are round or thread-like and unsegmented. They undergo no metamorphosis, the sexes are distinct, and there is a marked difference between the male and female—the male being the smaller.

**STRUCTURE**.—The Nematoda have a distinct alimentary canal with a mouth, œsophagus, stomach, intestine, and anus. There is a thick elastic ectoderm or cuticle and a well-developed muscular system. The genital pore, placed on the ventral aspect, is, in the female, situated about the middle of its length; in the male, near the anus, where there is a chitinous prehensile investment.

The most common Nematoda are—

1. *Trichina spiralis*; 2. *filaria sanguinis hominis*; 3. *filaria*



medinensis; 4. dochmius duodenalis or ankylostomum duodenale; 5. ascaris lumbricoides; 6. trichocephalus dispar; 7. oxyuris vermicularis.

# I. TRICHINA SPIRALIS

*Synonyms.*—Trichinella spiralis, Flesh-worm, Pseudalius trichina.

*Larva.*—Muscle trichinæ, Encysted trichinæ, Flesh-worms.

**General Characters.**—Trichina spiralis is a very minute worm, the male and female being distinct. The male is about  $\frac{1}{18}$  inch, and the female one-eighth inch in length.

The *Head* is narrow, pointed, unarmed, with a simple central oval aperture.

The *Body* is thread-like, bent upon itself, thicker behind than in front, and in both male and female the hinder part of the body is straight. In the male, however, it has a short, bilobed caudal appendage, between the lobes of which is the anus. The testes are convoluted tubes. The female is about one-eighth of an inch long, rounder and shorter behind than the male. There are an ovary, vagina, and uterus, and the genital orifice is near the head.

The *Ova* are  $\frac{1}{170}$  inch (0.15 mm.) long, and are hatched within the parent (ovoviviparous).

*The Larval Form*—the trichina of muscle—is a very small worm, about  $\frac{1}{30}$  inch long, coiled up in a spiral manner within a fibrous capsule or cyst, the long axis of which lies in the long axis of the muscular bundles. A single capsule may contain two or more larvæ, and there may be as many as 325,000 of these capsules in an ounce of meat. They are especially common in the abdominal and thoracic muscles, and appear as whitish spots, owing to calcification of the cyst towards the poles. This small worm has a digestive system and an imperfect sexual apparatus.

**Life History.**—When a piece of meat affected with trichinæ is eaten, the capsules are dissolved, and the embryo parasites which they contain are liberated. These mature in a day or two in the intestinal canal of the host. Embryos are formed within the ova within seven days of ingestion; and a single worm may produce as many as 1000. The fertilised females penetrate the intestinal villi, and the embryos are deposited in the lymphatics or lacteals, whence they are carried to the striped muscles.

Once in the muscles the embryos penetrate the primitive



bundles, reduce their contents to débris, and soon become mature muscular trichinæ, forming cysts, part of which is made up of a chitinous secretion of the parasite, part of a wall of fibrous tissue formed of the perimysium of the muscle bundles. These cysts, as above stated, may become partly calcareous, giving rise to the white shining spots in the muscle. They may remain quiescent for years.

*Intermediate Host.*—The trichinæ are found in pigs, rabbits, sheep, dogs, rats, mice, and other animals. The pig acquires them through eating dead rats, or feeding on trichinised offal.

*Host.*—Man, the trichinæ entering the body in uncooked or undercooked pork.

*Habitat.*—The adult worm inhabits the intestine, and lives for only a few weeks.

**Symptoms of Trichiniasis.**—Unless a large number of embryos are eaten, definite symptoms may not arise; but in well-marked cases of trichiniasis the symptoms are very characteristic. A few hours or days after eating the infected flesh symptoms of gastro-intestinal irritation appear; there may be vomiting, diarrhœa, and abdominal pain.

Towards the second week great soreness and stiffness of muscles develop, the temperature runs up, and may be remarkably remittent in character, and a characteristic œdema sets in. It begins in the face and spreads to the skin over the affected muscles. The respiratory muscles are implicated, and there may be intense dyspnœa. In protracted cases the patient becomes emaciated and exhausted, and a typhoid condition may supervene and end in death. In mild cases the symptoms subside in about a fortnight. There is marked leucocytosis, the eosinophil cells being especially preponderant (*eosinophilia*). The disease sometimes occurs in epidemics.

**Diagnosis.**—This is easy in the course of an epidemic, the characteristic features being—1. marked muscular pain; 2. œdema, beginning in the face; 3. dyspnœa; 4. eosinophilia.

The stools should be examined for adult worms, and the food for encysted embryos. If necessary, a piece of muscle may be "harpooned" and examined. During the second and third week the embryos may also be found in the blood by treating it with ten volumes of three per cent. acetic acid, and examining with a low power.

**Treatment.**—Prophylactic measures consist in efficient food



inspection and thorough cooking. The bowels and stomach should be cleared at the outset by a purge (calomel gr. v.—grm. 7·3) and an emetic, and the bowels should be kept open by repeating the purge every few days. Large doses of glycerin are said to destroy the worms by dehydrating them, but ordinary vermifuges are without effect. When the embryos have reached the muscles, they are unaffected by drugs, and all that can be done is to treat symptoms.

## II. ASCARIS LUMBRICOIDES

*Synonyms.*—Roundworm, *Lumbricus teres hominis*.

**General Characters.**—These parasites closely resemble the common earth-worm. The male measures about four to six inches in length, the female, ten to sixteen inches. The parasite is a broad, smooth, fusiform, translucent, brownish or pink coloured worm, with fine circular striæ. Its anterior extremity has a three-lobed mouth. The tail is bluntly curved in the male, and has a double spicule near its end. The numerous ova are oval in shape,  $\frac{1}{500}$  to  $\frac{1}{150}$  inch (average 0·07 mm.) in diameter, and have a hard shell and albuminous envelope. The embryo developed from them, probably in wells or pools, reaches the human intestine in polluted drinking-water, and there becomes an adult worm. An intermediate host is not necessary. One or two parasites are usually present, but there may be many.

The *habitat* of the worm is the small intestine, but it has a marked tendency to wander. It may pass into the stomach and be vomited, or into the colon and be voided. It is often found in the nasal passages or pharynx, sometimes in the larynx or trachea. It may block the bile-duct, causing jaundice; it may perforate an intestinal ulcer; and a knot of ascarides may cause intestinal obstruction.

**Symptoms.**—These are very indefinite and may be absent. Picking of the nose, grinding the teeth, foul breath, etc., are popularly attributed to the presence of this worm, but obviously any irritation of the intestinal canal may cause such symptoms. Febrile or nervous phenomena, suggesting enteric fever or meningitis, are regarded by some authorities as due to the toxins of the ascaris. Eosinophilia is usually present. Other symptoms may arise from the wandering habit of the worm.

**Treatment.**—Santonin, followed by purging, is the best remedy. It should be repeated for three or four nights in doses



varying from gr.  $\frac{1}{2}$  (grm. 0.03) for a child, to as much as grs. v (grm. 0.3) for an adult. Temporary yellow vision (xanthopsy) may follow its use, and it colours the urine bright yellow.

### III. OXYURIS VERMICULARIS

*Synonym*—Threadworm.

**Characters.**—The female is thread-like, with a tapering tail, and is three-eighths of an inch long. The male is one-eighth of an inch long, and the tail is curled. The numerous ova, each containing a partly formed embryo, are expelled with the fæces. The embryos develop fully when the ova are taken into the stomach, becoming adult in the small intestine. After conjunction the male dies, and the impregnated female passes into the colon and rectum. The worms often wriggle out at the anus, causing irritation; the patient scratches, and may thus transfer ova on his fingers to his mouth, and so re-infect himself.

**Symptoms.**—Presence of the parasite in the stools, irritation about the anus and genitals, and consequent sleeplessness.

**Treatment.**—Clear out the lower bowel by enema, then inject by the long rectal tube Inf. Quassiae, salt solution ʒij to Oj (grm. 13 to the litre), or tincture of ferric chloride ʒij to Oj. From ten to twenty ounces may be introduced, and retained as long as possible. Hands, nails, and perineum must be kept very clean, and carbolic lotion applied daily. In obstinate cases santonin and purgatives may be used.

### IV. ANKYLOSTOMA DUODENALE

A short, white, cylindrical worm, found in the upper part of the small intestine. The female is half an inch, the male one-third of an inch long. The mouth has four strong ventral hooks and two conical teeth. The ova, which are very numerous, and may be found in the fæces, are of oval shape, and about 0.05 mm. in length. The larval stage is passed in water or damp earth. The larvæ enter the human body by penetrating the skin. They are carried by the lymphatics from the subcutaneous tissues to the lungs and bronchi, whence they are coughed up, and being swallowed with the sputum reach their habitat in the intestine. The parasite is common in the tropics, especially Egypt, but is found also in Europe, and sometimes in this country, principally among miners. Besides ankylostoma, another species, *uncinaria* or



*necator americanus*, distinguished by having a prominent dorsal median tooth, is found in the Southern States of America and in Porto Rico, and causes similar symptoms known as *uncinariasis* or *hookworm disease*.

**Symptoms.**—The worm sucks blood from the intestinal mucosa. If there are many of them, this may lead to profound anæmia of the chlorotic type, epigastric pain, diarrhœa, dyspnœa, and dropsy. The symptoms are at least partly produced by bacterial infection of the wounded mucosa, and by toxins derived from the worms. Eosinophilia is pronounced. Severe cases end in death, and many of the workers in the St. Gotthard tunnel died of ankylostomiasis.

An eruption, characterised by the presence of papules, pustules, and wheals, occurs on the hands and arms when these are constantly exposed to the larvæ, as in the case of the Cornish miners, to whom the condition is known as “bunches.”

**Treatment.**—All drinking-water should be boiled in districts where the parasite is found. Keep the patient on fluid food for a few days, then give 20 to 30 grains (grm. 1·3–2·0) of thymol in cachet, and repeat two hours afterwards. Give a purge three hours later. After it has acted the patient may return to more solid food. Avoid alcohol, as it dissolves thymol, and may cause poisoning. Repeat the treatment once a week, so long as ova are found in the fæces. Beta-naphthol in doses of 60 grains (grm. 4·0), once or twice repeated, is said to be as effective, and much less depressant than thymol. Subsequently iron is needed for the anæmia.

*Trichocephalus dispar* (the whip-worm) inhabits the cæcum. It is about an inch long, and the anterior half of the parasite is thin and thread-like. It rarely causes symptoms.

## V. FILARIA SANGUINIS HOMINIS

Several species of nematodes occurring in tropical or sub-tropical countries give rise to embryonic forms which circulate in the blood, and present such close resemblances that they are included in the one designation of *filaria sanguinis hominis*. The most important adult form is—

*Filaria Bancrofti*, which is a long, thin, white organism, resembling a horse-hair, and inhabiting the lymphatics. The female is about three inches, the male one and a half inches long. The parasite is found in warm climates, and causes the various condi-



tions mentioned below. The embryos pass by the lymph stream to the blood. They are found in the peripheral circulation only at night, and hence are known as *filaria nocturna*. In the daytime they are found in the large vessels of the lungs and thorax.

The *filaria nocturna* is about  $\frac{1}{10}$  of an inch long. It has a rounded head and tapering tail, and is enclosed in a fine sheath. It is sucked up by the female mosquito from the infected blood. In the stomach of the mosquito it casts its sheath, becomes actively mobile, and passes into the thoracic muscles. There it grows for about a fortnight, again becomes mobile, and then passes to the base of the proboscis, from which it enters the blood of the next person bitten, and completes its development in the lymphatic vessels of man, where the sexes unite, and give rise to a fresh brood of embryos. The embryos do not cause symptoms, which are due to the blocking of the lymphatics either by the mature worm or by the *ova*, these being thicker than the full-grown embryos.

Other less important forms are *filaria diurna*, which is found in the peripheral blood by day, the adult form being probably *filaria loa*, and *filaria perstans*, which is present throughout the twenty-four hours. Its adult form is not known.

**Symptoms.**—When the parent worm aborts, ova instead of embryos enter the lymph stream, and block the vessels of the part where the parent is situated. The adult worm itself may block the thoracic duct. From one or other of these causes chyluria, lymph-scrotum, or elephantiasis results.

*Chyluria* is the passage of milky urine, sometimes slightly stained with blood, owing to the rupture of obstructed lymphatics in the urinary tract. The urine, which contains filariæ, coagulates on standing. It is albuminous, and contains fat granules. The condition is intermittent. In *lymph-scrotum* the scrotal tissues are greatly thickened, and the lymphatics are prominent and may rupture, allowing chyle to flow over the surface. Inflammatory complications are common. Tropical *elephantiasis* is also due to obstruction of the lymphatics by the filariæ, although the parasites often cannot be detected.

**Treatment.**—Beyond rest, dry diet and avoidance of excessive fat, there is no satisfactory treatment for chyluria. The treatment of lymph-scrotum and elephantiasis is chiefly surgical.

*Filaria* or *dracunculus medinensis* (the Guinea worm) is met



with in West Africa and the East Indies. The embryo inhabits the cyclops, a small fresh-water crustacean, and being swallowed, the impregnated female passes through the intestine to the subcutaneous tissues, in which it travels downwards to the foot or ankle, where it creates an ulcer through which the embryos are discharged. The adult worm is about  $\frac{1}{12}$  of an inch thick, and may be as much as three feet long.

## TREMATODA

(Τρῆμα—a hole)

*Synonyms*—Flukes, Flat-worms, Suctorial-worms.

The only parasite under this class that we will consider is—

### BILHARZIA HÆMATOBIA

*Synonyms*—Distomum Hæmatobium, Thecosomum, Schistosomum, Gynæcophorus.

**General Characters.**—The male is about half an inch long and is cylindrical in shape, with a canal or groove—the gynæcophoric canal—at the posterior end of the body, in which the female is lodged. The female is four-fifths of an inch long, and thread-like. The ova are oval in form,  $\frac{1}{180}$  to  $\frac{1}{160}$  inch (0·14–0·16 mm.) in length, with a spine at one end or one side of each egg. When they are discharged into the water of small ponds or canals along with the urine or fæces of an infected patient the ciliated embryos are liberated and enter the body of a fresh-water mollusc, in which they assume a larval form capable of piercing the human skin, through which they enter the blood. Bilharziosis may thus be conveyed by bathing or even washing in such pools.

**Habitat.**—The blood. The parasites are especially found in the inferior vena cava and portal veins, and in the vesical and hæmorrhoidal vein.

**Effects.**—The sexes unite in the blood, and the ova penetrate the vessel-walls and the walls of the bladder, ureter, and renal pelvis. If in large numbers, they give rise to cystitis, vesical papillomatosis, pyelitis or pyonephrosis, with hæmorrhage from the affected mucous membrane. In the colon and rectum they cause colitis and the formation of polypoid adenopapillomata. Hæmaturia is the chief symptom, and, if the bowel is affected, a mucous diarrhœa. Anæmia follows. Eosinophilia is marked.



Renal or vesical calculi may form around the ova.

The parasite is rarely met with in England, but is common in Egypt, the Cape, Natal, and also in Arabia.

**Treatment** consists in an occasional course of extract of male fern, but the results are only partially satisfactory. The recent discovery of the life-history of the parasite would point to drying up the small pools as the best means of prophylaxis.



## DISEASES OF THE LIVER

BEFORE attempting the study of diseases of the liver, it is advisable to recall to mind the chief facts regarding its function. It should also be remembered that the kidney is to a large extent the handmaid of the liver, excreting the waste material which it forms, and thus enabling it to perform its duties efficiently, and to keep the blood free from morbid products. Disease of the liver may thus alter the composition of the urinary secretion, and disease of the kidney, resulting in imperfect elimination, may prejudice the functional activity of the liver.

### FUNCTIONS OF THE LIVER

These will be considered briefly under three heads—

1. The metabolism of carbohydrates.
2. The metabolism of proteid material.
3. Bile formation.

#### I. METABOLISM OF CARBOHYDRATES

By the action of saliva (ptyalin) starch is converted into achroodextrin and erythroextrin, and these partially into maltose. Inhibited by the acid gastric juice, the process of conversion is resumed by the pancreatic juice (amylase) and succus entericus (invertin), the maltose being transformed into dextrose and to a lesser degree into lævulose. On reaching the liver through the portal circulation, these bodies are transformed into glycogen. According to the bodily requirements the glycogen is once more converted into dextrose, and passes into the general circulation, by which it is distributed to all the tissues. There, and particularly in the muscles, it is oxidised to its end products, water and carbon dioxide, the process being accompanied by the production of heat and energy.

Carbohydrates, therefore, after passing through various changes, get to the liver as grape sugar. In the liver a portion is stored up for a time, *not as sugar*, but as a *peculiar starch, termed glycogen*. The amount of glycogen which can be obtained



by extraction from the liver varies with the nature of the diet. Working with dried dogs' livers Pavy found the following results:—after animal food, 7·19 per cent.; after animal food with sugar, 14·15 per cent.; after a vegetable diet, 17·23 per cent. Thus the formation of glycogen is favoured by a free supply of carbohydrates, dextrose in particular, though lævulose is also used. But the liver also forms glycogen from proteid, though not to the same extent. It forms no glycogen from fat.

What becomes of the glycogen? The statements made above embody the views of Bernard, who considered that the liver simply formed it and kept it stored up, to prevent flooding of the organism with dextrose, and gave it out again to the tissues, retransformed into dextrose, when the amount of sugar in the blood had fallen below a definite minimum. Pavy's view, as we have already seen (p. 134), was that dextrose is not used in the body as such, but combined with proteids, and that the liver simply stores the sugar as glycogen until it has time to synthesise it into a form suitable for the tissues. Extensive formation of dextrose from glycogen is thus pathological, and only occurs in diseases such as diabetes or temporary glycosuria.

## II. PROTEID METABOLISM

The action of the various digestive juices upon proteids may thus be briefly stated:—Saliva aids in mastication, and so prepares them for digestion; the gastric juice converts them into acid albumin, proteose, and peptone; and the pancreatic juice and succus entericus form alkali albumin, proteose and peptone. The end-product, peptone, which cannot be absorbed as such, is broken up by the trypsin and erepsin of the pancreatic and intestinal juices into the series of aminoacids which form the component parts of the proteid molecule. The series is made up of monoamines, diamines, and aromatic amines, groups which are represented by such bodies as leucin, glycin, arginin, lysin, tyrosin, and tryptophane. If putrefactive changes have occurred, as they may do under bacterial influences when there is constipation, indol and skatol may also be present. The greater part of the proteids derived from food passes as aminoacids through the liver into the general circulation, to be used by the tissue proteins, and ultimately transformed into nitrogenous waste products (ammoniacal salts). But if the diet be purely nitrogenous, part of the food proteids may be split up, the non-nitrogenous portion of the proteid molecule



being synthesised into glycogen, and the remainder of it forming urea.

The ultimate result of the action of the liver in dealing with nitrogenous waste products, such as excess of aminoacids or the ammoniacal salts derived from muscle, is the formation of urea. Possibly some small proportion of urea is derived from the nitrogenous waste of every body tissue, but the main source of its formation is undoubtedly the liver. If the liver of a frog be removed, the formation of urea almost ceases. In mammals, if the liver be thrown out of action by joining the portal vein to the inferior cava, urea is much diminished. In both cases its place is taken by ammonia : in both, if a proteid meal be given, ammonia poisoning appears, and excess of ammonia is found in the blood. In birds, uric acid is the chief nitrogenous excretion in the urine. In them extirpation of the liver means disappearance of uric acid and appearance of ammonia. The liver therefore forms urea from the ammonium compounds circulating in the blood, whether these be the product of tissue waste or represent the excess of aminoacids in the proteid constituents of the food.

Compare these experimental results with the results of clinical examination of the urine in diseases of the liver. In cirrhosis there is marked diminution in the amount of urea, and increase of ammonia. In acute yellow atrophy, and also in phosphorus poisoning, the urea is enormously diminished, and leucin, tyrosin, and ammonia take its place. *Failure on the part of the liver to do its work results in imperfect oxidation of waste products*, the nature of the products so formed depending on the degree of oxidation. Comparatively slight defect may be attended by excess of uric acid (gout). If the defect be greater, there is excess of ammonia and its compounds in the blood and urine, and where the hepatic function is almost abolished, leucin, tyrosin, etc., appear unchanged.

### III. BILE FORMATION

Bile consists principally of water, cholesterin, bile salts, bile pigments, and mucin. It is to be regarded as—

- (1) A secretion, which is concerned in the proper assimilation of fats, and favours intestinal peristalsis.
- (2) An *excretion*—a means by which broken-down blood pigments are excreted, with also a certain amount of proteid waste,



Bile pigments and bile salts are formed only in the liver; cholesterin, lecithin, etc., exist as such in the blood.

The bile pigments, however, are ultimately derived from hæmoglobin, which in the liver is converted into hæmatin, and hæmatin, after separation of its iron, into bilirubin. They appear in the fæces as stercobilin, and in the urine as urobilin (traces). As might be expected, an increased amount of bile pigment is present in diseases such as pernicious anæmia, which are attended with *destruction of red corpuscles*. Now bile pigment increases the thickness of bile, and, as Quincke showed, great destruction of red corpuscles in the liver is accompanied by increase of mucin, and this in turn materially increases the viscosity of bile. In such diseases, then, the bile is rendered thicker both by increase of pigment and by increase of mucin, and the increased viscosity produces a retarded flow and an increase of tension in the ducts. In this condition it is possible to find an explanation of the sallowness which accompanies so many anæmias.

The *secretion* of bile is continuous, but the rate of secretion varies. It is at its highest about four hours after a meal, and again rises about thirteen hours afterwards. It is but little accelerated by drugs, but it is increased by albumoses and bile salts. On the other hand, the *expulsion* of bile into the intestine is intermittent, occurring only when the acid chyme passes over the orifice of the common duct.

Bile is not an antiseptic. In obstructive jaundice the fæces become highly offensive, but this is due to slower peristalsis, which gives the intestinal bacteria a longer time to act.

## JAUNDICE

Jaundice is the name applied to a group of symptoms arising from the circulation of bile-pigment in the blood, and is clinically manifested by a yellow or greenish-yellow tint of the skin and mucous membranes. The retention of bile within the liver and its absorption into the blood give rise to two classes of symptoms.

1. *The absence of bile from the intestines* interferes largely with perfect assimilation of fat, delays absorption, and slows peristalsis. Consequently fat is frequently present in the stools, and putrefactive changes take place in the intestines, with formation of skatol and indol, to the absorption of which some of the toxæmic symptoms of obstructive jaundice may possibly be due.

2. *The circulation of bile within the blood* produces toxic



effects both upon nerve centres and upon muscular fibres ; consequently we get imperfect action of the heart, slowness of the pulse, depression of the spirits, and mental torpidity. Persistent jaundice may end in profound coma.

Two types of jaundice are described, namely, *obstructive* and *toxæmic* or *hæmolytic*. In the former case the seat of obstruction, in the majority of instances, is in the large ducts, outside the liver itself, and no bile enters the intestine. In cirrhosis of the liver the large ducts are not affected, but it is commonly supposed that catarrh in the smaller ducts, secondary to the structural changes in the liver, may account for the jaundice. Recent evidence, however, would seem to show that in this case the jaundice is really hæmolytic. In toxæmic or hæmolytic jaundice, which is due to the circulation in the blood of various poisons, bacterial or other, increased destruction of red blood corpuscles causes an increased supply of hæmoglobin to the liver, and hence, as we have seen, an increase in the amount of bile pigment, giving rise to increased viscosity of the bile. At the same time the toxic or infective process probably sets up an inflammation of the smaller intrahepatic bile-ducts (cholangitis), which narrows their calibre and excites a thick mucous secretion. As the bile is secreted under very low pressure, this leads to a much diminished rate of flow, which in combination with the increased viscosity produces a condition tantamount to incomplete obstruction ; incomplete since bile is still found in the stools, and the jaundice is usually slight. In both obstructive and hæmolytic jaundice the increased pressure within the bile-ducts forces the bile into the lymphatics, whence it is re-absorbed into the circulation. *Suppression of bile formation does not occur*, even in long-standing obstruction where the ducts contain only clear mucus. The hepatic cells continue to secrete bile, but it passes directly into the lymphatics, and thence into the blood. The rare cases of nervous jaundice, following on violent emotion, are probably due to obstruction, and are attributed to spasm of the hepatic ducts.

The following tabular statement of the causes of jaundice is modified from Murchison.

**A.—Jaundice from Mechanical Obstruction of the Bile-duct.**

- I. OBSTRUCTION BY FOREIGN BODIES WITHIN THE DUCT :—Gall-stones, hydatids and distomata, foreign bodies from the intestines.
- II. OBSTRUCTION BY INFLAMMATORY TUMEFACATION OF THE DUODENUM OR OF THE LINING MEMBRANE OF THE DUCT, WITH EXUDATION INTO ITS INTERIOR (“Catarrhal Jaundice”).



III. OBSTRUCTION BY STRICTURE OR OBLITERATION OF THE DUCT :—  
Congenital deficiency ; stricture due to perihepatitis, to cicatrisation of biliary ulcers, or to spasm ; and closure of the orifice through cicatrisation of a duodenal ulcer.

IV. OBSTRUCTION BY TUMOURS CLOSING THE ORIFICE OF THE DUCT, OR GROWING IN ITS INTERIOR.

V. OBSTRUCTION BY PRESSURE ON THE DUCT FROM WITHOUT BY :—  
Tumours projecting from the liver itself ; enlarged glands in the portal fissure ; tumours of the stomach, pancreas, kidney, omentum, ovary, or uterus ; abdominal aneurysm ; faecal accumulation ; the pregnant uterus.

#### B. Jaundice independent of Mechanical Obstruction of the Bile-duct.

POISONS IN THE BLOOD INTERFERING WITH THE NORMAL METAMORPHOSIS OF BILE (TOXÆMIC OR HÆMOLYTIC JAUNDICE).

1. The poisons of the various specific fevers :—yellow fever, malaria, relapsing fever, typhus, enteric fever, scarlatina, pyæmia, pneumonia.
2. Animal poisons :—snake-venom.
3. Chemical poisons :—phosphorus, mercury, arsenic, etc. ; toluylene-diamin ; chloroform or ether.
4. Poisons of obscure infective origin :—acute yellow atrophy of the liver, and Weil's disease (epidemic jaundice).

#### Symptoms of Obstructive Jaundice—

1. Icterus or tinting of the skin, conjunctivæ, mucous membranes, secretions, etc. The range of colour is considerable, from lemon-yellow to a deep greenish-black (black jaundice). The urine and sweat are tinted, the saliva, milk, and sputum usually escape. Like that of the skin, the colour of the urine ranges from saffron through greenish-yellow to dark brown. Yellow vision or xanthopsia is sometimes present.

2. Gastric disturbances, flatulence, nausea and often complete anorexia.

3. Constipation, often alternating with diarrhœa ; the fæces are pale, intensely foetid, and pasty in character.

4. Slowness of the pulse, which may fall sometimes as low as forty per minute.

5. Extravasation of blood, and hæmorrhages from mucous surfaces or into the skin. The coagulability of the blood is diminished.

6. Cerebral symptoms. There is marked depression of spirits, the patient "sees things with a jaundiced eye," melancholia may occur, and, in the graver forms, an assumption of the typhoid state may end in death.

7. Itchiness of the skin.

The tests for bile acids and bile pigments will be found under Examination of Urine.



**Treatment.**—As jaundice is merely symptomatic, the cause must be treated. In the milder forms a catarrh of the alimentary canal, or the presence of small biliary calculi, will require attention. For detailed treatment, see Hepatic Diseases and Gall-Stones. The main indications are to remove the obstruction if possible; to keep the patient on a light and easily digestible diet; and to prevent constipation by exercise and saline laxatives, preceded at the outset by a mercurial purge. Itching should be relieved by warm baths, or by lotions such as carbolic acid (1 in 40).

### ICTERUS NEONATORUM

The form of jaundice which occurs amongst new-born infants may be of either a mild or a severe type. The *mild* form appears on the second or the third day, and lasts from seven to fourteen days; beyond the pigmentation few symptoms are present. There may be bile in the urine, and the fæces are pale.

The jaundice is possibly due to the large destruction of red corpuscles, which takes place in the first few days after birth. Patency of the ductus venosus, allowing the portal blood (which contains at this time bile pigment) to mix with the systemic circulation, has also been suggested as a cause.

The etiology is, however, very obscure.

The *severe* form is due to—

1. Congenital absence of the hepatic duct.
2. Congenital syphilitic hepatitis.
3. Phlebitis of the umbilical vein.

This form is invariably fatal.

### ACUTE YELLOW ATROPHY OF THE LIVER (FATAL JAUNDICE)

A rare disease due to some unknown toxin, and characterised by a rapid and often complete destruction of the hepatic cells throughout the gland.

**Etiology.**—Women are more often attacked than men, and pregnancy is sometimes associated with the condition. The disease is commonest between the ages of twenty and thirty. Emotional conditions, alcoholic excess, syphilis, and pre-existing disease of the liver are said to be contributory causes. There are many points of resemblance between acute atrophy and phosphorus poisoning.



**Morbid Anatomy.**—The liver is much diminished in size, soft and flaccid. The capsule is wrinkled. On section, destruction of the hepatic cells, and empty bile-ducts, are marked features. Yellow and red atrophic and necrotic patches are to be seen, while granules of pigment and *crystals of leucin and tyrosin* can be detected by the microscope. There is fatty degeneration of the kidneys, heart, and muscles, and petechial hæmorrhages occur subcutaneously and in mucous and serous membranes.

**Symptoms.**—Usually there are precursory symptoms, such as mental and bodily depression, constipation, and tenderness in the hepatic region. Slight simple jaundice may precede the fully developed disease. As a rule there is little fever, but in some cases it may run up to 106° F. In the confirmed stage, intense jaundice sets in, with vomiting, etc., severe hæmorrhages, evacuations of pale fæces (or the fæces may be black from mixture of blood); pregnant women abort, and the typical typhoid state ushers in the end. The liver dulness gradually diminishes, and may ultimately disappear. The urine is bile-stained, its urea is diminished, and it often contains leucin and tyrosin. Death usually takes place within seven days of the onset of the confirmed stage.

**Diagnosis.**—*The severity of the symptoms, with the jaundiced aspect, diminished hepatic dulness, and the presence of leucin and tyrosin in the urine, make the diagnosis fairly easy.*

As we have already seen, the appearance of leucin and tyrosin in the urine, and the diminution of urea, in a condition where the hepatic function is virtually abolished, are strong evidence of the part played by the liver in disposing of nitrogenous waste products.

**Treatment** is useless, but an attempt may be made to eliminate the toxin by purgation. Subcutaneous injections of saline solution may also be given.

## ABSCESSSES IN THE LIVER

Suppuration in the liver may be due to many causes, but, though sometimes occurring in the distribution of the hepatic artery (general pyæmia), usually arises through portal infection, of which there are three main forms:—

1. Suppurative pylephlebitis.
2. Pyæmic abscesses, usually multiple (portal pyæmia).
3. Tropical abscess, usually single.



## I. SUPPURATIVE PYLEPHLEBITIS

This disease follows upon suppuration in the portal area consecutive to diseases of the intestinal tract, and most often of the large intestine or appendix. It may follow suppuration of the mesenteric glands, abscess of the gall bladder, or disease of the pelvic organs. The liver is enlarged and congested. There may be cloudy swelling of the liver-cells. On the surface, and extending through the substance of the organ, are branching yellow areas, due to suppuration in the walls of the veins. The symptoms are similar to those of pyæmic abscesses, and treatment is purely palliative.

## II. PYÆMIC ABSCESES

Pyæmic abscesses of the liver are usually small and scattered throughout the liver substance.

**Pathology.**—The condition is dependent on the presence of pyogenic cocci, the organisms gaining entrance to the liver in one of three ways :—

1. *Through the hepatic artery* in cases of general pyæmia, especially after acute periostitis or middle ear disease. In such cases, the organisms must have first passed through the capillaries of the lung.

2. *Through the portal vein* in lesions of the portal area (bacillary dysentery, appendicitis, etc.).

3. *Through the bile-ducts* in ulceration from gall-stones, etc. In these cases the suppuration is in the course of the distribution of the portal vein.

**Symptoms.**—Hectic fever, rigors, sweating, prostration, and often vomiting, with jaundice and a tender, painful, regularly enlarged liver, are the main indications, but the symptoms are often very obscure, and pain may be absent. “Febrile jaundice” is a valuable diagnostic indication when present. The other symptoms of pyæmia are also present.

**Treatment** is that of pyæmia.

## III. TROPICAL ABSCESS

**Etiology.**—The disease is nearly always due to the amœba dysenteriae (*Entamœba histolytica*), but other organisms, such as the bacillus coli, are occasionally found. Most of the patients



have had dysentery. Predisposing causes are alcoholic excess and a tropical climate.

**Morbid Anatomy.**—The abscess is usually single, but there may be two or more. Its most frequent seat is in the right lobe. Its walls are firm and thick, and have often three distinct layers—

1. *Inner.*—Grey in colour, and composed of necrosed tissue, amœbæ, and pus cells—no membrane internally.
2. *Middle.*—Brownish-red friable liver tissue.
3. *Outer.*—Hyperæmic liver tissue, surrounded by connective tissue.

Small amœbic abscesses contain a glairy, translucent fluid in which are amœbæ and detritus of necrosed liver tissue; in larger abscesses the contents are thick, viscid, chocolate-coloured, or like anchovy sauce, and of a peculiar, disgusting, sourish odour. Bacterial abscesses contain ordinary pus. The abscess if left alone most often bursts *into the right lung*.

**Symptoms.**—The earlier symptoms are pain, sense of weight and fulness, and tenderness in the right hypochondrium, *i.e.* indications of acute hepatitis. Jaundice is sometimes present, but is slight. These symptoms are followed by rigors, fever of an irregular type, nocturnal sweats, pain over the liver and sometimes in the right shoulder, dry spasmodic cough, digestive disturbances sometimes including vomiting, and emaciation. The physical signs are irregular enlargement of the liver, increase in the area of hepatic dulness both upwards and downwards, downward displacement of the hepatic margin, sometimes a rounded, smooth prominence in the right hypochondrium, and local tenderness. Pleuritic friction, or crepitus over the lower lobe of the right lung, may be audible. It must be remembered that the symptoms may be latent until those of rupture appear. In such cases, marked leucocytosis may help to the diagnosis, but it is not always present. *When the abscess bursts into the lung, the expectoration of anchovy paste-like sputum is characteristic.* Other seats of rupture are the pleura, the peritoneal cavity, or the gastro-intestinal tract. If the diagnosis is doubtful, the liver may be punctured with a fine trocar, but this should not be done unless, if pus be found, the major operation is intended to follow at once. The exploration may be repeated if pus is not found at the first attempt.



**Treatment.**—In the stage of hepatitis, before abscess has formed, rest in bed, light diet, and saline laxatives may be tried. Ipecacuan in doses of 30 grains (grm. 2·0) once daily, or ammonium chloride should be given. The use of emetine (*see* p. 77) may at this stage prevent the formation of abscess. After its formation, incision and drainage was until recently considered the best practice, but aspiration with hypodermic injection of emetine, and injection of a solution of quinine into the sac, is now preferred, and gives better results.

## CIRRHOSIS OF THE LIVER

A chronic inflammatory condition (chronic interstitial hepatitis) characterised by a great increase of connective tissue at the expense of the proper glandular tissue. It occurs in two main forms (1) multilobular, atrophic, or portal alcoholic cirrhosis, and (2) unilobular, hypertrophic, or biliary cirrhosis. An intra-lobular or pericellular cirrhosis also exists, and there are mixed types.

### I. PORTAL OR ALCOHOLIC CIRRHOSIS

**Etiology.**—Portal cirrhosis is most common in the middle period of life, in the male sex, and *in those addicted to alcohol*, especially spirits. The disease may also occur in childhood, either from early intemperance or from hereditary influences, among which is syphilis. Alcohol is not the direct cause of cirrhosis, its specific action on the liver being to produce fatty change, but it lowers the resistance of the liver to the action of other poisons, or possibly even of micro-organisms, which are then free to set up connective tissue proliferation. These poisons may reach the liver either by the portal circulation, or much less commonly by the hepatic artery. Their nature is not conclusively ascertained, but among them probably are bacterial toxins due to infections or absorbed from the alimentary canal, certain irritating foods, and occasionally mineral poisons, such as arsenic.

**Morbid Anatomy.**—Cirrhosis of the liver is generally regarded as a chronic inflammation of the connective tissue in the portal areas, leading to secondary degeneration and atrophy of the hepatic cells. The view that it is due to a primary degeneration of these cells, and that the fibrosis is due to displacement, is not so widely held, but, as Rolleston suggests, it is probable that the causative poison affects both elements simultaneously, though in different ways.



The liver may be either enlarged, sometimes enormously (up to 200 ounces), or more commonly decreased in size or "atrophic," when it may weigh as little as thirty ounces. The enlargement may be due to active congestion, and therefore temporary, passing into atrophy at a later stage, or it may be due to extensive fatty change, when it is permanent. Fatty change and enlargement, as Rolleston shows, are not more common in beer-drinkers than in spirit-drinkers. The more atrophied the liver, the more likely is it to be rough, nodular, or "hobnailed" on the surface; and conversely, the larger the liver the greater is the probability of its being smooth or but slightly roughened. Atrophy is not necessarily preceded by a hypertrophic stage, but may be present from the first.

The following table gives the main features of the morbid anatomy both of portal and of biliary cirrhosis, and shows the differences between them.

PORTAL OR MULTILOBULAR  
CIRRHOSIS

1. The bile-ducts are not involved, and jaundice is a late symptom.

2. The new-formed connective tissue compresses the branches of the portal vein.

3. In the earlier stages, active congestion and proliferation of connective tissue in the portal spaces may cause increase in the size of the liver; later, there is usually contraction.

4. The capsule is much thickened, and the surface is rough and hobnailed.

5. The masses of liver cells vary in size, some consisting of several lobules, others being smaller than a lobule. Each mass forms a *distinct area* with a rounded outline, and is enclosed in a fibrous girdle.

6. On microscopic examination, the process is seen to be going on *chiefly* at the periphery of the lobules. The fibrous tissue is very dense.

BILIARY OR UNILOBULAR  
CIRRHOSIS

1. The smaller bile-ducts are inflamed (cholangitis); jaundice is early and severe.

2. The portal circulation is not impeded.

3. The new tissue is diffused throughout the organ, and causes a great increase in size.

4. The capsule is not thickened, and the surface is smooth (like morocco leather).

5. The masses of liver cells consist of isolated lobules. The cut surface has a uniform and finely-granulated appearance.

6. The fibrous tissue is not confined to the periphery, but invades the substance of the lobules. It is much more open than that of portal cirrhosis.

In portal cirrhosis the liver cells ultimately degenerate; in biliary cirrhosis they are for long well preserved. In both there may appear to be a new formation of bile-ducts, probably due to hyperplasia of the liver cells,



Portal cirrhosis, then, is a condition characterised pathologically by—

1. Increased growth of fibrous tissue in Glisson's capsule, and in the prolongations of it running into the liver substance.
  2. Obstruction to the portal circulation.
  3. Increased blood-pressure in the hepatic arteries.
  4. Obliteration of hepatic cells
  5. Obstruction of biliary ducts
- } *later.*

Ascites and hæmorrhoids are consequent upon the portal obstruction. Intestinal catarrh is very common, and enlargement of the spleen, due partly to toxæmia, partly to portal obstruction, is constant. Phthisis, pleurisy, or chronic peritonitis may be present as complications.

As the portal circulation becomes obstructed, a collateral circulation is established between the systemic veins and the branches of the portal vein. The most common anastomoses are between—

1. The gastric and œsophageal veins.
2. The superior hæmorrhoidal of the inferior mesenteric and the inferior hæmorrhoidal of the internal iliac vein.
3. The veins of the intestines and the retroperitoneal veins.
4. The accessory portal vein of Sappey—*i.e.* a vein running alongside the round ligament of the liver, connecting the portal vein with branches of the epigastric veins near the navel. This sometimes produces a large bunch of varicose veins, the so-called *caput medusæ*, surrounding the umbilicus.

**Symptoms.**—If the collateral circulation is well established, cirrhosis may remain latent and cause no symptoms. At first there is little disturbance beyond a catarrhal condition of the stomach—morning vomiting, anorexia, and acid eructations. Epistaxis is common in the early stages, and sooner or later a severe hæmatemesis or œsophageal hæmorrhage, coupled with a sense of weight in the hepatic region, reveals the true nature of the disease; and on examination, the liver is usually found to be enlarged, firm, sometimes slightly tender, and with a hard and sharp edge. Emaciation, pallor, and flatulent distension of the abdomen are common at this stage, and if the emaciation is marked, granulations may be felt on the hepatic surface (“hob-



nailed liver"). The spleen is also enlarged, although sometimes not palpably. There may be melæna. As time wears on, ascites and emaciation come to be the prominent features. The liver may remain enlarged, or may gradually contract, the area of hepatic dulness being diminished. Jaundice, probably hæmolytic in origin, is late and often slight; sometimes it is absent throughout. The ascites is well marked, and there may be œdema of the feet. The surface of the abdomen is covered by large distended veins, showing the attempted establishment of a collateral circulation. Fever as a rule is absent. There may be slight nervous symptoms at any time, but in the terminal stages drowsiness, delirium, or coma is specially liable to appear and to lead on to death, which may also result from asthenia, uræmia, or cardiac failure.

**Prognosis** is bad as regards cure; but if the collateral circulation be well established, the atrophy not marked, and if the patient is made to live temperately, bad symptoms may not arise for a considerable period. In far-advanced cirrhosis the treatment is merely palliative.

## II. BILIARY CIRRHOSIS OR HANOT'S DISEASE

**Etiology.**—Biliary, unilobular, or hypertrophic cirrhosis is most common between the ages of twenty and thirty, and not uncommon in young children. It may affect several members of the same family, and attacks males oftener than females. It has no definite connection with alcoholism, and the *causa causans* is unknown, though it is probably of a toxic nature, and derived from some chronic hæmic infection. The poison probably reaches the liver by the general circulation.

**Morbid Anatomy.**—The liver is uniformly enlarged, and may weigh as much as eight pounds. It is sometimes adherent to the diaphragm. The spleen is much more enlarged than in portal cirrhosis, and this may precede the hepatic enlargement. There is no intestinal catarrh. A secondary portal cirrhosis may be superadded to the biliary cirrhosis in old cases. For further details see the table on p. 221.

**Symptoms.**—The disease may begin with hepatic, splenic, or gastro-intestinal symptoms, the last being uncommon. The onset is generally gradual, and the course is slow. Jaundice is at first slight, but increases in intensity, and may at last be very marked. There are periodic attacks of abdominal pain,



accompanied by *fever*, and wasting is progressive. Ascites is absent until late in the disease, when it may follow secondary portal cirrhosis; but the upper part of the abdomen is distended by the enlarged liver and spleen. The liver is smooth, firm, slightly tender, and uniformly enlarged, and its lower border may be found at the level of the umbilicus or below it. Hæmorrhages such as epistaxis may occur in the late stages, but hæmatemesis is rare. The disease lasts several years and leads to death by toxæmia, coma, or the typhoid state, or through intercurrent affections.

**Treatment of Hepatic Cirrhosis.**—Whichever form is present, the principles of treatment are the same. Temperance in eating, a light and nourishing diet, abstinence from alcohol, and from hot condiments or irritating foods must be observed. In portal cirrhosis milk diet is advisable at first, until some improvement is apparent, when the diet may be made less rigid. The bowels should be kept open by salines, such as sulphate of soda or Carlsbad salts, which have also a beneficial effect on the intestinal catarrh. Chloride of ammonium may be tried, and iodide of potassium, if there is any chance that the cirrhosis is syphilitic. Tonics such as strychnine or nux vomica with hydrochloric acid are useful, while abdominal massage and regular exercise are beneficial in the early stages. Arsenic is contra-indicated.

In *ascites*, the fluid should be evacuated by means of Southey's tubes. The abdomen must afterwards be firmly bandaged, to avoid the consequences of too sudden relief of the pressure on the intra-abdominal veins. In lesser degrees of ascites, saline laxatives or jalap should be given. Stronger purgatives may set up intractable diarrhœa. *Hæmatemesis*, if moderate, should not be treated too energetically, as it often acts as a safety-valve by relieving portal congestion. If it be excessive, ice, ergot, acid sulph. dil., or a full dose of morphia should be resorted to.

The operation of *epiplopexy* (suturing the great omentum to the abdominal wall) is an attempt to increase the collateral circulation, and has proved serviceable in some cases of ascites, but its statistics are not very encouraging, and the mortality is high.

## SYPHILIS OF THE LIVER

In congenital syphilis the liver is very frequently affected, being enlarged, firm, and resistant, and showing histologically a pericellular cirrhosis. Less commonly, isolated gummata are



present, or a circumscribed gummatous hepatitis may simulate a tumour. The existence of other signs of congenital syphilis makes the diagnosis easy. When such cases are cured, the liver may be left with a diminished power of resistance to disease, and multilobular cirrhosis may arise in such children when its exciting causes are present. The liver may also suffer in late hereditary syphilis.

In adults, jaundice is sometimes present in the secondary stage of syphilis. It is due to a specific hepatitis, and readily yields to specific treatment. Far more important are the tertiary lesions, which may closely simulate cirrhosis of the liver. Gummata, cicatrices, and gummatous infiltration are all found. In the cases resembling cirrhosis, the infiltration is usually combined with the presence of gummata or of cicatrices. Ascites is the most prominent symptom, and there may be other evidences of portal obstruction, along with enlargement of the spleen. The outline of the liver is, however, irregular, and the enlargement is not uniform, the left lobe being often relatively enlarged as compared with the right. Jaundice is present in about half the cases; emaciation is usually less in syphilis than in cirrhosis. There may be other signs of syphilis, or a history of it, while an alcoholic history speaks for cirrhosis.

In other cases a large gumma may simulate tumour, and in yet others there may be irregular fever, along with enlargement and tenderness of the liver, suggesting abscess.

For *treatment*, see p. 107.

## CANCER OF THE LIVER

Cancer is the most common hepatic tumour. It may be primary, but is much more often secondary to cancer elsewhere, especially in the gastro-intestinal tract, and in women in the breast or pelvic organs. Sarcoma also occurs, but it is not common. Angioma is frequent, but of no clinical importance. Men are more frequently attacked than women, and oftenest in middle or later life. The predisposing causes are similar to those of cancer elsewhere, namely, heredity, injuries, etc.

**Morbid Anatomy.**—All types of cancer have been found in the liver, but the primary forms are nearly always epitheliomatous, and of the cylinder-celled variety.

*Primary Cancer.*—Two main types are distinguished—

1. The massive cancer, greyish-white in colour, occupies



a large portion of the liver, and is abruptly defined from the liver tissue.

2. Nodular cancer, in which the nodules vary much in size, and are irregularly scattered *throughout* the organ.

In a third form, which is rare, the growth is diffuse, and the liver is usually contracted.

*Secondary Cancer.*—The tumours have the structure of the primary growth. They occur as nodules scattered throughout the substance of the liver, which becomes enormously enlarged. The growth of each nodule is uniform in all directions, and the more superficial nodules thus protrude as rounded tumours on the surface. As they grow larger their central portions become necrotic and the surface consequently presents a central depression. Section of the liver shows scattered and irregular whitish areas of malignant growth, of which the central portions have undergone necrosis and fatty degeneration and into many of which hæmorrhage has occurred.

**Symptoms.**—With or without previous indications of gastrointestinal, uterine, or mammary cancer, *pain* in the hepatic area and often in the right shoulder, at first slight, afterwards severe and lancinating, is usually present. In some cases it is not marked. The patient rapidly emaciates, and assumes the characteristic cachectic appearance. The liver is very greatly enlarged, often extending below the umbilicus; its edge is irregular; it is hard and tender; and its surface is studded with nodules easily felt through the emaciated abdominal wall, often with a central depression or *umbilication*, and of a hardness even greater than that of the rest of the liver. The nodules are sometimes even visible below the ribs (the so-called “Farre’s tubercles”). Jaundice, from pressure on the common bile-duct, is present in about half the cases and is often deep; ascites from pressure on the portal vein is not uncommon. Attacks of local peritonitis may from time to time increase the pain. Febrile attacks may alternate with afebrile periods. It should be remembered that cancer may occur in a cirrhotic liver, in which case there may be no enlargement. Should the growth be a melano-sarcoma, pigmented nodules in the skin may point to its true nature.

The course is rapid, and death is seldom delayed more than a year.

**Diagnosis** between cancer and cirrhosis is often difficult



when there is a strong history of alcoholism. The following table may help—

	CIRRHOSIS.	CANCER.
<i>Progress.</i> .	1. Often slow.	1. Always rapid.
<i>Liver</i> . .	2. Enlarged or small; nodules absent or small, not tender, and appearing late.	2. Large and irregular; nodules large, tender, and early apparent.
<i>Pain</i> . .	3. Not marked.	3. Well marked.
<i>Ascites</i> . .	4. Usually present.	4. Often absent.
<i>Jaundice</i> .	5. Not till late.	5. Often a marked feature.

## HYDATID DISEASE

**Hydatid Cysts** occur more frequently in the liver than in any other organ. They are usually single, but may be multiple. They are most frequent in the right lobe.

For the life history of the parasite and the characters of the cyst, see p. 199.

**Symptoms.**—A hydatid cyst of the liver grows slowly, and usually upwards towards the pleura, or downwards below the ribs. It is painless unless it suppurates, and may cause no symptoms until it becomes extremely large or bursts. Pain, often severe, rigors and sweating, remittent fever, and emaciation (*i.e.* signs of septic infection) follow suppuration, which ends in rupture of the cyst. The blood shows a moderate degree of eosinophilia.

**Physical examination.**—When the cyst grows from the anterior surface, a globular, dense, elastic tumour may be felt, and with care fluctuation is detected. On palpation, a peculiar vibratory thrill (hydatid fremitus) is sometimes present. There is no tenderness, nor any sign of “pointing” as in abscess. Jaundice is absent. If the cyst is on the posterior surface, the area of hepatic dulness is increased in an upward direction. When the cyst is very large, there may be pressure upon the lung, the abdominal viscera, the vena cava, or the portal vein, causing respectively dyspnoea, constipation, oedema, or ascites; but pressure symptoms are usually absent.

**Prognosis.**—The cyst may undergo spontaneous cure without rupture, death of the parasite causing it to contract. As a rule it increases until rupture takes place into the lung or pleura,



into the intestines, into the hepatic duct, or into the peritoneal cavity.

**Treatment.**—As soon as the cyst is discovered, employ surgical means. Aspiration is often practised, but incision and evacuation of the contents is much the best method of treatment.

*Other cysts* of the liver may occasionally occur, but their interest is chiefly pathological.

### AMYLOID OR WAXY LIVER

The liver is often affected with lardaceous disease, and as elsewhere, the degeneration begins in the middle coats of the blood-vessels, and afterwards affects the capillaries, which become swollen and compress the hepatic cells, causing them to degenerate and atrophy. The liver is uniformly and enormously enlarged, and smooth. On section, its substance is glistening, firm, and resembles yellow wax, the cut surfaces showing only faint traces of lobules. If tincture of iodine be poured upon the cut surface after it has been washed free of blood, the amyloid substance (*lardacein*) takes on a reddish-brown or claret colour. It must be remembered that amyloid disease may co-exist with fatty or syphilitic disease of the liver.

**Etiology.**—Prolonged suppuration, as in tuberculous ulcerations, caries, necrosis, and phthisis; syphilis, even when there has been no suppuration; and very rarely chronic toxæmic conditions or the malarial cachexia.

**Symptoms.**—The only hepatic *symptom* is a sense of weight in the region of the liver; other symptoms, such as diarrhoea and albuminuria, are due to involvement of the bowel and kidney. The liver is enormously enlarged, firm, smooth on the surface, and not tender. The spleen is also much enlarged, and presents similar characters. Later, there may be a terminal ascites, but ascites is usually due to complications. *Pain is rare, and jaundice is absent.* Simultaneous implication of the spleen and kidney is important for the diagnosis. The disease is usually fatal.

**Treatment.**—Remove the cause if possible. Iodide of potassium is often of great service, especially when the disease is due to syphilis. The general treatment is similar to that of tuberculosis (*see Phthisis*, pp. 366–67).



## FATTY LIVER

Two conditions are included under this designation, fatty infiltration, or an excessive accumulation of fat-globules in the hepatic cells, and fatty degeneration, in which the cell-protoplasm is partially replaced by fat. Infiltration is merely an exaggeration of the normal condition, for the hepatic cells always contain fat-globules; degeneration is pathological. The two conditions tend to run into one another.

**Etiology.**—Fatty liver is met with (1) in obesity (infiltration); (2) as a result of poisoning by bacterial or other toxins (acute infections, *pulmonary tuberculosis*, *alcoholism*, anæmia, poisoning by *phosphorus* or other chemical poisons, acute yellow atrophy of the liver; (3) in combination with other diseases of the liver (cirrhosis, amyloid, passive congestion).

**Morbid Anatomy.**—The organ is uniformly and sometimes very markedly enlarged; its surface is smooth, its consistence sometimes firm, sometimes soft, and its edges are rounded. On section it is greasy. The fatty change is most marked at the periphery of the lobule, but the whole lobule may be affected.

**Symptoms.**—Any symptoms that are present are due to the causative disease. The physical signs are those of an enlarged liver, somewhat soft in consistence, regular in outline, and with rounded edges. The enlargement is *not* accompanied by pain or jaundice, or by enlargement of the spleen. The stools are pale but bile is not absent.

**Treatment** is that of the primary disease.

## PASSIVE CONGESTION OF THE LIVER

This common condition is secondary to dilatation of the right ventricle, whether produced by pulmonary conditions such as emphysema, or by lesions of the left side of the heart. *Post mortem* the liver is found to be enlarged, deeply engorged, and firm; and on section, the intralobular and sublobular branches of the hepatic vein are seen to be dilated, so that the centre of the lobule has a deep red or purplish hue, while the periphery is yellowish or pale from fatty change (nutmeg liver).

**Symptoms.**—There is a feeling of weight in the right hypochondrium, with digestive disturbances. The liver is enlarged, often very considerably, markedly tender, and it presents a



firm smooth surface. Occasionally it can be felt to pulsate. Slight jaundice is often present, and its yellow tint may combine with the cyanosis of the cheeks and lips to form the so-called mitral facies. Ascites is not infrequent.

**Treatment.**—The cardiac condition must receive attention, and the hepatic engorgement should be dealt with by saline or mercurial purges, and the local application of leeches or cups.

## DISEASES OF THE GALL-BLADDER AND BILE-DUCTS

### CHOLECYSTITIS

Inflammation of the gall-bladder may be catarrhal, suppurative, or phlegmonous. It is most frequently due to microbic infection produced by extension of inflammation from neighbouring parts. Parasites or calculi in the gall-bladder may act as exciting causes. The organisms chiefly found are the coliform group, the typhoid bacillus, the pyogenic cocci, and the pneumococcus. The gall-bladder is distended, and the cystic duct is often closed by swelling of the mucous membrane. The chief *symptoms* are fever, tenderness a little above and to the right of the umbilicus, and pain in the same situation or referred to the appendix. There may be vomiting. The gall-bladder can sometimes be felt. In the suppurative and phlegmonous cases, perforation may occur, and results in local abscess or general peritonitis. Treatment consists in confinement to bed, relief of pain, and soothing applications to the right hypochondrium; suppuration or the presence of calculi calls for operative measures.

### CHOLANGITIS

**Acute Cholangitis** (*catarrhal jaundice*) has been generally supposed to be an inflammation limited to the lower end of the common bile-duct, associated with catarrh of the stomach and duodenum, and therefore produced by the causes of that disease. Of late years, however, it has frequently occurred in epidemic form among children, and occasionally among adults, the cases being indistinguishable from those which usually arise in isolated instances. The inference would seem to be that the latter are sporadic cases of an occasionally epidemic malady caused by some organism as yet unknown, but possibly air-borne; and it is suggested that a diffuse hepatitis, involving the common bile-



duct by descending inflammation, may account for the obstructive jaundice.

Whatever its cause, it is one of the commonest forms of jaundice. Its onset is often gradual, the patient complaining for some little time of headache, drowsiness, anorexia or dyspeptic symptoms, possibly also of slight feverishness. In other cases obstructive jaundice may set in without warning. The skin is yellow, but never bronzed, bile is absent from the fæces and present in the urine, and there is often pruritus. There is no pain or tenderness. The liver is not usually enlarged, but a moderate enlargement has occasionally been noticed even in the sporadic cases, and some enlargement is not uncommon in the epidemic form. The condition lasts for several weeks, after which the jaundice gradually disappears. *Treatment* consists in light diet, abstinence from alcohol, and the use of saline laxatives, with an occasional dose of calomel. The patient should not be purged.

**Suppurative Cholangitis**, which is most frequent in the presence of gall-stones, is due to infection by micro-organisms (the pyogenic cocci, *b. coli*, *b. typhosus*), the activity of which is promoted either by local diseases or by general infections (enteric, pneumonia, cholera, &c.). Its chief symptoms are rigors, fever, nausea and vomiting, with jaundice and a smooth, tender enlargement of the liver. The gall-bladder is also enlarged. Pain may be severe or slight. The treatment is surgical.

## TUMOURS

**Cancer**, whether of the gall-bladder or bile-duct, is frequently associated with gall-stones, and is commoner in women than in men. Cancer of the gall-bladder may begin either at the fundus or near the neck, and presents itself as a firm, hard, nodular tumour, very tender on pressure, and in the situation of the gall-bladder. Pain is severe and often paroxysmal, and jaundice is usually present, but is not an early symptom. The liver may be secondarily affected. When the disease affects the bile-duct, the gall-bladder is distended, and may be felt as a smooth ovoidal swelling below the ninth costal cartilage. Jaundice is present from the first, and becomes progressively deeper. Pain is slight or absent. Secondary growths are not common.

## GALL-STONES

**Gall-stones** or biliary calculi are the result of a mild and chronic catarrh of the mucous membrane of the gall-bladder and



smaller bile-ducts. Such an inflammation leads, in the case of the bile-ducts, to the precipitation of bilirubin in combination with calcium—bilirubin-calcium, and in the case of the gall-bladder, to an abnormal secretion of cholesterin by the mucous cells of its walls. It is from these two substances that calculi are chiefly formed. Their formation takes place around a nucleus, which may be epithelial or mucoid, but is far more frequently *microbic*. The organisms commonly found in the nucleus are those of the colon group, and especially *b. coli* and *b. typhosus*. The more virulent pyogenic organisms set up an acute cholecystitis, which is not associated with calculi; but less virulent strains of staphylococcus or streptococcus may lead to their formation. Any causes which lead to stagnation of bile must also favour the occurrence of *microbic* infection. Thus sedentary occupations, constipation, corset-wearing, enteroptosis, and *pregnancy* are all predisposing causes. Women, therefore, are the most frequent subjects, and among female patients Naunyn states that ninety per cent. have borne children. Gall-stones are rare before the age of thirty.

**Characters and Composition.**—The stones may be single or multiple. When single, they are ovoid in shape, and if composed of pure cholesterin, as is usual, they are light, glistening, and with a granulated surface. Much more commonly they are multiple, in which case they are *faceted* from pressure of the opposing surfaces. The ordinary gall-stone consists chiefly of cholesterin, arranged in laminae, but its nucleus is of bile-pigment. It also contains salts of lime and magnesia. Externally it may be yellow or brown, and has a greasy surface when fresh. The numbers vary; there may be only a few, or many hundreds.

**Symptoms.**—If the calculi remain in the bladder, they may cause no symptoms whatever. If a calculus passes into the cystic duct or common bile-duct, it gives rise to *biliary colic*, of which the following are the main symptoms :—

1. Sudden and excruciating *pain*, usually paroxysmal, beginning in the epigastrium or right hypochondrium, and radiating in all directions over the abdomen, also into the lower part of the thorax, and sometimes into the right shoulder. Marked *tenderness* in the epigastrium and right hypochondrium, often with a palpably enlarged gall-bladder.

2. Rigors, profuse sweating, great feebleness of pulse, symptoms of collapse. Fever is present in more than half the cases, and is due to a concomitant acute cholecystitis.



3. Reflex vomiting, which often gives some relief (probably by the diaphragmatic contractions aiding peristalsis).

4. In the majority of cases, *jaundice*, arising sometimes a few hours, sometimes as long as three days, after the onset of pain, and persisting for a few days or even a few weeks.

The attack lasts from three to twelve hours, but a rapid succession of attacks may cause almost continuous pain for several days. The pain ends suddenly when the stone slips into the bowel, but the patient is left very prostrate. The fæces should be examined for the presence of calculi.

Instead of passing into the duodenum, the stone may be impacted in the cystic duct. An attack of biliary colic *without jaundice* follows; and the permanent results are (1) *Dropsy of the gall-bladder*, which may be felt as a smooth movable ovoid tumour beneath the lower edge of the liver, and has been mistaken for floating kidney. When the obstruction is chronic, the contents of the tumour are clear mucus. (2) *Cholecystitis*, simple or suppurative, the latter (*empyema* of the gall-bladder) being the commoner. (3) Atrophy of the gall-bladder, which may be a sequel to dropsy.

If the stone is impacted in the common duct, permanent jaundice follows. If the obstruction is partial, so that the stone forms a ball-valve, the jaundice varies in intensity, and there are recurrent attacks of colic with the so-called *hepatic intermittent fever* (rigors, pyrexia, and sweating). The jaundice deepens with each paroxysm.

Calculi in the gall-bladder may sometimes cause acute cholecystitis, or may lead to ulceration, with the establishment of a *biliary fistula* opening into the duodenum, colon, or other hollow viscus. A large stone passing through such an opening may give rise to obstruction of the bowel. Occasionally a fistula opens on the skin.

Cancer of the gall-bladder may follow the continued presence of gall-stones.

The **Treatment** of biliary colic consists in—

1. Hypodermic injection of morphia.
2. Chloroform inhalations till the morphia acts.
3. Hot fomentations or the hot bath, and laxatives.

Subsequently, stagnation of bile should be prevented by regular exercise, abdominal massage, and the avoidance of tight corsets or belts, the diet should be regulated, and salines, especially the sulphate or phosphate of soda, should be given.



Sodium salicylate in doses of gr. x (grm. 0.6) thrice daily is useful in promoting the flow of bile. Surgical measures are called for in recurrent attacks of colic, or in the presence of complications.

## DISEASES OF THE PANCREAS

These are comparatively rare, but not the less important. In a series of 6000 autopsies performed at Guy's Hospital, the pancreas was diseased in 99 instances, or 1.6 per cent. (Hale White). Impairment of pancreatic functions leads to imperfect digestion of fats and of proteids, and frequently to imperfect metabolism of sugar. The stools therefore contain excess of fat, and are pale, soft, and bulky, and undigested muscle fibre may be found in them, while diabetes is not uncommon. The tests for pancreatic insufficiency are either too elaborate for clinical use or of uncertain value.

**Pancreatitis** may be acute or chronic. In either case it is the result of infection extending along the duct from the intestine, or, if the ampulla of Vater is obstructed, from the common bile-duct. The *acute* form may be either suppurative or hæmorrhagic, and may lead to thrombosis of the splenic vein and fat necrosis of the subperitoneal fat. It occurs chiefly in males between the ages of twenty-five and sixty. Though there may be previous duodenal catarrh, the actual onset is abrupt. Sudden and severe epigastric pain, vomiting, and collapse are the chief symptoms. Tenderness and rigidity across the middle of the epigastrium, where in the course of a few hours a circumscribed swelling may appear, are aids to diagnosis; and sometimes there is absolute dulness in both flanks, unaltered by any change of position. This is due to the presence of coagulated blood, which has flowed into the kidney pouches. It may aid in distinguishing the disease from acute intestinal obstruction, with which, or with perforated gastric ulcer, it is very often confused. Slight jaundice may be present, and after the initial collapse some fever. Medicinal treatment can do little but relieve pain, and cases so treated end fatally within four or five days; but of late years, though the mortality is still high, there have been many recoveries under *early* incision and drainage.

The *chronic* form has no very definite symptoms. It leads to generalised fibrosis with atrophy of the glandular substance (diabetes), and sometimes to jaundice from implication of the common duct. Cysts or calculi may form in the ducts.

**Pancreatic Cysts**, if small and numerous, may be due to



chronic pancreatitis; if large, they are caused by an impacted calculus in the duct or by pressure upon it from without. They may reach a great size. The fluid contents possess diastatic and sometimes tryptic activity, an important point in diagnosis. The smaller cysts cause no symptoms; the larger cause tumour in the epigastrium (often with visible bulging) median in position, immobile, elastic, but non-fluctuant, and dull to percussion. Pain and fever are unusual, but wasting may be present, and sometimes glycosuria. Inflation of the stomach shows that the tumour lies behind it. The *treatment* is surgical.

**Cancer of the Pancreas** affects chiefly the head of the gland. It may be scirrhous or medullary, and causes metastatic growths in the liver and spleen. It may press upon the duodenum (dilatation of stomach) or infiltrate the hilum of the liver (jaundice, ascites). The tumour often reaches a considerable size. The early *symptoms* are severe and paroxysmal epigastric pain, with rapid wasting, and the other general signs of malignant disease. Later a tumour, or sometimes only increased resistance, is to be felt in the upper part of the abdomen, to the right of the middle line. The tumour, when present, is firm, tender and immobile. Pressure symptoms (see above) may be noted. Glycosuria is sometimes present. Death occurs from asthenia or gastric hæmorrhage.

**Hæmorrhage** into the pancreas may cause sudden death. The symptoms are severe and sudden epigastric pain, with nausea and vomiting, and sometimes epigastric tenderness. *Post mortem* no other lesion may be found.



# DISEASES OF THE PERITONEUM

## ASCITES

ASCITES or dropsy of the peritoneum is not a disease in itself, but is merely symptomatic of some condition which causes an increased transudation of fluid into the peritoneal cavity. Dropsy is due to some abnormal change in the blood-vessel walls, along with an increase of pressure within the vessels, and sometimes an altered composition of the blood. These conditions are to be found as the result of inflammation, vaso-motor paralysis, or obstruction. Since the venous blood of the intestines is returned *via* the portal circulation, we naturally look for the more common causes of ascites in morbid states of the portal vein; but it must be remembered that inflammatory changes of local blood-vessels due to morbid processes in the peritoneum or abdominal organs also tend to bring about dropsy of the peritoneum. It is important to remember that the causes of ascites are *not confined to obstruction* of the portal vein; sometimes the dropsy is of local inflammatory origin, and the fluid is then frequently collected into localised pools, and lies within a meshwork of inflammatory adhesions so extensive as to mask the typical signs of ascites.

**Etiology.**—The local causes of ascites are—

1. *Portal obstruction*, either within or outside the liver.
2. Chronic peritonitis, simple, malignant, or tuberculous.

Ascites may also be due to general causes, forming part of the dropsy of renal or cardiac disease.

It must be remembered that venous obstruction does not of itself produce dropsy *until some retrograde change is set up in the walls of the vessels*. But the retarded flow which it induces ultimately alters the nutrition of the vessel wall.

As causes of portal obstruction may be mentioned thrombosis of the portal vein; cirrhosis of the liver and perihepatitis; enlarged glands in the portal fissure; neoplasms of the liver or pancreas; and, secondarily, malignant disease in connection with the intestines or other abdominal organs.



**Signs.**—The cardinal signs are—

1. Enlargement of the abdomen, most marked in the flanks when the patient is lying on his back: the skin is tense and shiny, with dilated superficial veins on the surface.

2. Prominence of the umbilicus.

3. Fluctuation, or a thrill transmitted to the examining hand laid flat upon one flank when the opposite flank is tapped by the finger.

4. With the patient on his back, percussion yields a dull note in the flanks and hypogastrium, whilst the note at the umbilicus is resonant. The dulness changes its position as the patient turns on his side, the upper flank becoming clear, and the area of dulness in the lower flank increasing in extent as the fluid gravitates downwards.

5. In cases where the liver is enlarged, a sudden jerk with the finger upon the abdominal wall just beneath the right costal margin displaces the fluid, and allows the finger to settle down upon the surface of the organ (*dipping* for the liver).

The above conditions are sometimes associated with anasarca, or other symptoms of dropsy elsewhere.

Certain intra-abdominal conditions may alter these physical signs. If the amount of fluid is large and the intestines are bound by adhesions to the posterior wall, there will be no umbilical resonance, but the note will be dull all over. If the effusion of fluid is due to peritonitis, inflammatory adhesions may prevent its free movement as the patient changes his position. If the amount of fluid is small, no wave of fluctuation may be transmitted across the abdomen. If it is very small, even the dulness may be difficult to demonstrate in the dorsal position; but in the knee-elbow position a peri-umbilical dulness will be found.

Large ascitic accumulations have often been mistaken for the pregnant uterus or for an ovarian cyst. A tabular statement of the distinctions between them is appended (*see p. 238*).

In all cases an examination should be made of the ascitic fluid, both microscopically and macroscopically.

*Ordinary Ascitic Fluid* is light yellow or straw-coloured, generally of about 1010 sp. gr., and contains albumin. In *chylous* ascites, associated with disease of the pancreas and lacteals, the fluid is turbid and milky, exhibiting oil globules. In malignant ascites, the fluid is often dark from the presence of blood. "Cancer" cells may be discovered under the microscope.



Fluid due to inflammatory exudation is of a higher sp. gr., 1014-1020.

### DIAGNOSTIC TABLE

OVARIAN CYST.	PREGNANCY.	ASCITES.
1. Enlargement first noticed unilaterally.	1. Enlargement median.	1. Uniform distension of abdomen.
2. Abdomen dull to percussion in front, resonant in the flanks. Area of dulness greater on affected side, but extends beyond middle line.	2. Dulness median, flanks clear.	2. Dulness in flanks and hypogastrium, resonance in umbilical and epigastric regions.
3. Dulness does not change with change of patient's position.	3. No change on change of position.	3. Dulness leaves uppermost flank when patient lies on side.
4. Fluctuation over area of tumour.	4. No fluctuation except in hydramnios.	4. Fluctuation general over whole abdomen.
5. Umbilico-pubic measurement greater than umbilico-sternal; distance between umbilicus and anterior superior spine greater on affected side.	5. Umbilico-pubic measurement greater than umbilico-sternal; distance between umbilicus and anterior superior spines equal on the two sides.	5. Umbilico - pubic measurement less than umbilico-sternal; distance between umbilicus and anterior superior spines equal on the two sides.
6. No signs of pregnancy; general health deteriorated.	6. Other signs of pregnancy; health normal.	6. No signs of pregnancy; general health deteriorated.

**Treatment.**—Seek the primary disease, and direct the treatment to improving the general health on the principles already laid down under hepatic disease. In ascites of cardiac or renal origin, saline purgatives are often beneficial.

If the fluid causes dyspnoea, or other symptoms of pressure, it should be evacuated by Southey's tubes, or by aspiration. Paracentesis may be repeated many times without danger, if due antiseptic precautions are observed. It must not be forgotten that in old cases the vessels of the abdomen may be so far devitalised, that a sudden removal of the support afforded them by the dropsical fluid causes them to become engorged with blood, and may set up fatal syncope. The abdominal wall should therefore be supported during the operation by bandages, gradually tightened as the fluid is withdrawn.



## ACUTE PERITONITIS

Inflammation of the peritoneum is very rarely primary.

In the words of Hilton Fagge—"The peritoneum is a huge areolar space or lymph sac, and its most intimate pathological relations are not with the skin or mucous membrane—not even with the joints or the so-called arachnoid space—but with the pleura, pericardium, and tunica vaginalis. The diseases of these three divisions of the same original cavity are the same: acute inflammation, serous or purulent, traumatic or septic; chronic adhesive inflammation with hypertrophy; chronic irritative effusion, and passive dropsical effusion—hydrothorax, hydro-pericardium, and ascites. All three serous membranes are liable to be invaded by tubercle, and also by cancer. All three are prone to follow the pathological fate of the viscera which they cover; they are all apt to suffer in the course of Bright's disease, and, lastly, they are all affected together often by inflammation or tubercle, or more rarely by cancer."<sup>1</sup>

A peritonitic effusion differs, however, from pleuritic effusion in showing a greater tendency to become purulent, which is probably to be explained by the close proximity of the septic intestinal contents.

**Etiology.**—Acute peritonitis is almost always due to bacterial infection, by the pyogenic cocci, by intestinal organisms, or by the pneumococcus or gonococcus. It is very frequently secondary to perforation of an abdominal viscus, as in gastric ulcer, enteric ulcer, or *appendicitis*; or to extension of inflammation from neighbouring organs, such as the stomach, spleen, pancreas, liver, and *pelvic organs*. It may follow upon strangulation of the bowel, or upon penetrating wounds. The infection may occasionally be transmitted by the blood; and a fatal peritonitis may occur in the course of chronic Bright's disease.

**Morbid Anatomy.**—1. At first there is hyperæmia with loss of lustre; the hyperæmia is most marked where the intestinal coils *are not* in close contact with one another. This is followed by—

2. Fibrinous exudation, giving a more or less shaggy appearance; and by—

3. Effusion of fluid, which may be highly fibrinous and coagulate easily, forming extensive adhesions; or which may become—

4. Rapidly purulent.

<sup>1</sup> Hilton Fagge's *Medicine*, 3rd edition, vol. ii. p. 305.



**Symptoms.**—The symptoms of an *acute general peritonitis* present a most characteristic clinical picture, the main features of which are—

1. Great pain and tenderness over the abdomen, which is usually tense from tympanites, and *rigid* from muscular contraction.

2. Hurried, shallow, *thoracic* breathing.

3. Quick, wiry, and incompressible pulse.

4. *Facies Hippocratica*—i.e. “a sharp nose, hollow eyes, collapsed temples; the ears cold, contracted, and their lobes turned out; the skin about the forehead being rough, distended and parched; the colour of the whole face being brown, black, livid, or lead-coloured.” The face is also anxious.

5. Constipation; *vomiting* and often hiccough; dry, small, red tongue.

6. Moderate fever.

7. Moderate and uniform abdominal distension, increased resistance, *absence of visible peristalsis*. In later stages dulness in the flanks (fluid effusion).

8. In *perforative* peritonitis the abdomen may be tympanitic all over, the hepatic and splenic dulness being completely obliterated. Partial obliteration of the hepatic dulness may be due to meteorism. The onset of symptoms is sudden.

The abdominal tenderness is sometimes so marked that the slightest touch, or even the movements of sneezing, coughing, or respiration, may cause exquisite agony.

Whilst the above are the cardinal symptoms of a general acute peritonitis, it must not be forgotten that the condition may occasionally be latent, or that the temperature may be subnormal, diarrhoea may be present instead of constipation, and the patient's face may be actually *apathetic* instead of anxious. This latter state is probably more frequent when the peritonitic fluid speedily becomes purulent, as in some cases of puerperal or appendicular peritonitis, or those that follow perforation of an enteric ulcer.

*Acute localised peritonitis* may be appendicular (see Appendicitis, p. 183), or pelvic, originating in the Fallopian tubes or uterus, or it may implicate the cavity of the lesser peritoneum—*subphrenic peritonitis*. This condition may follow direct injury, but is usually due to disease of an abdominal organ—stomach, duodenum, appendix, liver, gall-bladder, or pancreas—and much most commonly results from perforation of a gastric ulcer. It may be non-suppurative, but generally ends in *subphrenic abscess*, of which the symptoms are often obscure. The local signs have



been mentioned under Ulcer of the Stomach (pp. 166-7); they are usually associated with hectic fever and sweating, rigors, and emaciation. Such an abscess is often mistaken for an empyema: when it is due to perforation it frequently contains air, and may simulate a pyopneumothorax. Attention to the history and to the previous symptoms should obviate mistakes.

**Prognosis** depends largely on the cause. When the disease is the result of perforation it may be fatal in a few hours. The prognosis is of course much more favourable when the peritonitis is localised.

**Treatment** is almost entirely operative. The extent of the operation depends upon the local conditions found when the abdomen is opened, and upon the general condition of the patient. It should be done as soon as possible; if for any reason it is delayed, the previous treatment must consist mainly in alleviating pain and lessening peristalsis by opium or morphine, and locally by ice or warmth, whichever is best borne. It is desirable not to mask the symptoms by using morphine too freely, but that is not to say that the appeal of humanity should be neglected. Where there is much shock or collapse, intravenous injection, or injection into the subcutaneous cellular tissue, of normal saline solution is of great value. Large quantities may be used. Besides producing a temporary restoration of the strength, such as may fit the patient for operation, these injections tend to eliminate the toxins by flushing the kidneys.

In peritonitis due to perforation, the prospect of recovery is in inverse ratio to the amount of delay in operating.

## CHRONIC PERITONITIS

Chronic peritonitis may be simple, tuberculous, or malignant.

**Simple Chronic Peritonitis** may be localised or general. The local form most commonly affects the capsule of the liver or spleen (*perihepatitis*, *perisplenitis*), where it may be sometimes recognised by a rubbing friction sound upon auscultation, and less commonly the intestinal peritoneum. In either situation it causes adhesions, and in connection with the intestine, these may form bands which lead to intestinal obstruction. The *general* form is common in association with cirrhosis of the liver, chronic Bright's disease, and chronic alcoholism. It sometimes follows a subacute attack, and may form part of a general serositis (*polyorhomenitis*), in which the pleuræ and pericardium also suffer. The peritoneum is greatly thickened, the mesentery and omentum are



shortened, the calibre of the bowel is diminished, and adhesions are numerous. Effusion may be moderate in amount, the fluid being divided by adhesions into separate compartments, or it may be extensive, the fluid being free in the abdominal cavity. Constipation is often a symptom. The physical signs are increased resistance, sometimes with friction, and evidences of a localised effusion of fluid or of ascites. Treatment consists mainly in removal of the cause, if that be possible, attention to the general health, and repeated tapplings.

**Tuberculous Peritonitis** may occur at any age, but is most common in children, in whom it may arise by direct infection from the bowel as a consequence of the ingestion of tuberculous milk, the bovine type of bacillus being frequently found in abdominal tuberculosis. In many instances, however, the abdominal lymphatic glands are the primary site of infection; and the disease is often associated with the presence of tubercle elsewhere. In women it is generally an extension from the Fallopian tubes, and in men it may follow testicular disease. It may complicate phthisis, and sometimes other serous membranes are affected at the same time. After childhood, it is most common between the ages of twenty and forty.

*Morbid Anatomy.*—The peritoneum is studded with tubercles, most abundantly on the under surface of the diaphragm and in the flanks. There are numerous adhesions, by which the intestines may be matted together. The omentum is often thickened and rolled up so as to form a sausage-shaped tumour lying across the upper part of the abdomen. The mesenteric glands may be much enlarged, especially in children, in whom they may form tumour-like thickenings (*tabes mesenterica*). Caseous masses lie between the matted coils of intestine. Locular collections of serum or pus may be present, or a moderate amount of free fluid, which is sometimes hæmorrhagic.

*Symptoms.*—The disease may set in acutely with considerable fever, meteorism, and abdominal pain; or the onset may simulate that of enteric fever. More commonly the condition is chronic from the outset, and in children the symptoms are gradual enlargement of the abdomen, with areas of dulness and resistance, and others of resonant percussion, sometimes with distinctly palpable nodules due to the enlarged glands, at other times with the sausage-shaped omental tumour mentioned above. The wasted limbs and thorax contrast with the abdominal enlargement. Diarrhœa is frequent, and there is often irregular fever. The pleura is sometimes involved.



In adults the *diagnosis* may be difficult, but it is assisted by the presence of tuberculosis elsewhere. For the tuberculin tests, see p. 365.

*Treatment.*—Rest in bed, with a plentiful supply of fresh air, and an appropriate diet are the chief general measures. If there is diarrhoea, milk alone must be given, but when diarrhoea and fever have subsided, the diet should be highly nutritive, and rich in fats. Cod-liver oil, creosote, or guaiacol are useful internally, and in children the syrup of the iodide of iron. Various local applications (iodoform, potassium iodide, etc.), are recommended in the form of ointment; the most useful is mercurial inunction, the mercurial ointment being rubbed once daily into the abdomen, which is then covered with a flannel binder. Many cases recover under this treatment; if the condition is obstinate, laparotomy with evacuation of the fluid is often followed by cure. Laparotomy is especially indicated in the ascitic type of case.

**Cancer of the Peritoneum** generally takes the form of a *malignant peritonitis*, although it may exist without inflammatory change. It is almost invariably secondary to cancer elsewhere, of which the stomach and ovaries are the most common seats, and it is most frequent in women. The peritoneal surface is studded with cancerous nodules which tend to cause it to pucker, and thus the omentum is drawn up into a cylindrical mass, like that described above in connection with tuberculous peritonitis. The intestine may also be narrowed, sometimes so much as to cause obstructive symptoms, but the most frequent symptom is ascites. The fluid is usually hæmorrhagic, and may contain the cell-groups of Foulis. When large, the tender nodules can be felt through the wasted abdominal wall. The skin about the umbilicus is sometimes infiltrated, and there may be an enlarged inguinal gland.



# DISEASES OF THE CIRCULATORY SYSTEM

## I. THE HEART

THE heart lies obliquely in the thorax, extending upwards to the lower limit of the second intercostal space. Its upper border is almost horizontal, and runs behind the sternum, just above the third costal cartilages, between two points respectively half an inch to the right and one inch to the left of the sternal margin. Its right border is formed by the right auricle, and runs in a curved line between a point three-quarters of an inch to the right of the articulation of the sixth rib with the sternum and a point in the second intercostal space half an inch to the right of the sternum. The left border, also curved, runs from the apex beat to a point in the second interspace an inch to the left of the sternum, and is formed by the left ventricle. The lower border joins the lower limits of the right and left borders, and is formed by the right ventricle. The apex beat lies in the fifth left intercostal space slightly internal to the nipple line and three and a half inches from the middle line of the body. The anterior surface of the heart is formed almost entirely by the right auricle and ventricle, only a thin strip of the left ventricle, which includes the apex, being represented at its left border. In children under twelve the heart is relatively broader, and lies higher in the chest, the apex beat being in the fourth interspace and also a little further to the left, in the nipple line.

The four valves lie close together, the pulmonic horizontally at the level of the third costal cartilage beneath its junction with the sternum, the aortic more deeply situated behind the sternum and running obliquely downwards from the lower border of the third cartilage, the mitral also running obliquely across the sternum at a slightly lower level, and the tricuspid running almost vertically downwards under the right half of the sternum between the fourth and fifth ribs. Hence auscultation over the site of the valves does not differentiate the sounds produced at each, and these sounds must therefore be listened for



at points where they are conducted to the surface (see Auscultation, p. 246).

*Events in the Cardiac Cycle.*—During the greater part of the long pause both auricles and ventricles are relaxed in diastole, and the blood-stream flows into the auricles from the great veins and is drawn into the ventricles by negative pressure. Towards the end of the pause occurs the short auricular systole, by which the ventricles are filled, and the auriculo-ventricular valves are floated up. This is immediately followed by the ventricular systole, which locks these valves and opens the semilunar valves. Its beginning coincides with the first sound and with the apex beat. It is followed by the ventricular diastole, at the beginning of which the semilunar valves are closed, and the second sound is produced, to be followed in turn by the succeeding long pause, and by a repetition of the cycle.

## PHYSICAL EXAMINATION<sup>1</sup>

### PERCUSSION

1. In health the area of *superficial cardiac dulness* is of triangular shape; its truncated upper angle reaches as high as the fourth left costal cartilage; the right border descends vertically along the left border of the sternum; the left border passes obliquely downwards and to the left until it reaches the outer limit of the apex beat; the base cannot be percussed out owing to the proximity of the liver, but corresponds to a line drawn from the outer and inferior limit of the apex beat inwards until it meets the perpendicular limit of cardiac dulness, about mid-sternum. The measurement of the basic line is important; normally it measures about three or three and a half inches.

2. The area of *deep dulness*, obtained by heavy percussion, corresponds in shape to the area of superficial dulness, but is more extensive. It overlaps it on the right by about an inch, on the left by about half an inch. It corresponds roughly with the actual size of the heart, while the superficial dulness represents that portion of the heart which is in direct contact with the chest-wall.

NOTE.—In disease the areas of superficial and deep dulness may be specially extended to the right or left, according as the right or left chambers of the heart are specially enlarged.

3. The percussion of the region of the *aortic arch* (above the level of the third costal cartilages) is especially important in cases of aneurysm and of mediastinal tumour.

### PALPATION

1. Of the *præcordia*.

(a) Determine the *position* of the apex beat. The normal position is between the fifth and sixth ribs, about half an inch within the vertical line of the nipple. When the left ventricle is enlarged

<sup>1</sup> This conspectus of the physical examination of the heart is mainly taken, by permission, from Dr. Wyllie's original notes and diagrams.



there is displacement of the apex beat downwards and to the left. When the right ventricle is enlarged there is diffuse pulsation to the right of the position of the apex beat, and there is often pulsation in the epigastrium.

- (b) Note the *limitation* or *diffusion* of the apex beat.
- (c) Note the force and character of the beat : whether moderate and deliberate, as in health ; or strong and sudden, as in nervous excitement ; or strong and slow (heaving), as in hypertrophy ; or irregular, as in dilatation ; or weak or imperceptible, as in debility. Note also the presence or absence of presystolic or systolic thrill.

The range of variation is considerable even in health, owing to the shape of the chest, etc. The beat may be imperceptible even in a healthy heart if the apex is overlapped by an emphysematous lung.

2. Of the *aortic region*. In cases of suspected aneurysm note presence or absence of pulsation or thrill.

3. Of the *great vessels at the root of the neck*. Venous pulsation is scarcely palpable, though strikingly visible ; arterial pulsation is as strikingly palpable as visible.

## INSPECTION

1. Of the *præcordia* and *aortic region*.

- (a) *Form*. Is there bulging over the præcordia or over the aortic region ?
- (b) *Movements*. (1) Movement of the apex beat ; its situation, amount, and diffusion ; (2) pulsation in the epigastrium ; (3) pulsation in the region of the pulmonary artery, common in anæmic debility ; (4) pulsation in the aortic region, often present in cases of aortic aneurysm.

2. Of the *great vessels of the neck*. (a) Fulness of the great veins ; (b) pulsation in these veins ; (c) excessive pulsation in the arteries.

3. Of the *general circulation* : as exhibited in the patient's complexion, the condition of his peripheral arteries and veins, the presence or absence of dropsy, etc.

4. Of the *pupils*, in cases of aneurysm of the aortic arch.

## AUSCULTATION

### A. THE CARDIAC SOUNDS

The areas in which sounds or murmurs produced at the different valves are to be listened for are as follows :—

- 1. Aortic area : the second right costal cartilage half an inch from the sternum.
- 2. Pulmonary area : the second left interspace close to the sternum.
- 3. Mitral area : the region of the apex beat.
- 4. Tricuspid area : the left edge of the sternum, just above its junction with the xiphoid cartilage.

Attention must be given to the characters of the sounds as well as to the existence of murmurs in each of these areas. Either the first or the second



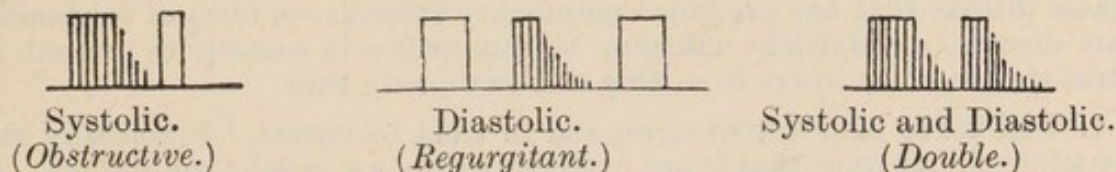
sound may be weakened or accentuated or reduplicated ; the first sound may be prolonged or booming ; or the cardiac rhythm may be altered, the long pause being shortened and the sounds succeeding each other like the tic-tac of a clock. Weakness of the first sound indicates cardiac failure.

## B. ENDOCARDIAL MURMURS OR BRUITS

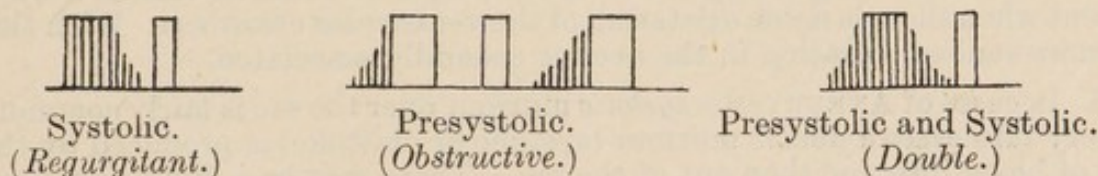
The first sound marks the beginning of systole. Systole continues through nearly the whole of the short pause.

The second sound marks the beginning of diastole. Diastole continues through nearly the whole of the long pause.

### 1. AORTIC—



### 2. MITRAL—



1. AORTIC MURMURS.—As shown in the above diagram, there may be at the aortic orifice a systolic murmur indicative of obstruction, or a diastolic indicative of regurgitation ; and these murmurs are frequently combined so as to constitute a double aortic murmur. A systolic murmur does not always indicate obstruction, as it may be due to simple roughening of the cusps, to dilatation of the aorta beyond the valve, or exceptionally to anæmia. Such a hæmic murmur is usually softer than a murmur of organic origin. Aortic murmurs, organic and hæmic, are produced at the aortic orifice, which is situated at the sternal articulation of the third left costal cartilage. They are thus basic murmurs.

2. MITRAL MURMURS.—These are heard best at the apex of the heart. A systolic murmur indicates regurgitation. This may be due either to organic disease of the valve, or to dilatation of the ventricle and its auriculo-ventricular orifice, the latter causing a "murmur of debility," owing to "disparity of size" between the dilated orifice and its valve ("relative insufficiency"). The obstructive murmurs are almost always organic, being due to stenosis of the mitral valve. Two forces cause the blood to flow through the mitral orifice during the diastole of the ventricle, viz., the suction of the ventricle (*a vis a fronte*), and the propelling force of the auricle (*a vis a tergo*). The suction is strongest near the beginning of the diastole, and the propelling force at the end of it, immediately before the ventricular systole. When there is obstructive disease, the murmur is developed at the time when the flow of blood through the contracted orifice is rapid enough to produce a murmur. Generally it is limited to the period of auricular contraction, and is therefore presystolic. Sometimes, when the auricle is weak, it occurs only at the period of greatest ventricular suction, and is therefore diastolic. Occasionally, again, it may be both diastolic and presystolic. Obstructive murmurs are rough and purring. They are frequently succeeded by the



blowing murmur of regurgitation, since the disease which produces obstruction often renders the valve incompetent at the same time. A common double mitral murmur is thus a rough, obstructive, presystolic, running up to, and immediately succeeded by, a blowing, systolic, regurgitant murmur. The relations of these murmurs to each other are shown in the diagrams. In sound the rough presystolic murmur might be represented by the letters *rrrp*, the terminal *p* representing the first sound of the heart, which, in such cases, is often loudly accentuated. A double mitral murmur may be represented by the letters *rrrfff*.

3. MURMURS AT THE PULMONARY ORIFICE correspond in time to aortic murmurs. A *systolic* murmur in this situation is fairly common. It may be "functional," and due to hæmic causes, as anæmia; or it may be due to organic disease that has produced pulmonary stenosis—a form of congenital heart disease. A *diastolic* murmur at this orifice is sometimes present in mitral stenosis, but apart from this it is extremely rare.

4. TRICUSPID MURMURS correspond in time to mitral. Practically the only tricuspid murmur that is not extremely rare is a *systolic regurgitant*. It is a "murmur of disparity of size," and is due to enlargement of the orifice without corresponding enlargement of the cusps, a condition that is always present when there is much dilatation of the ventricular chamber. With this murmur venous pulsation in the neck is generally associated.

5. In cases of ANEURYSM a *systolic* murmur over the sac is fairly common. In very rare cases a *double* murmur (systolic or diastolic) is produced by the flow of blood into and then out of the sac. There may be a double murmur in aneurysm of the first part of the arch, where aortic regurgitation co-exists with the aneurysm. In many cases there is no murmur at all.

6. The MURMURS OF DEBILITY, so common in anæmia, are both vascular and cardiac.

The *Vascular* murmurs are (a) The *Arterial* murmur, systolic in time, heard over the great arteries of the neck. This is generally supposed to be common, but it is very often produced artificially by the pressure of the stethoscope. (b) The venous hum (the humming-top murmur, or "Bruit de Diable"), heard over the great veins of the neck, and sometimes over other large veins, such as the ophthalmic veins and the cerebral sinuses. This is very common and important.

The *Cardiac* murmurs of debility are variously classified and explained, but there is as yet no consensus of opinion as to their origin. They are all systolic in rhythm, and usually soft and blowing in character. In order of frequency, they may be heard in the pulmonary, mitral, tricuspid, or aortic area; but aortic systolic murmurs are very rarely functional.

Of these *six* murmurs (vascular and cardiac) *three* are common in anæmia, namely, (1) the bruit de diable in the neck, (2) the basic murmur in the pulmonary region, (3) the mitral murmur at the apex; and that is the order of their development.

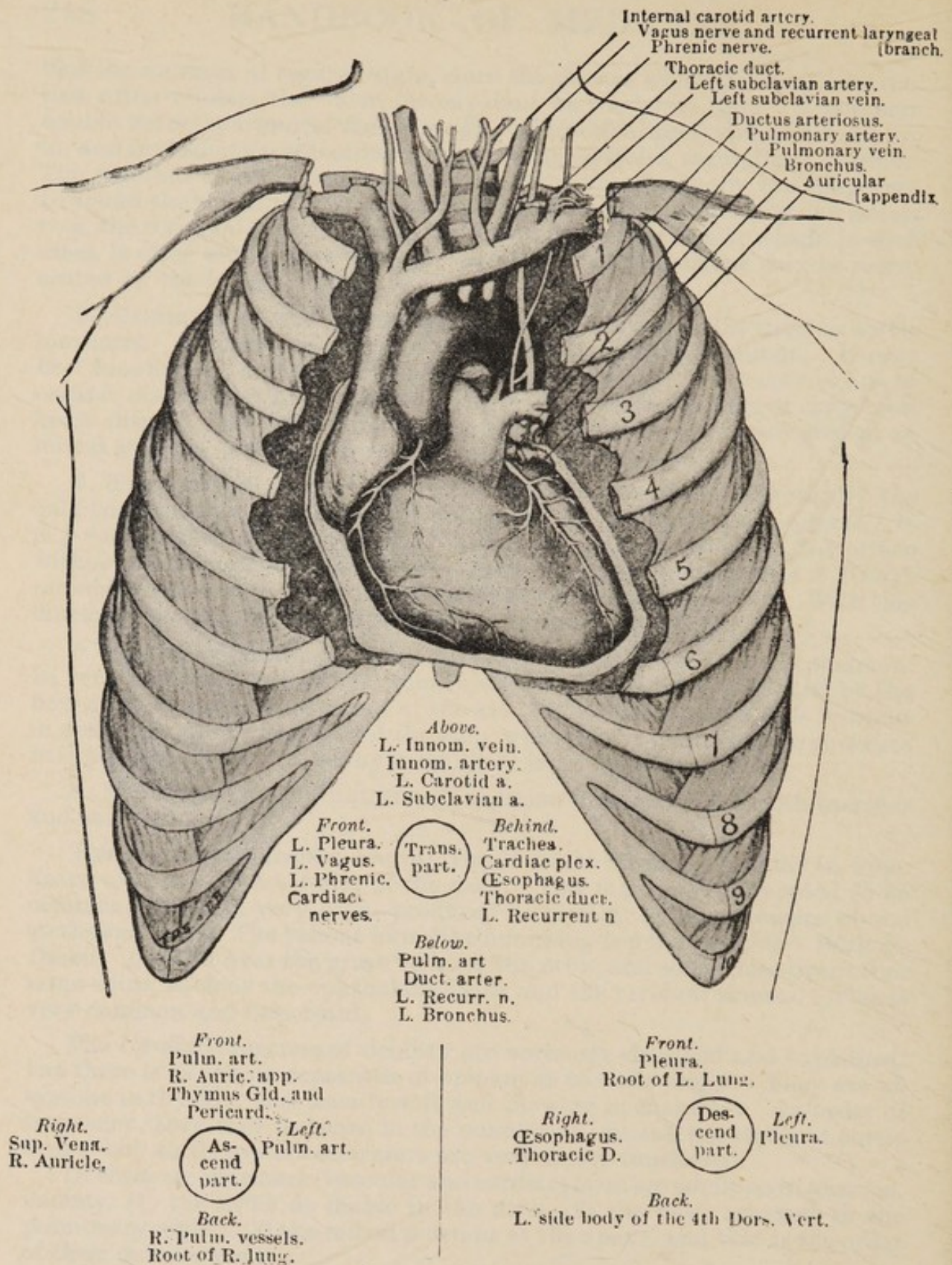
Observe that all the cardiac murmurs of debility are *systolic* in time. Systolic murmurs may thus be either of functional or of organic origin, while presystolic and diastolic murmurs are always of organic origin.

7. EXOCARDIAL MURMURS.—(a) *Pericardial friction*, due to pericarditis, is generally a "to-and-fro" or double murmur (systolic and diastolic). It is most apt to be confounded with a double aortic murmur, but its superficial rubbing and shuffling character generally renders the distinction easy. In most cases it appears first at the base of the heart, and spreads thence, if not





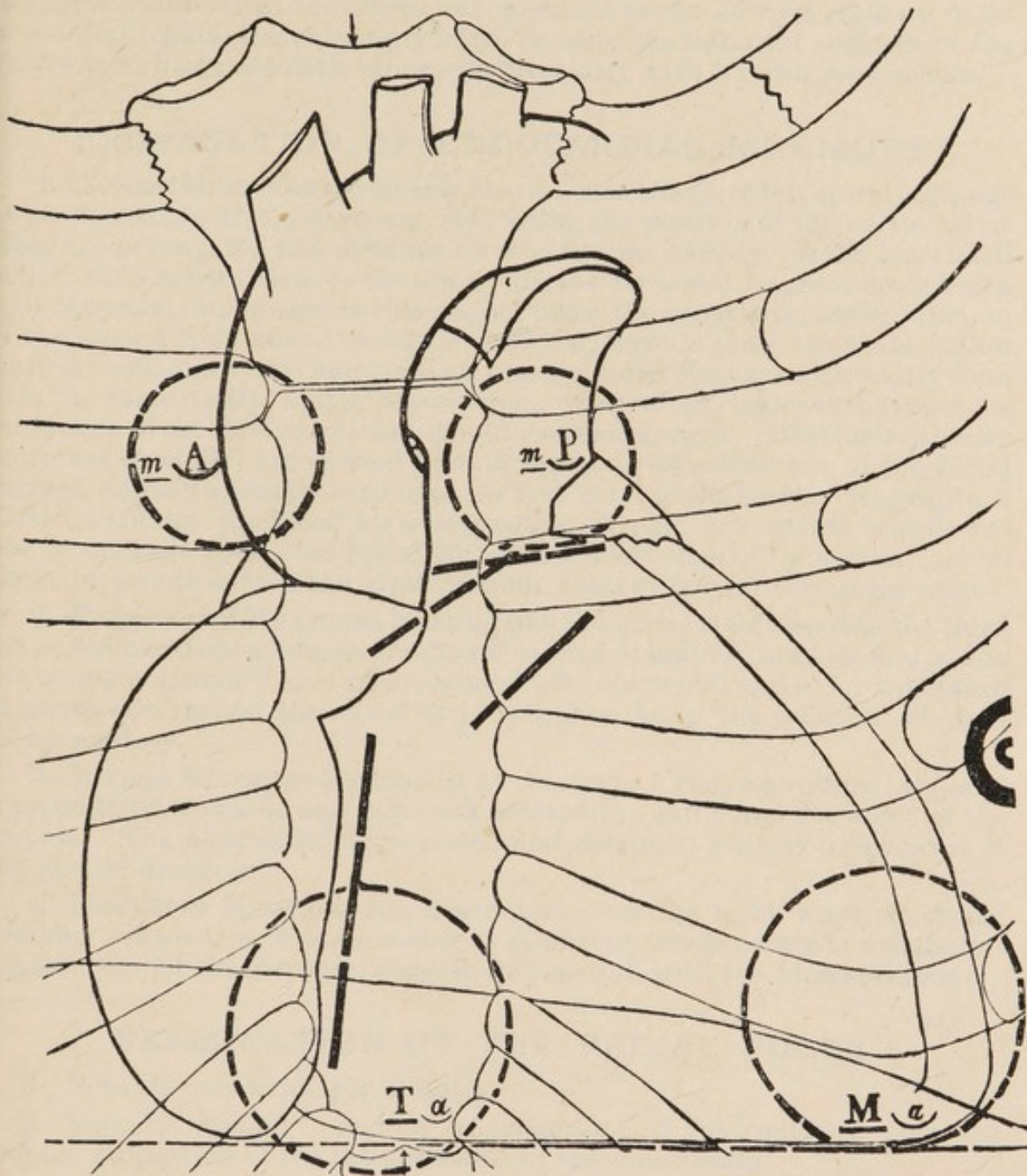




Position of heart and great vessels relatively to anterior wall of chest. In front the right ventricle, above and to its right, right auricle, into which opens superior vena cava; directly above it the pulmonary artery and remains of ductus arteriosus; to its left the portion of left ventricle seen on anterior surface of heart, and at upper end a portion of left auricular appendix. The arch of aorta arises from left ventricle posteriorly. The relations of its ascending, transverse, and descending parts are shown diagrammatically above.

(From Savill's "Clinical Medicine," by kind permission of Mr. Edward Arnold.)





Projection of the position of the cardiac valves on the anterior surface of the chest. The pulmonic valve is represented by the tripartite line running horizontally along the inner part of the third left costal cartilage; the aortic runs obliquely downwards from it towards the middle of the sternum; the mitral, beginning at the lower edge of the cartilage, crosses the third interspace; and the tricuspid runs nearly vertically along the sternum, separately from the other valves. The broken circles, M., P., A., T. indicate the areas in which mitral, pulmonic, aortic, and tricuspid sounds or murmurs are best heard.

(From Ewart's "Cardiac Outlines," by kind permission of Messrs. Baillière, Tindall & Cox.)







arrested, over the whole organ. (b) A to-and-fro friction sound, of precisely the same character as the above, is sometimes produced by a *pleurisy in the præcordial region*, the subjacent heart causing the inflamed surfaces of the pleura to rub against each other synchronously with its own movements.

### PROPAGATION OF ENDOCARDIAL MURMURS

1. **AORTIC MURMURS** are clearly heard about the third left costal cartilage at its junction with the sternum, that being the position of the aortic valve. They are propagated to a distance by *three* agents, namely, (a) the *heart* itself which often carries them to the apex; (b) the *aorta* and its great branches, a spot of special importance in this respect being the *aortic area*, at the junction of the second right costal cartilage with the sternum (here the aorta makes its first bend, and aortic murmurs are usually heard even more distinctly than over the valve itself); and (c) the *sternum*, which often conducts the sonorous vibrations of such murmurs throughout its whole length. Obstructive aortic murmurs (systolic) are carried best upwards, in the direction of the blood current, and are specially loud over the first bend of the aorta. Regurgitant aortic murmurs, produced by a descending current, are carried best downwards; and are very often heard better at the left edge of the sternum, close above its junction with the xiphisternum, than even over the aortic valve.

2. **PULMONARY MURMURS**, starting like the aortic from opposite the third left costal cartilage at the sternum, are carried obliquely upwards and to the left to the pulmonary area in the second left intercostal space, for a distance of about two inches, the agent of propagation being the trunk of the pulmonary artery.

3. **MITRAL MURMURS** are loudest at the apex. The regurgitant (systolic) is propagated upwards and outwards toward the axilla and the angle of the scapula. The obstructive (presystolic and diastolic) are not propagated in any special direction.

4. **TRICUSPID MURMURS** are heard best over the right ventricle, being audible over an area of some inches in diameter, whose centre is situated at the left edge of the sternum, close to its junction with the xiphisternum.

### EXAMINATION OF THE RADIAL PULSE

1. Note the *pulse rate* per minute.

2. Note its *rhythm*: regular or irregular; if intermittent, note the average proportion of the intermissions to the pulse-beats.

3. Note the size, force, and character of the *blood-wave*: large, moderate, or small; deliberate or sudden; strong or weak; a double wave (dicrotism).

**NOTE.**—In *weak heart* the radial pulse may be almost or wholly imperceptible; or only a proportion of the heart's contractions may produce blood-waves sufficiently strong to be propagated perceptibly to the radial artery, and thus the radial pulse may appear to be much slower than the rate of the heart's contractions; or the weak pulse may be affected by the patient's respiration, its beat being weakened by inspiration and strengthened by expiration.

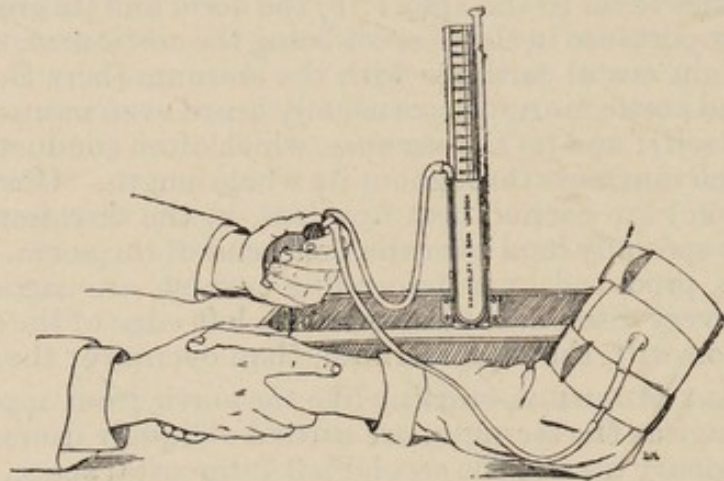
4. Between the beats of the pulse, test particularly the *resistance* of the artery to pressure. Marked resistance may be due either to rigidity of the artery's coats or to high blood-pressure. Press the artery firmly against the bone, and examine the coats by rolling it beneath the finger. The pressure may be more accurately estimated by the sphygmomanometer.



5. Compare the two radial pulses, and note any *difference in strength*, or *want of synchronism*, in their beats.

6. In special cases take a sphygmographic tracing. While the trained finger is the most convenient instrument for the examination of the pulse, and the best for appreciation of the characters of the arterial wall, the sphygmogram both indicates the period during which arterial pressure is sustained, and constitutes a permanent record. The student should be familiar with the use of the *sphygmograph*, and with that of the *sphygmomanometer*, by which the *height* of the arterial blood-pressure is estimated.

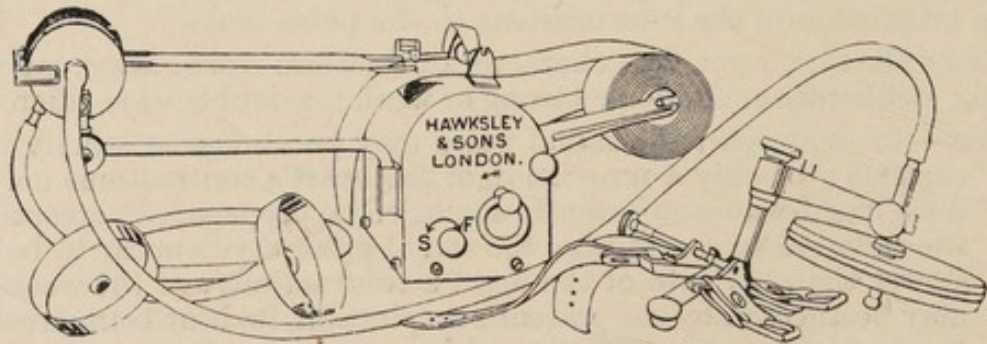
The sphygmomanometer most in use is that of Riva-Rocci, or one of



Riva-Rocci Sphygmomanometer modified (Martin).

its modifications. It consists in principle of a broad, distensible bag, which surrounds the upper arm like a cuff, and is connected with a mercurial manometer. Air is pumped into it by a rubber hand-pump until the pressure is sufficient to stop the radial pulse. By means of a screw valve, the air is allowed to escape very slowly, and the pressure at which the pulse begins to return is read off on the manometer, and regarded as the systolic pressure. The point of maximum oscillation of the mercurial column is taken as the diastolic pressure. The average systolic pressure in a healthy man of thirty is about 120 mm. of mercury. It is rather less in children and women, and rises to 140 or more with increasing years.

By means of the *polygraph* simultaneous tracings of the pulsations of



The Polygraph (Mackenzie's).

the radial artery and jugular vein may be obtained, while the *electrocardiograph* registers the changes in potential produced by the muscular action of the different parts of the heart.



## PERICARDITIS

Inflammation of the pericardium is never idiopathic. It may arise by direct extension of inflammation, from infective processes, or from constitutional diseases.

**Exciting Causes.**—

- (1) *Rheumatic fever* and chorea.
- (2) Other specific infections :—*scarlet fever*, measles, septicæmia and pyæmia, pneumonia ; and less commonly variola, enteric fever, and influenza.
- (3) Tuberculosis.
- (4) Malignant disease.
- (5) Chronic Bright's disease, gout, scurvy, and sometimes diabetes.
- (6) Extension of inflammation from neighbouring parts.
- (7) Injury.

The disease is rather more frequent in males than females. Rheumatic cases occur chiefly in children or early adult life, renal and gouty cases later. Apart from the rheumatic cases, the organisms most frequently present are the pyogenic cocci and the pneumococcus.

**Morbid Anatomy.**—In fibrinous pericarditis, the usual form, the stages are—

1. Hyperæmia, with loss of lustre.
2. Exudation of fibrin, which gives the pericardial surfaces a peculiar shaggy, or “bread-and-butter-sandwich” appearance (*cor villosum*). The process may stop at this stage, constituting the dry or *plastic type of pericarditis*. Much oftener it goes on to the next stage.
3. Effusion of fluid, serous or sero-fibrinous.
4. Absorption, resulting in slight or extensive adhesions, which may permanently hamper the cardiac action.

*Suppurative pericarditis* is due to septic infection (pyæmia, local processes), and the fluid is generally purulent from the onset. In tuberculous or malignant pericarditis the effusion may be hæmorrhagic.

In almost all cases the myocardium shares in the inflammatory change, and is sometimes profoundly affected. In some the mediastinal tissues are also inflamed (*mediastino-pericarditis*).

**Symptoms.**—The symptoms are somewhat obscure, and may be masked by previously existing disease. Taking a typical



case as it occurs in the course of rheumatic fever, we usually find—

1. Præcordial distress. Sharp pain is rare, but when present it is most marked at the lower end of the sternum.

2. Moderate fever, or exacerbation of already existing fever, at the onset.

3. *Dyspnœa*, short cough, and in severe cases  *dusky appearance* of the face.

4. Rapid action of the heart, sometimes with feeble pulse.

5. Symptoms due to pressure by the fluid on the neighbouring organs (trachea and œsophagus, etc.).

6. Great restlessness.

These symptoms, it must be understood, are due to the presence of fluid effusion. If the pericarditis remains dry, there may be no symptoms, or at most slight præcordial pain.

It is evident that we must rely more upon physical signs than subjective symptoms for diagnostic information.

**Physical Signs.**—1. *Before effusion of fluid.*—On auscultation is heard the characteristic “*to-and-fro*” friction rub, variable in intensity, and not accurately synchronous with the cardiac sounds. As a rule it is a double sound, approximately systolic and diastolic; but it may have a triple or cantering rhythm. It usually begins at the base of the heart, and then extends more or less over the whole surface. The friction may sometimes be felt by the hand (friction fremitus). It becomes less pronounced as the effusion increases, but is *rarely entirely absent at the base* until complete resolution or organisation takes place.

2. *Effusion stage.*—The physical signs are—

(1) *Marked increase of the cardiac dulness.*

(2) Displacement of the apex beat.

(3) Muffling of the heart sounds.

(4) Displacement of other organs (if effusion be great).

*The shape of the dulness is characteristic.* It is conical, the apex of the cone being truncated, and situated at the level of the second rib, owing to the close attachment of the pericardium to the great vessels at this point. The apex beat is generally pushed upwards and to the left. It lies, when it is palpable at all, distinctly *within* the left border of cardiac dulness, not, as in enlargement due to valvular disease, in close relationship to it. The marked distension of the pericardial sac surrounds the heart with fluid, and causes a dulness extending much beyond the



limits of the organ itself. The amount of bulging and displacement of organs will, of course, vary with the amount of fluid present. As resolution takes place the friction returns, and may be very coarse in character. Muffling of the heart sounds is not always present, and is not entirely due to the presence of fluid, for the foetal heart is quite distinctly heard through an amount of fluid greater than is usually present in pericarditis. The muffling is therefore due mainly to weakness of the cardiac muscle from accompanying myocarditis, although where the quantity of fluid is very great, in serous and chronic pericarditis, this may in part account for it. In *children* the onset is often insidious, and may precede any obvious signs of rheumatism.

**Diagnosis** is sometimes difficult in fat people, but the main difficulty is to distinguish between pericarditis and dilatation of the heart. The upper limit of dulness is higher in pericarditis, and its shape is *conical*; in dilatation the sounds are not muffled. Pleuritic friction has a different rhythm, and pleuropericardial friction is heard at the *border* of the cardiac dulness.

**Prognosis** depends on the cause; that of simple sero-fibrinous pericarditis is good, and the fluid may be absorbed in a comparatively short time. Permanent damage to the heart may result from complication with endocarditis or myocarditis, or from extensive adhesions. The greater the amount of fluid, the worse is the prognosis, which becomes very grave if the fluid is purulent or hæmorrhagic. Pericarditis complicating pneumonia or renal disease is often fatal.

*Adherent Pericardium*.—After absorption of the fluid, the sac may be almost or quite obliterated by adhesions. Symptoms of this condition may remain latent, but in young people, in whom the parietal pericardium has become fixed to the chest-wall and pleura by coincident mediastinitis, the following signs may be found—

1. Great hypertrophy and dilatation of the heart.
2. Retraction of the apex during *systole*, and fixation of the apex.
3. Diastolic rebound of the chest-wall.
4. Collapse of the cervical veins during diastole (Friedreich's sign).
5. *Pulsus paradoxus*, the pulse becoming smaller at the end of inspiration.

None of these signs is by itself pathognomonic, but taken together they establish the diagnosis. The prognosis is bad.



**Treatment.**—

1. *Absolute rest in bed.*
2. In the early stages, ice-bags or leeches to the præcordia ; warm applications or a small blister if ice fails to relieve pain. Morphia may be necessary.
3. Treat the cause—if due to rheumatism be careful in the use of salicylates. They perhaps tend, if given early, to prevent cardiac lesions ; but when these are established, their depressant action in large doses may be harmful. Believing in their specific action, Lees pushes them, as detailed under Rheumatism (p. 67), but symptoms of poisoning must be watched for if this treatment is employed. Digitalis or stimulants may be needed in cardiac failure.
4. Paracentesis should be performed if the effusion is very great, and does not tend to become less, or produces urgent cardiac symptoms.

To promote absorption after the acute symptoms have subsided—

1. Blister.
2. Employ diuretics and saline laxatives. Iodide of potassium is also useful.

When these fail, paracentesis should be performed. The puncture should be made through a skin incision one inch from the left sternal margin in the fourth or fifth interspace. If the fluid be purulent, incise and drain. If an organism can be isolated from the fluid, vaccine treatment may be employed.

## ACUTE ENDOCARDITIS

By endocarditis is meant inflammation of the lining membrane of the interior of the heart, the valves being most commonly affected. It may be acute or chronic. The *acute* form is divided into the simple or benign and malignant or ulcerative forms.

**Etiology.**—Endocarditis is rarely a primary disease, but like pericarditis, it is secondary to other affections. Its causes are similar to those of pericarditis. The simple form is closely associated with rheumatism and chorea ; it occurs in scarlet fever, but is uncommon in the other infections of childhood ; it is often met with in pneumonia, sometimes in enteric fever and in phthisis ; and it may occur in Bright's disease, diabetes, or gout.

Malignant endocarditis may be primary, or at least the source



of infection may not be discoverable, but it is much more frequently the result of some septic or infective condition, such as diphtheria, scarlet fever, puerperal sepsis, gonococcal infection, pneumonia, septicæmia, or pyæmia. It very often attacks valves already the seat of chronic endocarditis. It is also associated with rheumatism and chorea, but not nearly so often as simple endocarditis.

The distinction between simple and malignant endocarditis, it should be noted, is by no means absolute. The same organisms may be found to exist in either form, and even clinically a case may commence as simple, and end as malignant endocarditis, or *vice versa*. In the majority of cases, however, the distinction is sufficiently marked to warrant a differentiation between the two types.

**Morbid Anatomy.**—*Simple Form*—

1. Cloudiness, followed by œdematous thickening of the valvular endocardium.
2. Superficial erosions, and formation of small granulations.
3. Deposit of layers of fibrin and corpuscles from the blood, the whole process resulting in the formation of small warty vegetations. These vegetations are most marked at a slight distance from the free borders of the valves—*i.e. those parts which come into apposition during closure*. In the course of time they are transformed into fibrous tissue.

According to Poynton and Paine the infective organisms are conveyed to the base of the valve by the capillaries, and thence pass to the subendothelial tissue by the minute nutrient channels in the valvular substance; others hold that the organisms are derived from the blood circulating over the surface of the valves.

*Malignant Form (Ulcerative Endocarditis).*—The initial changes are similar, but there are some important differences, inasmuch as ulcerations may *completely replace the vegetations*. The appearances differ as follows from those of simple endocarditis—

1. The vegetations when present are larger and fungating.
2. The underlying tissues are necrotic, and show loss of substance and round-celled infiltration.
3. They contain masses of micrococci, while in simple endocarditis the organisms are scanty. The two forms cannot be distinguished by the organisms producing them; either simple or malignant endocarditis may arise from a pyogenic infection.



4. When the vegetations become detached they form *septic emboli*, giving rise to metastatic abscesses.
5. The ulcerative process causes great destruction of the valves, and may even lead to perforation of the curtains.
6. The subsequent or permanent changes in the valves, if the patient survive, are much more marked.
7. If the vegetation touches the mural endocardium as it flaps to and fro, the part touched becomes affected by contact.

As regards the side of the heart most affected—

1. *Congenital* endocarditis attacks the right side of the heart (but note that many congenital cardiac lesions are due not to endocarditis, but to developmental faults).
2. The simple endocarditis attacks the left only.
3. The malignant attacks *both sides*, though the *left* is much more implicated than the right side.

The vegetations are upon that side of the valve opposed to the blood-stream—viz. at the aortic valve the vegetations project into the ventricle, at the mitral valve into the auricle.

As in pericarditis, the myocardium almost always shares in the inflammatory affection, and may be either slightly or extensively involved. The auriculo-ventricular node or the bundle of His may be affected. Pericarditis is a common complication.

**Symptoms.**—*Simple Endocarditis.*—The signs are often extremely ill marked; possibly increased rapidity of pulse, slight dyspnoea or præcordial distress, etc., may attract attention to the heart. More commonly the condition is discovered during the daily routine examination, when some dilatation of the heart, from the accompanying myocarditis, may be found, and a *recently developed murmur* of a soft blowing or bellows-like character may be heard in the mitral or aortic areas. The commonest murmurs are those of mitral regurgitation (systolic), or mitral stenosis (presystolic).

It should be remembered, however, that in most fevers the heart is somewhat dilated, and a murmur, *not due to endocarditis*, may be present, particularly in rheumatism, where marked anæmia is so common. We must therefore be cautious in coming to a too rapid conclusion that a suddenly developed murmur is indicative of endocarditis. An important distinction is that the onset of endocarditis is often accompanied by a smart rise in temperature above the previous level, while in hæmic murmurs,



or those due to simple dilatation, this is absent. In other cases fever may persist after the pain and swelling of the joints are gone. A diastolic murmur in the aortic area is likely to be organic (aortic regurgitation). In many instances a rise in the pulse-rate, or irregularity of the pulse, is the first sign of endocarditis.

*Malignant Form.*—Three types may be distinguished—

1. *The Septic Type* is characterised by the symptoms of septic infection—viz. rigors, sweats, oscillating temperature, emaciation and metastatic abscesses. The symptoms may continue for months.
2. *The Typhoid Type* is characterised by irregular or intermittent fever, looseness of the bowels, petechial rashes, and a rapid assumption of the typhoid state. Great difficulty may be experienced in distinguishing this form from typhoid fever or meningitis.
3. *The Cardiac Type* is that in which symptoms of acute endocarditis, with fever of a septic type, appear in the course of a chronic valvular lesion. In some of these cases death is rapid; others may recover after a protracted illness.

Along with these general symptoms there are usually definite cardiac signs—development of murmur, dilatation of the heart, cardiac irregularity, and so on. But the cardiac symptoms may be altogether latent, causing difficulty in diagnosis. Enteric fever and miliary tuberculosis are the chief sources of confusion. In malignant endocarditis there is leucocytosis, in enteric leucopenia; in endocarditis the murmur may change its character, or fresh murmurs may appear. In enteric the Widal reaction is present; in endocarditis blood-culture may show the presence of an organism. In miliary tuberculosis tubercle may be present in other regions of the body, and pulmonary signs are often marked.

In both simple and malignant endocarditis vegetations may be detached from the affected valve, and plug the smaller arteries. In the simple form these *emboli* are of importance when they implicate end-arteries (brain, lungs, kidneys, spleen), and they then result in hemiplegia, hæmorrhagic infarction of the lungs, hæmaturia, and so on. In the malignant form the same results are produced when end-arteries are affected, but an abscess subsequently forms at the seat of the embolus, while in the case of other arteries metastatic abscesses ensue. Obviously the risk of detachment is greater in the ulcerative type.



It should be remembered that patients with chronic valvular disease may have frequent attacks of subacute endocarditis.

We must also remember that endocarditis is never a solitary process. To some extent the myocardium always shares in the inflammatory changes; and the prognosis, in cases of chronic endocarditis following upon the acute form, depends as much upon the condition of the cardiac musculature as upon the actual valvular lesion.

**Treatment.**—All forms of endocarditis require absolute rest, which should be prolonged for weeks or even months. The primary disease should be treated, and what has been said under pericarditis of the use of salicylates applies also to endocarditis. *Over-stimulation* of the heart must be avoided, and it is in acute endocarditis that most harm is likely to be done by the indiscriminate use of digitalis, though it may be called for if the heart is failing. Rest, light diet—milk while fever is present—attention to the bowels and to sleep, form the best treatment of simple endocarditis. The malignant form should be treated like septicæmia. If the organism can be isolated from the blood, anti-streptococcic serum or a vaccine may be tried, but under any treatment most cases have a fatal ending.

### CHRONIC ENDOCARDITIS AND VALVULAR DEGENERATION

The main causes of chronic valvular disease of the heart are chronic endocarditis and degenerative changes in the curtains of the valves. Somewhat similar changes—namely, sclerosis and thickening of the cusps and of the base of the valves—are to be found in both conditions; but the sclerosis may be inferred to be due to chronic endocarditis when there is a history of a previous acute attack due to rheumatic fever, or even when there is a clear history of rheumatism without acute endocarditis. It will be remembered that other infections may also, though less commonly, cause the acute disease, and therefore the chronic form.

The conditions which may cause degenerative changes are syphilis, alcoholism, chronic Bright's disease, gout, and vascular overstrain, however produced. Most of these favour the development of high blood-pressure, and consequently of arteriosclerosis and atheroma, the valvular lesion being often consecutive to degenerative changes in the arch of the aorta, and therefore usually affecting the aortic valve. Such lesions arise at a later period of life than those which follow rheumatism, and



are most common about middle age. Syphilitic disease of the valves, on the other hand, may arise either in early or in later adult life. Like the degenerative lesions, it affects the aortic oftener than the mitral valve, but may also implicate the latter. Rheumatic endocarditis most frequently attacks the mitral valve, although it may involve the aortic also, or attack it singly. Aortic lesions, then, often originate in middle life, mitral lesions as a rule in early adult life.

**Morbid Anatomy.**—The principal changes to be found in chronic valvular lesions are as follows :—

1. Thickening and stiffening of the cusps, often with warty vegetations, firmer than those of the acute disease, at their margins. The thickening may affect the whole valve or the marginal portion only.

2. Shortening of the cusps, which thus become incompetent ; or adhesion between their margins, causing stenosis of the orifice. Incompetence and stenosis may coexist.

3. In the aortic valve, the cusps are usually curled inwards towards the wall of the vessel, and the coronary arteries are often implicated ; in the mitral valve, the cusps are often adherent along the whole length of their margins, leaving a slit-shaped orifice at the bottom of a funnel (mitral stenosis), and the fusion may extend to the chordæ tendineæ and musculi papillares.

4. Calcification or atheromatous change in the fibrotic cusps.

5. The changes may extend to the mural endocardium ; and the auriculo-ventricular node or bundle may be affected.

## EFFECTS OF CHRONIC VALVULAR DISEASE

We have seen that as a result of inflammatory or degenerative affections very serious structural changes occur in the valves of the heart. We must now consider in detail the effects of such morbid changes.

In order that the heart should carry out its function of supplying the organs and tissues with the necessary amount of oxygenated blood, it must be perfect in its structure and properly nourished. Serious interference with this function may be brought about by—

- (1) Disorders of the cardiac innervation.
- (2) Defective cardiac nutrition.
- (3) Valvular or myocardial defects.



It is with the last of these causes that we are here concerned.

In studying the mechanism of chronic valvular lesions, it is well to bear in mind three important points :—

1. The normal cardiac mechanism is adapted to meet a certain amount of sudden strain, working under ordinary circumstances with a large reserve of power.

2. This reserve power is developed, and *actually increased under increased strain, provided the heart is adequately supplied with blood*, and the strain *gradually* applied.

3. Notwithstanding the existence of this reserve, a time comes when reserve force must fail, and symptoms of heart failure develop. In other words, hypertrophy keeps up the balance for a time; but ultimately dilatation becomes disproportionate to the hypertrophy, and an enfeeblement of the heart begins, which ends in complete failure.

Let us take for an example, although it seldom exists as an isolated lesion, the condition of aortic stenosis, a narrowing of the aortic orifice which leads to an obstruction to the outflow of blood. The first effort will be an extra strain upon the valve and chamber behind (left ventricle). Under the extra strain the chamber at first dilates *slightly*, as the ventricle has no previous preparation for the sudden establishment of a lesion in front of it. But the reserve energy of the myocardium is soon called into play, the ventricle contracts more forcibly to overcome the obstruction, and its walls begin to hypertrophy. If the narrowing is slight, a moderate degree of hypertrophy will be enough to overcome the obstacle without exhausting the cardiac reserve, and this condition may remain unaltered for a very long time. But the lesion may be progressive, and as the narrowing increases, so must the hypertrophy increase to overcome it, until in course of time it may come to be very great. The obstruction to the onflow of blood is thus met by more forcible and more prolonged contractions, the arterial system remains well supplied, and for a time no bad symptom may develop. This is known as the "stage of compensation" of the valvular lesion. Finally, however, the reserve energy of the ventricle becomes exhausted, owing to prolonged overwork, aided perhaps by external causes, such as faulty regulation of habits, diet, or exercise, or mental worry and anxiety. Then the exhausted muscle yields to the strain, and dilatation becomes more prominent than hypertrophy. The walls of the ventricle are carried apart, and in consequence the curtains of



the mitral valve, though healthy, are no longer able to close the orifice ("*relative insufficiency*"). Regurgitation of blood into the auricle (mitral regurgitation) follows, and upon that engorgement of the pulmonary circulation.

The right ventricle is now called upon to force the blood through the engorged lungs to the left side of the heart, and it must obviously pass through changes similar to those that affected the left ventricle. But, as it is a less muscular structure, these changes are completed sooner. Relative tricuspid insufficiency is established, and with it the last stage, known as "failure of compensation," which will be subsequently discussed.

The aortic symptoms first developed may thus *become masked by mitral symptoms*, although death may ensue before these appear.

It is evident that in cases of disease of the mitral valve, causing either back-flow of blood into the left auricle or obstruction to the passage of blood from the auricle into the left ventricle, the right ventricle is the main compensatory force, the capacity for hypertrophy of the left auricle being only limited. Dilatation of the left auricle is therefore followed by moderate dilatation, and then by hypertrophy, of the right ventricle, while the left ventricle is either not hypertrophied or only moderately so.

The duration of compensatory hypertrophy, in each individual instance, depends *mainly* upon the extent to which the myocardium has been involved in the original endocarditis. With an efficient musculature a serious valvular lesion may be longer compensated than a slight one where the musculature is disabled.

Another factor of importance is the age at which the lesion has originated, young muscle being capable of much greater hypertrophy than that of the elderly. Lesions which involve the coronary arteries, as in many cases of aortic disease, interfere with the blood-supply of the cardiac wall, and in these cases also the period of compensation is likely to be brief. Apart from its share in the causative endocarditis, the myocardium may be weakened as the result of the patient's habits (alcoholism), when early cardiac failure may be anticipated.

## FAILURE OF COMPENSATION

Upon the establishment of relative tricuspid insufficiency (*v. supra*) there follows at once regurgitation of blood into the right auricle (tricuspid regurgitation), which becomes engorged, and



offers an obstacle to the return of blood from the systemic veins. As the mitral lesion led to engorgement of the pulmonary, so does the tricuspid lead to engorgement of the systemic circulation. There is thus established a state of *passive congestion* of the great internal organs and of the subcutaneous circulation—and also, in extreme cases, of the serous cavities. Cough, some, times with hæmoptysis, dyspnœa or orthopnœa, lividity of the face, palpitation, insomnia, and irregular action of the heart are the chief symptoms of failure of compensation as it affects the right ventricle.

The *physical signs* are of two classes:—

I. Direct signs of tricuspid regurgitation.

1. Systolic murmur in the tricuspid area.
2. Weakness of the second pulmonic sound (part of the blood which should flow into the pulmonary artery regurgitating into the right auricle).
3. Marked irregularity of the pulse.
4. Venous pulse, best seen in the neck (there being no valves between the jugulars and the incompetent tricuspid valve), and also in the liver (hepatic pulse).

The study of the venous pulse, rendered possible by the use of the clinical polygraph, and the observation of the auricular contractions by means of the electrocardiograph, have of late years thrown much light on the subject of cardiac irregularity. By these means the permanently irregular pulse of the late stages of valvular, and particularly of mitral, lesions has been shown to be due to auricular fibrillation, a condition in which the orderly contraction of the auricle as a whole is replaced by inco-ordinate and independent contractions originating at different points of the auricular wall (*see Arrhythmia*, p. 281).

In each cardiac cycle three waves, the “a,” “c,” and “v” waves, are normally found in a polygraphic tracing taken from the jugular vein. The “a” wave is produced by auricular systole, the “c” wave by the transmitted pulsation of the carotid artery, and the “v” wave towards the end of ventricular systole. In a case of auricular fibrillation the “a” wave disappears, its place being often taken by a series of abortive wavelets which barely raise the lever of the instrument. Similarly it is found on examining electrocardiographic records that the P wave characteristic of auricular systole disappears from the tracing in cases of this nature.



## II. Signs of passive congestion.

1. Œdema, beginning where the circulation is normally at the greatest disadvantage, *i.e.* in the feet and ankles, and extending gradually upwards till the trunk and arms may be affected.
2. In grave cases ascites, hydrothorax, hydropericardium.
3. Congestion of internal organs :—
  - (a) Liver. Enlargement, tenderness, slight icteric tinge (*see* Passive Congestion of the Liver, p. 229).
  - (b) Kidneys. Scanty, high-coloured urine, abundant urates, albumin in varying amount, a few hyaline casts and isolated red-blood corpuscles. Urea not diminished.
  - (c) Stomach. Dyspepsia, catarrh, often hæmatemesis.
  - (d) Lungs. Signs of congestion and œdema; fine moist râles and dulness at the bases.

Once the valvular lesion has been established, the subsequent course of events is thus its necessary *physiological* consequence; and after this extended explanation, the course of each individual lesion will be readily divined. When we arrive at their discussion, the separate lesions will therefore be only briefly dealt with.

## HYPERTROPHY OF THE HEART

Many causes besides valvular lesions give rise to hypertrophy and dilatation of the heart, which are strictly to be classed with myocardial affections; but they are so constantly associated with valvular lesions, that the two conditions are best considered at this point. *Hypertrophy of the heart* is the natural sequence of increased vascular or cardiac strain, however brought about, *provided the heart muscle itself receives a sufficient blood supply to keep up its nutrition.*

## Etiology—

1. Valvular lesions.
2. Adherent pericardium.
3. Diseases of the lungs.
4. Increased peripheral resistance, the result of atheromatous or arterio-sclerotic changes in the arteries, as seen in Bright's disease, gout, etc.



5. Aneurysm of the aorta.
6. Over-exertion of a healthy heart, as seen in soldiers, hammermen, professional runners, etc.
7. Long-continued functional excitement (tachycardia).

Hypertrophy is a compensatory change, and the heart meets increased work by increase of power. But, whilst it is beneficial *per se*, it must also be regarded as a distinct weakness; for the normal reserve force is used for the ordinary work of the heart, and there is less to meet emergencies. Thus a time comes when the nutrition becomes inadequate, or the reserve is exhausted. Dilatation then overcomes hypertrophy, and compensation fails. The man with a hypertrophied heart may be compared to a country menaced and irritated by an enemy, in which, in order to prevent actual war, the reserve forces should be called out to supplement the standing army, leaving no second line to replace the losses of the actual conflict.

**Hypertrophy of the Left Ventricle** presents the following *physical signs*—

1. Bulging of the præcordia, if the disease has begun in early life; if after complete ossification, bulging is uncommon.
2. Alterations in the apex beat.
  - (1) Its *visible* area is largely increased.
  - (2) It is slow and heaving.
  - (3) The apex beat may be *felt* in the sixth, seventh, or even eighth interspace *outside* the nipple.
  - (4) The area of cardiac dulness is increased downwards and to the left.
  - (5) The first sound at the apex is prolonged and of low pitch.

**Hypertrophy of the Right Ventricle** is usually due to lung disease, mitral obstruction, mitral regurgitation, or a combination of the two. For years the effects of mitral regurgitation *per se* may be counterbalanced by perfect hypertrophy.

*Physical Signs*—

1. Bulging of the lower part of the sternum (occasional).
2. Epigastric pulsation, and forcible impact of the right ventricle in the epigastric region.
3. Moderate increase in the cardiac dulness transversely and towards the *right*.
4. Accentuation of the second sound in the pulmonary area, due to increased tension in the pulmonary artery.



The degree of hypertrophy of which any section of the heart is capable is largely determined by the age of the patient. If one submits to the same course of gymnastic exercises a lad of eighteen and a man of fifty, the increase in volume of the muscles is much more apparent in the former. So with the heart; suppose (*ceteris paribus*) two cases of aortic regurgitation, the one commencing at eighteen, the other at fifty. In the former the hypertrophy of the left ventricle will be very great and long maintained; in the latter the ventricle is no longer capable of great hypertrophy, and relative mitral insufficiency is likely to be sooner established.

## DILATATION OF THE HEART

This condition may be primary or secondary. It is *primary* when it is due to causes directly affecting the myocardium, such as bacterial toxins or chemical poisons, of which alcohol and tobacco are the chief examples, and when it follows emotion, shock, or physical overstrain. *Secondary* dilatation is the result of valvular lesions, adherent pericardium, increased peripheral resistance (arterio-sclerosis, Bright's disease), disease of the coronary arteries, and persistent tachycardia. Fatty and fibroid degenerations of the cardiac muscle predispose to it, and so does inadequate nutrition of the muscle, such as is met with in anæmia. Dilatation resulting from prolonged pyrexia has already been mentioned (p. 4). The causes of secondary dilatation, then, are much the same as those of hypertrophy, and, indeed, in all cases of valvular lesions, a moderate degree of dilatation of the chamber behind the damaged valve precedes and accompanies the hypertrophy. In aortic valvular lesions and in renal disease, the left ventricle is the first to suffer, in mitral lesions and diseases of the lungs, the right.

**Morbid Anatomy.**—The heart is more globular in shape. In pure dilatation the walls of the affected ventricle are much thinned, especially towards the apex. The auriculo-ventricular valves are usually relatively or absolutely incompetent.

### Physical Signs—

#### I. *Dilatation of the Left Ventricle.*

1. Apex beat, when visible, displaced downwards and to the left, feeble and diffuse.
2. Dulness increased downwards and to the left.



3. First sound at the apex short, sharp, and loud ; interval between the sounds either much shortened, or lengthened so that the sounds are equidistant (tic-tac rhythm) ; first sound may be replaced or accompanied by murmur.
4. Pulse rapid and irregular.

## II. *Dilatation of the Right Ventricle.*

1. Diffuse pulsation over præcordia ; apex beat often absent ; right ventricular impulse feeble.
2. Præcordial dulness greatly increased to the right, and to a less degree to the left.
3. First sound in the tricuspid area short and sharp, or replaced by murmur ; second pulmonic sound *weak*.
4. Pulse rapid and irregular.
5. Venous pulsation.

**Symptoms** have been already summarised under Failure of Compensation (p. 261).

## INDIVIDUAL VALVULAR LESIONS

In consequence of endocarditic or degenerative changes, each of the cardiac valves may be affected in one of the three ways, viz. :

1. Its orifice may be narrowed, impeding the flow of blood—obstruction or stenosis.
2. It may be incompetent—regurgitation or insufficiency.
3. It may be both incompetent and obstructed.

These conditions are diagnosed by—

- (1) The position, rhythm, and direction of propagation of the murmurs which accompany them.
- (2) The general symptoms which they produce.

Murmurs are propagated in the direction of the blood-stream, and are best heard in the situations already defined (p. 246), a little way from the valvular orifice where they are produced.

In studying a murmur, attention should always be given to—

1. Its point of maximum intensity.
2. Its rhythm.
3. The direction of propagation.
4. Any modification of the sounds which it induces.
5. The accompaniments (if any).



**Characters of Valvular Murmurs :—**

*Aortic Stenosis.*—A loud, rough murmur heard in the aortic area, systolic in time ; propagated up the sternum into the carotids, and often *accompanied* by a systolic thrill at the base of the heart.

*Aortic Regurgitation.*—A soft, blowing murmur heard in the aortic area, *diastolic* in time ; propagated *down* the sternum and towards the apex, and modifying or replacing the second sound.

In both conditions there are hypertrophy of the left ventricle, greater in regurgitation ; anæmia of the systemic circulation and tendency to syncope, greater in regurgitation ; and very often cardiac pain of an anginoid type, or true angina pectoris.

*Mitral Stenosis.*—A rough, vibratory murmur heard in the mitral area, *pre-systolic* in time, and running up to the first sound, in which it abruptly ends. It is not widely propagated, but is limited to a small area at and internal to the apex beat. The first sound is short and sharp, and the murmur is often accompanied by a marked presystolic thrill (purring tremor). This is the most variable of the valvular murmurs. In later stages of the lesion, when the narrowing of the orifice is considerable, a mid-diastolic murmur, or a murmur occupying the entire diastole, may take its place, and the second sound disappears. When compensation has failed, the murmur may also disappear, nothing being audible at the apex but the slapping first sound.

*Mitral Regurgitation.*—A blowing murmur heard in the mitral area, systolic in time ; propagated to the axilla, *and modifying or replacing* the first sound. If the murmur be loud, it may be heard at the back, close to the left side of the spine.

In both conditions the symptoms, before compensation has failed, are mainly pulmonary—increased liability to bronchial affections, dyspnœa, cyanosis, hæmorrhage, œdema of the lungs. Pain is not usually severe, though there may be a sense of emptiness or cardiac distress. In both conditions, before compensation has failed, there is accentuation and sometimes reduplication of the second pulmonic sound—accentuation because the impediment to the pulmonary circulation is met by more forcible contraction of the right ventricle, and reduplication because the pulmonary engorgement causes the right ventricle to contract more slowly than the left.

*Tricuspid Regurgitation.*—A murmur heard over the fourth right costal cartilage and lower part of the sternum ; propagated to the right and slightly to the left ; systolic in time.

*Pulmonic Stenosis* (the commonest of the congenital lesions).



—A murmur heard in the second left interspace, close to the sternum; systolic in time; propagated upwards. The murmur is often very loud, the right heart is enlarged, and cyanosis is marked.

*Pulmonic Regurgitation* is indicated by a soft blowing diastolic murmur in the second and third left interspaces close to the sternum, and conducted towards the xiphoid cartilage. It is most commonly due to dilatation of the artery produced by the increased pressure following mitral stenosis.

*Tricuspid Stenosis* is rare. The murmur is presystolic, rough, and localised to the lower end of the sternum. Other valvular lesions are often present at the same time.

Too much importance may easily be attached to the mere localisation of murmurs, if *little* notice is taken of the state of the cardiac nutrition, which is indicated *by the character of the sounds*. Post-diastolic and mid-diastolic murmurs are often diagnosed with accuracy by the student, who, at the same time, fails to note whether compensation is established or beginning to fail, whether the sounds are weak, accentuated, or reduplicated. Reduplication of sounds indicates a want of synchronism between the two sides of the heart. Remember, the second sound is produced by the closure and stretching of the aortic and pulmonary valves; the first sound, by closure and stretching of the auriculo-ventricular valves, *plus the contraction of the ventricular muscle*. The loudness and abruptness of the first sound in dilatation is thus an indication of weakening of its muscular element.

**General Symptoms.**—Do not forget that the loudness of a murmur is but a poor index of the gravity of the lesion. A murmur indicates at best only the existence of a particular valvular defect; how long the heart will be able to compensate it depends upon the condition of the myocardium. When compensation has failed, and the circulation through the heart is much embarrassed, the force of the cardiac beat may be insufficient to generate a murmur, the return of which, or the increasing loudness of a murmur previously feeble, may be an actual sign of improvement. Consideration of the general state of the heart and *general symptoms* is of far greater importance than mere attention to any murmurs that exist. For any estimation of the general state of the heart, careful note must be taken of the character of the *sounds*, as has been already indicated, and also of the *rhythm* of the heart (*see Arrhythmia*), and of the degree of exertion which is necessary to induce symptoms. Aortic lesions tend to produce in the first instance



a poorly filled arterial system, while mitral lesions tend to cause venous congestion. The symptoms of the two conditions consequently differ; but remember that the final tendency of all organic valvular lesions is to produce *both arterial emptiness and venous congestion*. The following table will show the more important differences between the symptoms of aortic and those of mitral affections.

## AORTIC DISEASE

Symptoms are mainly due to defective arterial supply, viz.—

1. Pallor.
2. Visible pulsation of the arteries (carotids, radials, &c.).
3. Præcordial pain, often of an anginoid type (malnutrition of the cardiac wall, involvement of coronary arteries).
4. Dyspnœa frequent.
5. Gastric symptoms uncommon.
6. Œdema late and slight.
7. Emboli uncommon.
8. Cerebral symptoms (headache, vertigo, &c.) common, also syncopal attacks from cerebral anæmia.
9. The left ventricle is usually much hypertrophied.

## MITRAL DISEASE

Symptoms are mainly due to venous congestion, viz.—

1. Cyanosis.
2. Pulsation in the veins of the neck in the later stages.
3. Acute pain uncommon; palpitation and cardiac uneasiness frequent.
4. Dyspnœa frequent.
5. Gastric symptoms frequent.
6. Œdema frequent and considerable.
7. Emboli common.
8. Pulmonary symptoms most common, *e.g.* bronchitis, hæmoptysis, infarction, pleural effusion. The bronchitis contributes to the strain upon and consequent dilatation of the right heart.
9. The left auricle and ventricle may be somewhat hypertrophied, but the main hypertrophy is that of the right ventricle.

*Tricuspid Disease.*—This usually takes the form of incompetence of the valve, and as a rule tricuspid regurgitation is due to a relative insufficiency following mitral disease or pulmonary affections, such as emphysema. When primary it is generally a congenital lesion. The general symptoms of failure of the right side of the heart are due to imperfect aëration of blood and congestion of the systemic veins. They have already been summarised under Failure of Compensation. The local signs are epigastric pulsation, replacement of the apex beat by a diffuse and feeble præcordial pulsation due to the dilated right ventricle, permanent irregularity of the cardiac action, great increase of the area of cardiac dulness to the right, systolic murmur in the tricuspid area, and plusation in the veins of the neck. The jugular vein, if obstructed by the finger, *fills up from below during systole*.



**Combinations of Valvular Lesions** are very common. Probably the most frequent is a combination of stenosis and regurgitation affecting the same valve. It is quite rare to meet with aortic stenosis without some degree of aortic regurgitation, and even in mitral stenosis there is usually a regurgitant element, though it may be only slight. Single lesions, proved so *post mortem*, are generally regurgitant. There may be combined lesions of different valves, as aortic regurgitation and mitral stenosis, and so on.

#### THE PULSE IN CARDIAC LESIONS

*Aortic Stenosis* (see Fig. 9).—Here the blood is obstructed in its flow into the aorta. The pulse is thus small, and the sphygmographic line of ascent is slow, and often interrupted (anacrotic).

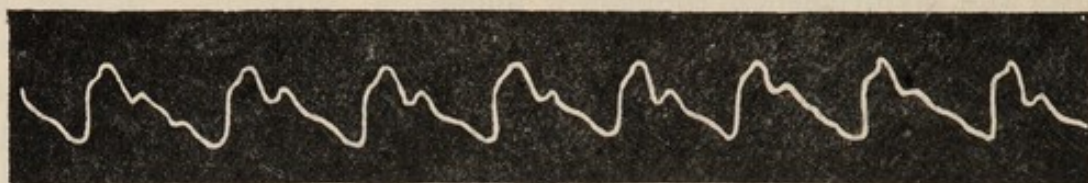


FIG. 9.—Pulse of Aortic Stenosis.

The tension will depend on the amount of obstruction and the degree of hypertrophy. The lesion is seldom solitary, but is usually accompanied by some degree of regurgitation, which modifies the characters of the pulse.

*Aortic Regurgitation* (see Fig. 10).—The ventricle in this condition is filled during diastole by *two* streams—one the re-gur-

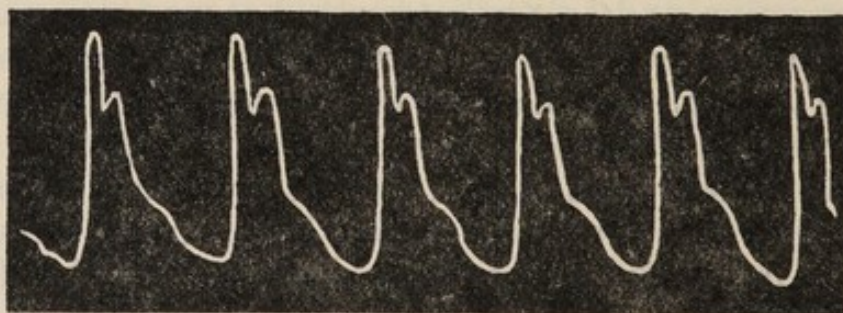


FIG. 10.—Pulse of Aortic Regurgitation.

gitant aortic stream, and the other in the usual direction from the auricle. The chamber is thus filled more quickly, and the pulse made to beat faster. The sudden rise and the sudden fall



of the pulse give it a peculiar kicking or water-hammer character. This is due to the collapsed state of the arteries between pulsations, owing to regurgitation into the ventricle, and their sudden filling with each forcible systole. The throbbing of the carotids often prevents sleep. *Capillary pulsation is often well marked.*



FIG. 11.—Irregular Pulse of Mitral Regurgitation.

In *Mitral Disease* (see Fig. 11) the pulse is often small and of low tension. After failure of compensation has set in, irregularity in force and rhythm is its chief feature, but in mitral stenosis it may be temporarily irregular even before compensation is permanently disturbed.

**Prognosis.**—*Aortic Stenosis.*—Prognosis is comparatively favourable when the lesion originates in the elderly, as its development is slow, and the left ventricle may maintain compensation for a considerable time. In younger patients the outlook is not so satisfactory. When stenosis is the only valvular lesion the vessels are often atheromatous, and their undue fragility may lead to cerebral hæmorrhage.

*Aortic Regurgitation*, on the other hand, is a much more dangerous lesion; sudden syncope through anæmia of the brain is often fatal. Angina pectoris is common in this lesion, and may cause speedy death. It has been recently shown by means of the Wassermann reaction that many cases are due to syphilis, and in these the prognosis is grave on account of the coincident damage to the myocardium. In rheumatic cases the myocardium is not as a rule so severely affected, and compensation lasts longer. The prognosis depends largely upon the extent to which hypertrophy is possible, and therefore upon the age.

Either lesion may go on to dilatation of the left ventricle and implication of the right side of the heart, thus ending like mitral disease, or the exhausted ventricle may cease to beat before the right side is materially affected (death by asystole).

When both lesions exist in the same cases, the stenotic lesion



may be regarded as in a sense protective. Evidently, when the aortic orifice is narrowed, less blood must flow back into the ventricle during diastole, and hence the arteries are not so completely emptied.

*Mitral Regurgitation* is as often due to relative insufficiency as to disease of the valve. In such cases it must be regarded as a symptom, and the prognosis depends upon its cause. Even in cases due to endocarditis the lesion is in many instances compatible with long life if the myocardium is little affected. *Mitral stenosis* is a graver lesion, since it is usually progressive, and tends to an earlier failure of compensation. In women it is especially apt to be fatal during pregnancy and the puerperal period.

*Tricuspid disease* is always grave, since the only compensatory force is the right auricle, of which the capacity for hypertrophy is soon exhausted. The prognosis depends less upon the existence of the murmur than upon the degree of venous stasis in a given case.

In all lesions of the left side of the heart the possibility of embolism must be kept in mind, the most serious being, of course, embolism of the brain, with its resultant hemiplegia.

**Treatment of Valvular Diseases.**—It cannot be too clearly understood that there is no *routine* treatment for valvular diseases of the heart. Although digitalis is to be looked upon as the typical cardiac tonic, and must be used at some stage in the majority of cardiac lesions, yet its indiscriminate use may be productive of much harm, by causing excessive stimulation of the muscle. Similarly, in the same case, much benefit may be obtained at one time from rest, at another from carefully graduated exercise. Certain general indications, however, point towards the selection of a particular line of treatment.

1. Hypertrophy is nature's cure ; and, in order—
2. To get hypertrophy there must be a wholesome supply of blood to the heart tissue.
3. Remember the golden rule—"Avoid putting excessive strain on a diseased tissue."
4. Do not look upon the heart as an isolated structure, but as a *component and essential part* of a complex machine ; and consequently sharing in the anabolic and catabolic changes of the body generally. In other words, if the system generally be lowered, then the heart must also suffer ; if the general tone be raised, the cardiac tone must share in the improvement. But in special circumstances it is also necessary to use those drugs



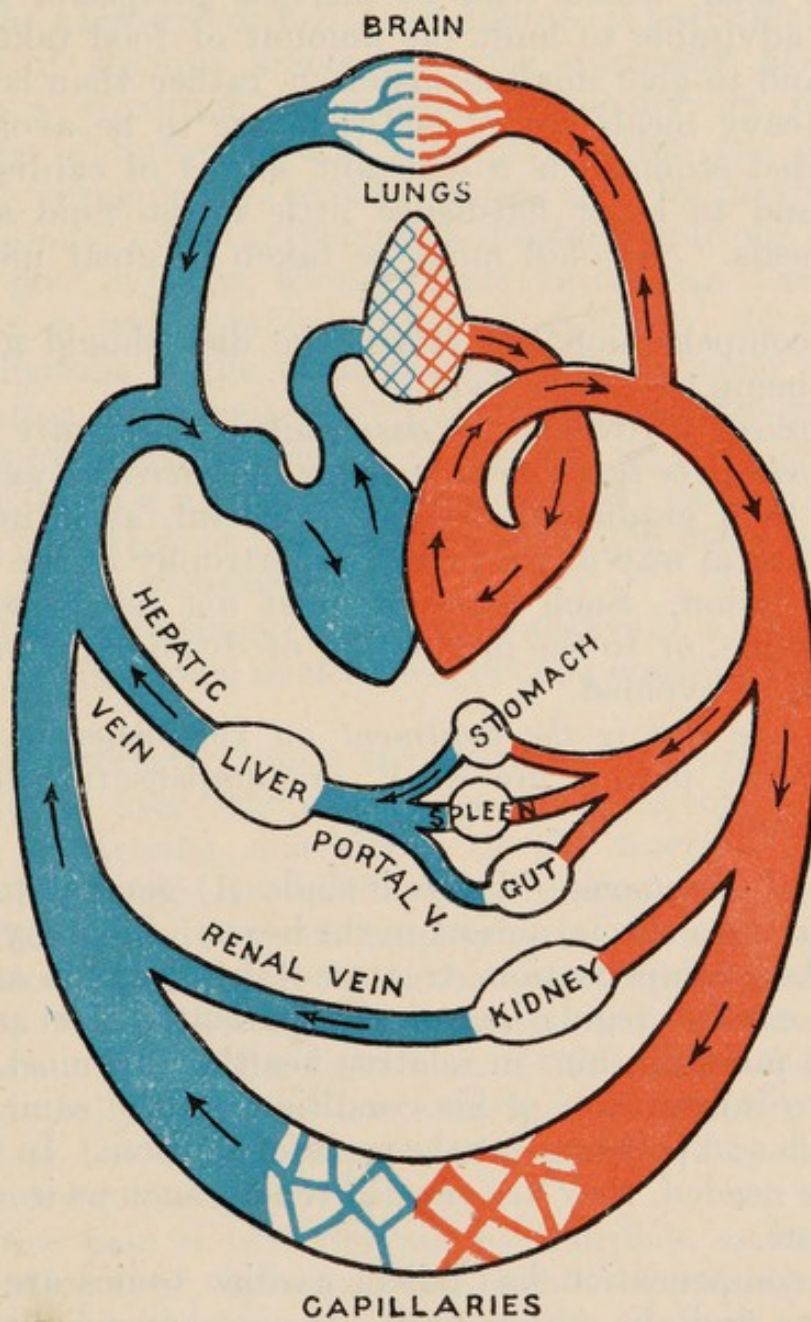


DIAGRAM for studying the results of backward pressure. Note the areas in blue will become the seat of changes consequent on venous congestion.



which have a distinct and particular action upon the heart. Our treatment must be then both general and medicinal.

*General Treatment.*—(1) *Diet.*—Avoid excesses, especially of nitrogenous food, which tend to increase *peripheral resistance*. It is often advisable to limit the amount of food taken at any one meal, and to give small meals often, rather than large meals seldom. Heavy meals before bed-time are to be avoided. An over-distended stomach is a constant source of cardiac embarrassment, and to avert flatulence little or no fluid should be taken at meals. Alcohol must be taken in great moderation, if at all.

Where compensation has failed, the diet should at first be fluid, milk being the staple food.

(2) *Exercise.*—Provided the *assimilative powers are good, and plenty of food can be taken to provide for the increased* expenditure of energy, then graduated exercise is useful, as it imitates or helps the natural way of producing hypertrophy of the chambers behind the lesion. Such exercise must not be pushed to the point of fatigue, or to the production of dyspnoea; and sudden strains must be avoided.

(3) *Absolute rest is the treatment* on the other hand, when the assimilative powers are weak, or compensation once established is failing.

*Medicinal Treatment.*—*Drugs* include (1) general tonics, and (2) those having a special action on the heart. In many instances of established compensation, treatment by drugs is almost unnecessary, careful regulation of the patient's diet and habits sufficing to maintain him in relative health. In most instances it is well to inform him of his condition, at the same time reassuring him and pointing out the need for caution. In this stage, if drugs are needed, they are general tonics, such as iron, arsenic, or strychnine.

Where compensation has failed, cardiac tonics are required, and of these digitalis stands first. It acts beneficially by slowing the action of the heart and prolonging its diastole, thus increasing the natural rest. It also increases the force of the cardiac beat. In addition, it has a diuretic action, and thus tends to relieve dropsy. Its disadvantages are that in toxic doses it produces contraction of the peripheral arteries and so raises the general blood-pressure, and that in aortic regurgitation the prolonged diastole permits of a longer back-flow. Consequently it should not be given in aortic regurgitation unless there are mitral symptoms. Its action is most satisfactory in



cases of mitral regurgitation or obstruction, in which it tends to restore the tone of the dilated and weakened right ventricle. In mitral stenosis the prolongation of diastole counteracts the effect of the obstruction by giving longer time for the blood to pass the narrowed orifice ; and digitalis is particularly indicated in cases of stenosis associated with *auricular fibrillation* (see p. 283), a condition recognisable clinically by the patient's sensations of fluttering and thumping within the chest, by the disorderly irregularity of the pulse, and by disappearance of the *presystolic* murmur, though a diastolic murmur may persist. Do not give digitalis, though there be cardiac disease, if compensation is well established ; and, secondly, do not continue giving digitalis if the urine decreases in quantity after its administration, or if the pulse tends to become more irregular. To avoid its cumulative effect, it should not be given for more than three weeks to a month at a time. It may be resumed after an interval of a week or ten days, during which other cardiac tonics may be used if necessary.

Strychnine is always a powerful adjuvant to digitalis, and is the best cardiac tonic in cases where compensation has not broken down.

Of other cardiac tonics *strophanthus* holds the second place, and may be used where digitalis disagrees, or in combination with it. *Sparteine*, *convallarin* or the tincture of *convallaria majalis*, *casca*, etc., are less frequently employed.

Sudden breakdowns of compensation call for *complete rest* and stimulation of the heart by ammonia or ammonium carbonate, digitalin, or camphor, which is much used in Germany in these conditions. Where the right heart is over-distended, strychnine hypodermically, along with a mercurial purge, and, if necessary, removal of blood by leeches over the liver or by bleeding from the arm, are required.

Cardiac pain is best relieved by morphia, or in cases of the degenerative type, by a course of iodide of potassium. If there be arterial constriction, nitrite of amyl is the best remedy.

Where there is much œdema and congestion of internal organs, diuretics may be used in conjunction with digitalis. Absolute rest in bed and milk diet will also help to relieve the condition. Purgatives are not indicated, although saline laxatives may be useful. The subcutaneous tissues may be drained by acupuncture or by Southey's tubes.

A CAUTION.—If you are called to see a patient who has been picked up insensible and smells strongly of alcohol, do not hastily



conclude he is drunk, but *make sure of the state of the heart*. Many patients, finding themselves getting faint, take a dram of brandy, which, however, may fail to prevent syncope. Rough usage or a cold cell would probably kill a patient under such circumstances.

## DISEASES OF THE MYOCARDIUM

**Acute Affections** of the myocardium are met with, apart from rheumatism, in connection with the specific fevers, most commonly in diphtheria and influenza, in both of which rapid fatty degeneration of the muscular fibres is liable to occur, and may lead to acute dilatation, with such symptoms as enlarged area of dulness, marked irregularity, feeble impulse, and tic-tac rhythm. Death may be sudden. But in most cases the attack is mild, and the chief danger is that of syncope during convalescence. The septic diseases may also cause acute myocardial symptoms.

**Chronic Myocardial Affections.**—Of these, hypertrophy and dilatation have already been discussed. Fatty and fibroid degenerations and syphilitic disease are the remaining conditions which require attention. The *causes* of fatty and fibroid degeneration are (1) previous acute myocarditis; (2) *disease of the coronary arteries*; (3) *alcoholism*, or the prolonged administration of other chemical poisons; (4) *anæmia*; (5) *emphysema*; (6) cardiac overstrain and increased peripheral resistance. Of these, disease of the coronary arteries is the most important. It may be *secondary* to chronic aortitis or atheroma of the aorta; or it may be *primary*, when it takes the form of coronary arteritis, acute or chronic, or, in syphilitic cases, of endarteritis obliterans, from either of which affections thrombosis of a coronary vessel may result. Such thrombosis affecting a large branch causes an anæmic infarct, which may lead to aneurysm or rupture of the heart. *Embolism* of a coronary artery results in sudden death.

**Fatty Heart.**—Any of these coronary affections, or the other causes mentioned above, may lead to fatty heart. The anatomical changes are sometimes only visible under the microscope, which shows that some of the fibres are completely destroyed, while others have lost their transverse striation and are filled with fat droplets. But in many cases the muscle is visibly pale and of a streaky yellowish colour, soft, friable, and greasy to the touch. The right ventricle is usually most affected; and the heart may or may not be dilated. The disease is one of elderly people.



*Symptoms* are often obscure, but there may be coldness of the feet, drowsiness after meals, dyspnœa on exertion, and syncopal or epileptiform attacks. Angina pectoris may also occur. In some instances *sudden death* takes place before any symptoms have declared themselves. The heart may be moderately enlarged, the apex beat feeble or absent, and the sounds short and clear. The pulse is often strikingly slow, and sometimes irregular, especially after exertion.

**Fibroid Degeneration** generally follows gradual obstruction of a large branch of the coronary artery, but it may also be caused by gummatous deposits, and by chronic venous congestion, the result of emphysema. Dyspnœa on exertion, slight cyanosis, puffiness of the ankles, all of gradual onset, are accompanied by enlargement of the heart, and a muffled and prolonged first sound. Death may be sudden or due to gradual cardiac failure.

**Syphilis of the Heart** frequently attacks the aortic valve, giving rise to incompetence, but still more commonly it affects the myocardium and causes either gummata or a diffuse fibrosis. The heart is not usually enlarged, but the pulse is rapid and irregular; anginoid attacks may be present, and also palpitation and dyspnœa. These symptoms in a young adult with no rheumatic history, but with a history or other evidences of syphilis, should excite suspicion.

**Heart-block** is a condition usually due to lesions of the auriculo-ventricular bundle, though it may occur upon stimulation of the vagus or as a result of the action of digitalis. Lesions of the bundle, through which the impulse of contraction is conducted from auricle to ventricle, interfere to a greater or less extent with conduction, and thus give rise to partial or complete heart-block. In partial block the conduction may be merely delayed, the interval between the auricular and ventricular contractions being increased, or some of the impulses may altogether fail to pass the barrier, when the auricle and ventricle beat at different rates. Thus, with an auricle beating at 80, every second impulse may fail to pass, and the ventricular rate will be only 40 ("2 to 1 block"). Other variations, such as 3 to 1 block, may occur; or the block may be sometimes of one ratio, sometimes of another, when the ventricular action will be not only slow but irregular. In complete heart-block the conduction is altogether interrupted, and the ventricle beats independently of the auricle at its own rate, which varies from 24 to 36. Being due to a myocardial lesion, heart-block is produced by those causes which excite myocardial change, *i.e.* the



infective and degenerative processes which have been already mentioned. It is not necessarily incompatible with fair health, but many cases of complete block are associated with a group of symptoms known as the Stokes-Adams syndrome.

**The Stokes-Adams Syndrome** (paroxysmal bradycardia) is characterised by paroxysmal infrequency of the pulse associated with syncopal or epileptiform attacks. It is commonest in males over fifty years old, and is generally associated with lesions of the coronary arteries leading to fibroid degeneration, or with myocardial gummata affecting the a.-v. bundle.

*Symptoms.*—There may be evidence of valvular lesions, especially of the aortic valve; or there may be merely evidence of dilatation and cardiac failure. The arteries are often tortuous and rigid. The patient is usually pallid, but the nose and finger-tips may be cyanosed. Dyspnoea is common, and may amount to cardiac asthma. The pulse is persistently infrequent, but in the paroxysms falls to a very low rate (twenty per minute or less). Examination by means of the polygraph or electrocardiograph shows the independent rhythm of the auricles and ventricles.

The paroxysms vary in frequency and in character. There may be a rapid succession of them, or they may occur only at long intervals. They take the form of *nervous disturbances*, varying from mere giddiness or transient loss of memory to unconsciousness (syncope) or even epileptiform attacks. *Cheyne-Stokes breathing* may be present. Pain is not a constant feature, but there may be anginoid attacks. The seizures are due to cerebral anæmia, produced by arrest of the ventricular contraction.

Such cases end fatally, but life may be prolonged for several years. The end is usually sudden.

**Treatment of Myocardial Diseases.**—Exercise and diet must be regulated according to the individual case, but moderation in eating and drinking must always be enjoined, and the life should be quiet and as free as possible from worry. Iodides are the most reliable medicament in arterio-sclerotic or specific cases, and in the latter mercury may also be of service. The use of salvarsan is risky in myocardial disease. The iodides may be combined with nitrites if there is much arterial spasm. Digitalis and strophanthus must be used with caution, if at all, especially in fatty heart, where any increase of peripheral tension may dangerously overstrain the friable cardiac muscle. Cane-sugar in large quantities (two to four ounces per diem) has recently



been advocated in myocardial diseases for its nutrient effect upon the cardiac muscle, and in many cases it seems to have been beneficial. During the syncopal attacks diffusible stimulants are necessary. If there be marked dyspnœa or Cheyne-Stokes breathing, oxygen, or hypodermic injections of strychnine in large doses, may tide over the crisis.

## ANGINA PECTORIS

("Breast Pang." Stenocardia)

A condition characterised by sudden attacks of severe pain in the cardiac region, with a sense of impending death.

**Etiology.**—It occurs most frequently amongst men above the middle age. Predisposing causes are *all conditions which interfere with the nutrition of the walls of the heart*; such as extensive fatty disease, arterio-sclerosis or atheroma, obstruction of the coronary arteries, lesions of the aortic valve, sometimes adherent pericardium, and also affections of the cardiac ganglia or cardiac plexus. It may be associated with gout, diabetes, or syphilis, and occasionally with influenza. It is sometimes hereditary. The exciting causes are:—

*Sudden strain, an over-distended stomach, powerful emotional disturbances, and chill.* John Hunter said of himself: "My life is in the hands of any rascal who chooses to annoy me." He died in an attack induced by a fit of anger.

**Morbid Anatomy.**—Obstructive lesions of the coronary arteries or of the first part of the aorta are the most constant changes. Fatty or fibroid degeneration of the myocardium is often present, and sometimes tumours, aneurysms, or adherent pericardium lead to involvement of the cardiac nerves. In a few cases no lesion of the heart or vessels has been found.

**Pathology.**—Various theories have been advanced to explain the occurrence of angina. That of *intermittent claudication* is based upon the analogous attacks of pain which sometimes occur in the legs when their arteries are partially obstructed. In such a case the circulation suffices for rest or restful movement, but prolonged exertion causes cramp-like pain and temporary lameness (claudication). Similarly in a heart poorly supplied with blood (ischæmic), or in which the driving power is defective, exertion may cause a temporary exhaustion of the muscle. This condition would be favoured by increased peripheral tension, which is sometimes permanently present, and sometimes exists



during an attack. It may also be produced by chill (contraction of the peripheral arterioles). But in some cases the tension is permanently low, and then we may suppose that a slight increase of work which would be no obstacle to a healthy heart, is sufficient to cause exhaustion of a damaged one. It is in cases of hindrance to the work of the *left ventricle* that angina occurs.

Other theories are that the pain is a neuralgia of the cardiac nerves, or that it is due to tension of the ventricular walls, induced by acute dilatation. Allbutt considers that it is not truly cardiac, but originates in the first part of the aorta. Mackenzie, adopting generally the claudication theory, compares the pain to that produced by the obstructed evacuation of other hollow viscera (*e.g.* biliary colic), and regards it as a protective reflex.

**Symptoms.**—The patient is suddenly seized with an acute pain and sense of constriction across the chest. If he is walking, he stops rigid and motionless; if he is sitting or in bed, he leans forward, fixing the shoulder girdle by grasping any convenient support. The terrible feeling of anxiety is reflected in his expression. The pain is most marked at the lower end of the sternum and generally radiates down the *left* arm, but it may also affect the left side of the neck, and sometimes the right arm. The patient feels as if an iron band were fixed around the chest, and experiences a sensation of impending death. The feeling of suffocation is intense, but the usual cyanosis of dyspnoea is absent, the face often being extremely pale. Respirations are very shallow and difficult, though there is no obstruction to the entrance of air. A sphygmographic tracing of the pulse taken during an attack may show increased arterial tension. An attack may last from a few seconds to many minutes; may kill the first time, or recur at various intervals.

A first attack may be mild. Those that follow increase in severity, and are brought on by slighter causes. In some instances (*angina sine dolore*) there is no pain, although the other symptoms are present; and in others, associated with coldness and pallor of the extremities (*angina pectoris vasomotoria*) the pain is comparatively slight.

*Angina abdominis* is a condition in which sudden and brief attacks of intense pain in the epigastric or umbilical region are brought on by causes similar to those of angina pectoris. Brunton regards it as due to disease of the gastro-intestinal blood-vessels, others consider it as a referred pain of cardiac origin, and the probability is that both explanations are necessary to cover all the cases. The treatment is that of angina pectoris.



**Diagnosis.**—True angina pectoris must be carefully distinguished from neurotic or pseudo-angina. The differences are set forth in the following table (from Huchard).

TRUE ANGINA	PSEUDO-ANGINA
Most common past middle life.	At every age from six years.
Most common in men. Attacks brought on by exertion.	Most common in women. Attacks spontaneous.
Attacks rarely nocturnal or periodical.	Often periodical and nocturnal.
Not associated with other symptoms.	Associated with <i>nervous</i> symptoms.
Agonising pain and sense of constriction.	Pain less severe—distension more than constriction.
Pain of short duration. Attitude—silence, immobility.	Pain lasts one or two hours. Agitation and activity.
Lesions of arterial sclerosis.	Neuralgic affection.
Prognosis grave; often fatal.	Never fatal.

**Treatment.**—During the attack administer nitrite of amyl by inhalation; chloroform may be used as a substitute, or, if nitrite of amyl fails, morphine. Warmth should also be applied to the limbs. During the interval, careful attention to the bowels, diet, and avoidance of severe mental or muscular exercise, and above all of sudden strain, considerably lessen the chance of *another attack*. In the choice of diet all articles of food tending to produce flatulence should be particularly avoided; and if flatulence is present, carminatives such as spirits of chloroform and the compound tincture of cardamoms should be prescribed. When the cause is known, it should be attacked, and the most reliable drug in the treatment of arterio-sclerosis or syphilitic endarteritis is iodide of potassium in doses of gr. x-xx (grm. 0.6-1.3) thrice daily. If the blood-pressure is persistently high, nitroglycerin, given in doses of ℥ j of the 1 per cent. solution thrice daily, and gradually increased if necessary, is often of great benefit. Other nitrites such as sodium nitrite in doses of half a grain to two grains (grm. 0.03-0.12) may be used in its place. In pseudo-angina, the treatment must be directed to the nervous system.

## DISORDERS OF THE CARDIAC RHYTHM

**Arrhythmia.**—It is now the generally accepted view that the action of the heart is due to an inherent property of the cardiac musculature, and that its contractions originate independently of nervous influences in the remains of the primitive cardiac tube. This is the *myogenic theory*, which, in the words of Tawara, as



translated by Brunton, "assumes that the rhythmic activity of the heart in all animals, both in their embryonic and developed condition, resides in the cells of the cardiac muscle themselves, and that the nervous element possesses merely the secondary function of regulation. It also maintains that the transmission of stimuli between the individual parts of the heart does not occur through nerve fibres, as the neurogenic theory assumes it does, but through the block fibres," *i.e.* fibres of the a.v. node and bundle. Whether all the observed facts connected with the transmission of cardiac stimuli can be explained on the myogenic theory, to the complete exclusion of the neurogenic, is a matter on which entire agreement has not yet been reached, and concerning which two schools of thought may still be found; but there is a practical unanimity of opinion that the "block fibres" represent the *customary* route by which such stimuli travel. Normally the contraction begins at the sino-auricular node (remains of the sinus venosus) at the mouth of the superior vena cava, and is conducted thence to the auricle, and from auricle to ventricle by the auriculo-ventricular (a.v.) bundle. But there are other points at which, under an abnormal stimulus, contraction can be originated; and cardiac irregularity may be the result either of stimulation of these points or of abnormal conditions originating in the sinus venosus. The points in question are (1) the a.v. node, in the wall of the right auricle, near the coronary sinus; (2) the a.v. bundle (bundle of His, Gaskell's bridge), on the ventricular side of the node; and (3) the auricular or ventricular tissue. Different types of irregularity result from stimuli originating at these different points.

A. *Sinus arrhythmia* is a condition in which the discharge of impulses begins as usual at the sino-auricular node, but although the strength of the beats is unaffected their rate varies, generally in accordance with the phases of respiration. It is thus a rhythmic irregularity, which may be found either in the healthy hearts of young children and young adults or in convalescence from febrile diseases. It has no unfavourable import, and does not call for treatment.

B. *Premature beats* or *extra-systoles* are due to a premature contraction originating in the auricle or ventricle, or sometimes in the a.v. node, independently of the sinus rhythm. When such a systole takes place, the subsequent normal sinus stimulus occurs during the refractory period, and hence does not cause a beat. Diastole is therefore prolonged until the succeeding sinus stimulus is due. The beat due to an extra-systole is smaller than the normal pulse, and may not be felt at the wrist, thus



causing an apparent infrequency; but it can be made out by auscultation over the heart. In one form of incomplete heart-block, missed beats occasionally occur, and this may be confused with extra-systole; but in the case of heart-block no sound can be heard at the apex. Extra-systole is the commonest form of irregularity, and is often due to functional disturbances—dyspepsia, the abuse of tea or tobacco, and so on—ceasing when the cause is removed. It also occurs in myocardial diseases, but is then associated with other signs of cardiac disease. Occasional intermissions from this cause are not uncommon after middle life, and are often perceptible to the subject. If there is no other indication of cardiac disease, he should be reassured as to their unimportance.

C. *Auricular flutter*.—In this condition the normal beats of the auricle in response to the impulses from the sino-auricular node are replaced by a series of rapid and rhythmical contractions emanating from an abnormal focus in the auricular wall. It is usually associated with chronic organic disease, either myocardial or valvular, and is most common in the middle-aged and elderly. The auricles beat at a rate of between 200 and 350 per minute, but as some degree of heart-block is usually present the ventricles beat at a much lower rate, 100–150, for example. An auricular rate of 280 with a 4 to 1 block may even give a normal ventricular rate of 70. Frequently there is irregularity, although the auricular beat is regular, since the impulses transmitted to the ventricle may occur at variable intervals. Usually the pulse is *persistently* rapid (130–160) for weeks or months, regular, and its rate does not vary when the patient lies down. Treatment is by rest and digitalis in considerable doses (up to a drachm daily). So given, the drug may either produce auricular fibrillation (*v. infra*), which may give place to a normal rhythm when it is withdrawn, or by intensifying the heart-block may cut down the number of stimuli causing ventricular contraction, thus giving the ventricle more ample diastolic rest. Diagnosis is much facilitated by the use of the electrocardiograph.

D. *Auricular fibrillation*.—This condition, into which auricular flutter may merge, is most commonly found in the stage of failure of compensation in rheumatic valvular disease, and particularly mitral stenosis; but it also occurs in the late stages of cardiac failure of the arterio-sclerotic or myocardial type. Clinically it is characterised by perpetual or total irregularity of the pulse, of which no two beats are alike in force or rhythm, by increased gravity of the general condition, and by disappearance of the evidences of auricular systole, *i.e.* in mitral stenosis the



presystolic murmur and thrill disappear, although diastolic murmurs may persist. Exercise augments the irregularity, while it decreases or abolishes that of extra-systolic and sinus arrhythmia. The condition is due to a shower of stimuli emanating irregularly from different foci in the degenerate auricular wall, and causing the replacement of an orderly systole by an irregular and ineffective fibrillary twitching. Once established, it is usually persistent, but the treatment by rest and digitalis (*v. supra*) protects the ventricle by blocking the disorderly impulses, and leads in many cases to a striking improvement. It is indeed in auricular fibrillation that the beneficial effects of digitalis are most manifest.

E. *Irregularities due to failure of conductivity*; Heart-block (see p. 277).

F. *Irregularities due to depression of contractility*: *Pulsus alternans*.—This condition, due to defective contractility of the ventricle, is one in which, while the rhythm remains regular, every alternate beat is weaker and smaller than that which pre-

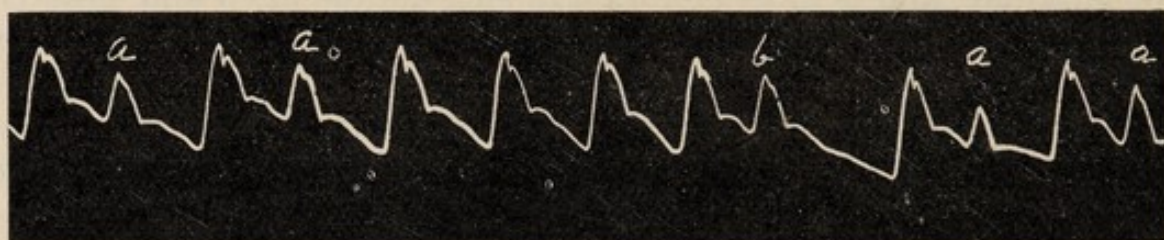


FIG. 12.—Pulsus alternans. At *a a a a*, feeble pulsations preceded by a stronger beat: diastolic intervals equal. At *b*, premature contraction (extra systole) followed by prolonged diastole.

cedes it. The weak beat due to an extra-systole is *premature*, and is followed by a long diastolic pause. Pulsus alternans may be continuous, or may be present only for a few beats at a time. In the former case the condition is grave, and even in the latter it is a serious warning of cardiac failure.

**Tachycardia**, as the term is loosely used, implies merely increased rapidity of the heart's action, and this may be due to such conditions as anæmia, dyspepsia, neurosis, or the abuse of tea, coffee, or tobacco, to exophthalmic goitre, to disease of the medulla oblongata, or to paralysis of the vagus in peripheral neuritis. *Paroxysmal tachycardia*, however is a definite cardiac affection, of which the attacks have an abrupt beginning and ending, are accompanied by restlessness and exhaustion, and last from a few minutes to a few days or even weeks. During the attack the pulse rate may rise to 150 or 200 beats per minute. Violent pulsation is present in the jugular veins during the



paroxysm. Some of the cases end in death from dilatation and cardiac failure. In these cases irregularity is frequent. The condition is due to the temporary replacement of the normal impulses from the sino-auricular node by a sudden rhythmic stimulation of an abnormal focus in the auricle, a.v. bundle, or sometimes in the a.v. node. When the stimulus arises in the node, the so-called *nodal rhythm* (simultaneous contraction of the auricles and ventricles) is established. Paroxysmal tachycardia is commonest in young adults, and often follows an infective process such as rheumatism, scarlet fever, or syphilis. An attack may be excited by exertion, emotion, or flatulence. Little beyond rest in bed can be done in the way of treatment of the paroxysm; in the intervals the habits should be regulated, a quiet life enjoined, and any obvious disturbance of digestion corrected. Digitalis is sometimes of benefit.

**Bradycardia** is a term employed to denote infrequency of the heart. It is met with in a permanent form in cases of heart-block and Stokes-Adams disease, in which it is associated with organic cardiac mischief. Apart from these, infrequency may be normal, or due to organic or functional nervous diseases, to diseases of the digestive system, to uræmia, or to poisoning by digitalis, alcohol, *lead*, or *bile*, as in jaundice. It also occurs in convalescence from acute diseases and after parturition. The *treatment* is that of the cause.

**Palpitation**, though a symptom not specifically cardiac, is apt to arouse in the patient's mind the suspicion that he suffers from heart disease. It is unduly forcible and sometimes irregular cardiac action, *perceived by the subject*. It may indeed occur in organic disease of the heart, but is much more frequently functional and of neurotic origin. Hence it is oftener met with in women than men, and in them is associated with anæmia, uterine affections, worry or emotion, and gastric disorders. Neurasthenia and allied nervous conditions predispose to it. In males it is often due to over-smoking or to alcohol, and sometimes to sexual excess or physical overstrain. The attacks are sometimes paroxysmal, and may come on during rest. On examination, the heart's action is found to be forcible, frequently accelerated, and sometimes irregular, but the organ is otherwise normal. *Treatment* consists in removing the cause, allaying the anxiety, and regulating the diet and habits. Tea, coffee, alcohol, and tobacco should be forbidden; and sedatives, such as bromides, or, in other cases, nervine tonics, such as nuxvomica, may be required.



## II. THE ARTERIES

### ARTERIO-SCLEROSIS

(Arterio-capillary Fibrosis. Atheroma)

A condition of increased rigidity of the arteries, due to a thickening of their walls, beginning in the intima and extending to the media and adventitia.

**Etiology.**—In old age arterio-sclerosis is a physiological condition, the result of wear and tear. To some extent it may be present even in middle life, but the age of onset varies greatly. Hereditary predisposition accounts in many cases for its early appearance, and another important cause is *high arterial tension*, whether this be primary, as Allbutt holds that in a certain group of cases it is, or secondary to other morbid states. Of these the most common are Bright's disease, chronic intoxications (lead, gout), over-eating, and overstrain. Syphilis and alcoholism have been hitherto regarded as very prominent causes; but Ruffer's recent observations on mummies of the eighteenth to twenty-seventh dynasties show that arterial disease was as common 3000 years ago, when syphilis was unknown, as it is to-day, while he also finds it common and of early occurrence in abstaining Mohammedans. Recent work upon cholesterin has shown that rabbits fed with it develop in the intima changes almost identical with those of human arterio-sclerosis, and in the human subject there is a marked increase of cholesterin in the blood-serum both in arterio-sclerosis and in chronic Bright's disease. Such observations would seem to increase the probability that a too richly animal diet may be of importance in the etiology of the disease. It should be added that arterial degeneration may also follow the acute infections.

**Morbid Anatomy.**—The disease may be diffuse or nodose. In the *diffuse* form, besides implicating the arteries, it may extend to the capillaries and veins. The subendothelial connective tissue of the intima is conspicuously thickened, and the media, especially in the smaller arteries, undergoes hyaline or fatty degeneration. Where high tension exists, its muscular coat may be hypertrophied, but otherwise the fibres are atrophied. The capillaries are also thickened. In the *nodose* form the larger arteries are principally affected, and especially the aorta, but the smaller vessels may also suffer. Flat, raised, yellowish-white patches



stud the surface of the intima. They are secondary to degenerative changes in the media and adventitia, which cause a weakening of the wall which the intimal thickening tends to compensate. At a later stage the patches may become infiltrated with lime salts, forming nodules or large plates, according to the size of the artery. They may also soften, forming an atheromatous abscess, and this may rupture through the intima, leaving an atheromatous ulcer; or the calcareous plates may crack, and lacerate the intima. Ultimately the muscular coat atrophies or becomes fibrosed.

**Symptoms** of arterio-sclerosis are a pulse of high tension, high blood-pressure, a hypertrophied left ventricle, with accentuation of the second aortic sound, and thickening of the peripheral arteries. The signs of atheroma are rigidity, visibility, and tortuosity of the peripheral arteries,—radial, brachial, carotid, temporal, etc. The wall of the vessel can be felt after obliteration of the pulse. Irregular thickenings are to be felt in the wall, and the edges of the calcareous plates are often clearly palpable. The pulse is visible.

Atheroma may be far advanced in the great vessels (especially the aortic arch) when there is little sign of it elsewhere.

**Results.**—Cerebral arteries—aneurysm, rupture, or thrombosis; coronary arteries—narrowing, thrombosis, angina pectoris; arteries of the limbs—thrombosis, senile gangrene; arch of the aorta—aneurysmal dilatation or sacculated aneurysm, also extension to the cusps of the valve, with aortic stenosis or regurgitation, or both. The prolonged strain upon the left ventricle due to high blood-pressure may lead in the later stages to dilatation and relative insufficiency, with signs of cardiac failure.

**Treatment.**—A careful, quiet life, plain food, avoiding excess of meat, abstinence, gentle exercise, and regular evacuations form the general treatment. A prolonged course of iodide of potassium is the best medicinal treatment. Periodic administration of a mercurial purge is the most effective means of lowering a high blood-pressure. Nitroglycerin or the nitrites should be used only if it is dangerously high.

## ANEURYSM

Before discussing the essential features of thoracic aneurysm, we must recall a few facts relative to *aneurysms in general*. An aneurysm may be defined as “a localised and persistent dilatation of a blood-vessel.”



Aneurysms may be classified as follows :—

1. Fusiform, or spindle-shaped, where the dilatation of the vessel is more or less uniform. All the arterial coats are present in the wall of the sac.

2. Sacculated—where a limited part of the vessel only is dilated. The inner and middle coats are ruptured, and the sac is formed by the outer coat.

3. Dissecting. The inner coats being ruptured, the blood forces its way between their layers, and breaks back into the vessel lower down.

**Etiology.**—The two main factors are—

1. Damage to the vessel walls.
2. Increased vascular strain.

1. Damage to the vessel wall is due mainly to arterio-sclerosis, especially when of *syphilitic* origin, and occasionally to causes acting from without, as pulmonary tubercle.

2. Increased vascular strain is the result of laborious occupations (hammermen, riveters, soldiers subjected to forced marches with heavy kit, etc.).

It is possible that even a single very violent overstrain may rupture the coats of a previously healthy artery. At least a history of “something giving way” within the chest, as a result of such strain, is not uncommon.

**Morbid Anatomy.**—In a fusiform aneurysm the *inner coat* may be much thickened by atheromatous changes; in a saccular it may be traced for a varying distance into the sac, and is then lost.

*The muscular coat.*—The muscular fibres become stretched and dissociated, and ultimately disappear.

*The outer coat* becomes much fibrosed, and adherent to the surrounding tissues by an inflammatory process; thus the “sac” of an aneurysm is most frequently composed of dense fibrous tissue.

**Course.**—A sacculated aneurysm once started tends to increase, and, if not arrested, finally ruptures. But it may cure itself by becoming so large that the sac, by pressing on the artery, checks or even obliterates the blood supply. Again, when the orifice is small in a sacculated aneurysm the circulation in the sac may be so impeded that thrombi form, producing ultimately a large, pale, laminated clot, which may become



organised, transforming the aneurysm into a fibrous nodule. This occurs, however, only in the smaller aneurysms; but this natural cure gives the physician the cue as to what he should attempt.

**Symptoms.**—The cardinal symptoms of an aneurysm are—  
 (1) Tumour. (2) Pulsation. (3) Systolic murmurs. (4) Pain.  
 (5) Other pressure effects.

The extent of these symptoms depends upon the site of the tumour and the nature of the adjacent structures. In many cases pressure symptoms are but few; in others the diagnosis may rest almost exclusively upon them. Absence of direct physical signs does not necessarily imply the absence of aneurysm.

## ANEURYSM OF THE THORACIC AORTA

The aorta is the most frequent seat of aneurysm in the body, both the fusiform and the saccular varieties being common in this situation. The distinction, however, is of little clinical importance. There are many reasons why aneurysms should be so common in the arch of the aorta—

1. It is much curved.
2. The first part of the arch has very little support.
3. The blood stream ejected during systole of the heart tends to bulge the aorta locally.
4. This part is much more affected by the variation of the cardiac pressure than the distal arteries.
5. Aortitis and atheroma are very common in this situation.

Aortic aneurysm occurs most frequently amongst men who are either prematurely old through intemperance, syphilis, etc.; or in those engaged in occupations which tend to increase the normal aortic strain, such as hammermen, brewers, young soldiers, etc. Of all its possible causes, *syphilis* is the most constantly present, and atheroma is also of very frequent occurrence. Septic emboli lodging in the inner coat account for some cases of small localised aneurysms, but death occurs from the sepsis before these are clinically recognisable. Traumatism may act as a determining cause. The disease is much more frequent in men than women.

**Symptoms** depend on the portion of the arch affected, and on the size and shape of the aneurysm.



Briefly they are—

1. *Symptoms in connection with the Circulation.*—Palpitation, anginoid pains, imperfect filling of the arteries, and sometimes difference in the two radial pulses.

2. *Symptoms due to Pressure*—

(1) *Œsophagus.*—There is difficulty in swallowing, especially solids.

(2) *Respiratory System.*—Less air enters the lung the bronchus of which is pressed upon, and the breath sounds are consequently weak. There is much dyspnœa, attacks of the so-called aneurysmal asthma, or a peculiar alteration of the voice and cough (leopard growl and gander cough). Hæmoptysis may occur later, due to “weeping” of the aneurysm.

(3) *Implication of Nerves.*—The symptoms will depend on the amount of pressure exerted on the nerves. Thus, if *s'ight*, we get symptoms due to *irritation*; if *severe*, symptoms due to *paralysis*. The nerves most likely to be involved are—

	IRRITATION.	PARALYSIS.
<i>Left Recurrent Laryngeal</i>	Alteration of voice; and stridor, due to spasm.	Aphonia. Paralysis of left vocal cord.
<i>Phrenic</i> . . . .	Painful and persistent hiccough.	Intercostal breathing. Death.
<i>Sympathetic</i> . . . .	Dilatation of the pupils. Pallor from constriction of the vessels on the one side. Rapid action of the heart.	Contraction of the pupil and flushing of that side of the face.
<i>Vagus</i> . . . . .	Depressed action of the heart. Vomiting and nausea.	Irregular action of the heart. Pneumonia. Death.

(4) *Veins.*—Œdema of the superior extremities, one side of the head, etc.

(5) *Bones.*—Erosion and absorption; the process being accompanied usually with intense boring pain. When the spine is involved the pain is intense, due to irritation of the intercostal nerves and meninges; there may be much deformity, and even paraplegia through implication of the spinal marrow; and a murmur may be heard over the spine.



- (6) *Thoracic Duct*.—Rapid emaciation, and fatty stools. Pressure on the thoracic duct is very rare.

**Physical Signs.**—*Inspection* may reveal a pulsatile swelling, usually to the right of the sternum; or there may be merely a diffuse heaving impulse above the third right costal cartilage, or visible pulsation in the suprasternal notch. If the descending part of the arch is affected, there may be bulging to the left of the spine.

*Palpation* often detects the expansile character of the pulsation. There may be a *systolic* thrill and *diastolic shock*.

*Percussion*.—The note is dull or flat, and the resistance is increased.

*Auscultation* may reveal, over the dull area, a ringing accentuated second sound and a soft or harsh systolic murmur.

The X-rays may show the presence of a pulsating shadow in the mediastinum.

Difference in the two radial pulses has already been mentioned. The pulse may also be *retarded* in the arteries beyond the aneurysm, and Osler calls attention to a possible obliteration of the pulse in the abdominal aorta or the femorals, without impediment to the circulation, in the case of a large thoracic aneurysm. *Tracheal tugging* may be found when the aneurysm presses downwards upon the trachea or left bronchus. The patient standing or sitting erect with elevated chin, the examining fingers are placed below the cricoid cartilage and draw it upwards, when a downward tug is felt at each systole.

Aneurysm of the *intrapericardial part of the aorta* (sinuses of Valsalva) may present no symptoms or physical signs; but sometimes anginoid pain is complained of, and there may be pressure on the superior cava. Death is usually due to rupture into the pericardium.

The physical signs and pressure symptoms vary according to the part of the arch involved. The following table shows the chief differences :—



PART INVOLVED.	ASCENDING.	TRANSVERSE.	DESCENDING.
<i>Physical Signs.</i>	<p>Pulsation often expansile, in second and third inter-spaces.</p> <p>On palpation, systolic thrill and diastolic shock to right of sternum.</p> <p>Dulness to right of sternum, above cardiac area.</p> <p>Rough systolic murmur, loud clanging second sound. May have diastolic murmur from implication of aortic valve.</p>	<p>Pulsation in episternal notch.</p> <p>Systolic thrill in episternal notch.</p> <p>Dulness over manubrium sterni.</p> <p>Murmur more distinct over manubrium. Diastolic murmur rare.</p>	<p>Pulsation, if any, to left of spine.</p> <p>Absent.</p> <p>No dulness anteriorly, sometimes dull to left of spine.</p> <p>Murmur may be absent; when present systolic, to left of spine.</p>
<i>Parts liable to Pressure and Results of Pressure.</i>	<p>Vena cava superior; dilated superficial veins, œdema of head and neck.</p> <p>Innominate artery; weakness of right radial pulse.</p> <p>Heart; downward displacement of apex.</p> <p>Ribs to right of sternum; pain.</p> <p>Right bronchus; defective respiration on right side.</p> <p>Right recurrent laryngeal (rarely); paralysis of right vocal cord.</p>	<p>Left innominate vein; œdema of left side of head and neck.</p> <p>Any branch of the arch; weakness of right or left radial pulse.</p> <p>Manubrium sterni; pain.</p> <p>Trachea or left bronchus; paroxysmal dyspnoea, altered cough, defective respiration on left side.</p> <p>Left recurrent laryngeal; paralysis of left vocal cord.</p> <p>Sympathetic; dilatation or contraction of pupil, usually left.</p> <p>Œsophagus; dysphagia.</p>	<p>Spinal column and ultimately cord; dorsal pain, afterwards paraplegia.</p> <p>Left bronchus; defective respiration on left side.</p> <p>Left recurrent laryngeal; paralysis of left vocal cord.</p> <p>Left sympathetic (often); dilatation or contraction of left pupil.</p> <p>Œsophagus; dysphagia.</p> <p>Thoracic duct; rapid emaciation, sometimes chylous ascites.</p>
<i>Rupture may occur</i>	<p>Externally.</p> <p>Into pericardium.</p> <p>Into right pleura.</p> <p>Into right bronchus.</p> <p>Into superior cava</p>	<p>Into trachea.</p> <p>Into one or other pleura.</p> <p>Into left innominate.</p>	<p>Into left bronchus.</p> <p>Into left pleura.</p> <p>Into œsophagus.</p>



It will be seen that in aneurysm of the descending part the physical signs may be very few, or even entirely absent, and the diagnosis must then be made from pressure symptoms alone, with the help, when it can be obtained, of an X-ray examination. In some cases symptoms may be latent till death occurs from internal rupture.

*Aneurysm of the descending thoracic aorta* beyond the arch is very often latent. Pain in the back, from erosion of the vertebræ, is the most prominent symptom. There may be dysphagia, and occasionally there is a pulsatile swelling to the left of the spine.

**Treatment.**—*General.*—Everything must be done to quiet the circulation, by the observance of absolute rest and abstinence from all alcoholic drinks, etc. The diet should be nourishing, but limited, and *the quantity of liquid taken reduced to a minimum*. The rigid dietetic treatment of Tufnell is only suitable for early cases, and is often badly borne. After some weeks or months of absolute rest, the patient may be allowed to resume a very carefully guarded life.

*Medicinal.*—Anodynes and sedatives are called for, but iodide of potassium in considerable doses daily is the most efficacious among drugs. It is difficult to say how the iodide acts; it certainly does not lower the blood-pressure to the same extent as many other drugs which are *less* efficacious in benefiting the aneurysmal condition. It has some value in all cases, but is most effective in those which are definitely of syphilitic origin. It should be given in considerable doses, *e.g.*, gr. x–xx (grm. 0·6–1·3) three or four times daily. Salvarsan is contra-indicated.

*Local treatment* is highly unsatisfactory. All methods aim at starting coagulation. Briefly, they are—

1. Introducing needles, and scratching the walls of the sac to form a rough surface, and so bring about coagulation.
2. Introduction of fine steel wire or horse-hair.
3. Electrolysis.
4. Large injections, 6 to 8 oz. (180–240 cc.) of a sterilised two per cent. solution of gelatine in normal salt solution have also been used. The injection is made into the buttock, and repeated weekly till coagulation is induced; but it is by no means free from danger, and several fatal cases have been recorded. It may be more safely introduced by the rectum, and, it is said, with as much effect.



Considering the hopeless character of this affection, morphia may in the last stages be given with some freedom if it is called for by severe pain.

### ANEURYSM OF THE ABDOMINAL AORTA

**Site.**—Most commonly near the coeliac axis. The aneurysm may grow upwards and push the diaphragm before it, or forwards and project anteriorly, or backwards and erode the spine.

**Diagnosis.**—Palpation reveals a definite tumour with the characteristics of aneurysm already described.

Especial care must be taken in diagnosing this condition in pregnant or hysterical females; also in cases of tumours lying *over* the aorta, which are best distinguishable from aneurysm in the knee-elbow position. The expansile character of the pulsation is especially valuable, but neurotic pulsation of the aorta must not be mistaken for aneurysm. forcible and expansile pulsation is present in both, but in neurotic pulsation there is no tumour, and pain is absent, while in aneurysm lumbar pain is not uncommon. The pulsation of aneurysm is of gradual onset, and is persistent; nervous pulsation may arise suddenly, to disappear and recur without obvious cause.

**Treatment.**—As in thoracic aneurysm. Compression under chloroform may be tried, though it is decidedly risky.

**Prognosis.**—Bad. Death may take place from—

1. Compression paraplegia.
2. Embolism of the superior mesenteric artery.
3. Complete obliteration of the lumen by clots.
4. Rupture (oftenest into the duodenum).



# DISEASES OF THE BLOOD AND DUCTLESS GLANDS

## I. THE BLOOD

### EXAMINATION OF THE BLOOD

IN cases where anæmia or other altered conditions of the blood may be suspected, the precise diagnosis rests upon an examination of the blood itself. In such an examination several points must be considered. They are as follows :—

1. Estimation of the number of corpuscles, red and white.
2. Estimation of the percentage of hæmoglobin.
3. Observation of the changes in form, size, or staining reaction of the red corpuscles.
4. In cases where the leucocytes are increased in number, determination of the relative proportion of the different *kinds* of leucocyte.
5. Presence of micro-organisms or other parasites in the blood (less important from the point of view of blood diseases, but often confirmatory of the diagnosis of acute infections, such as anthrax, enteric, tubercle, etc.).
6. Presence of other abnormal constituents, as pigment (*melanæmia*) free in the blood plasma (severe malarial cachexia, melanotic sarcoma, etc.), or fat (*lipæmia*) in cases of diabetes mellitus.

1 and 2. The number of corpuscles and the percentage of hæmoglobin may be estimated, for the first, by the hæmocytometer of Gower or Thoma-Zeiss, and for the second by the hæmoglobinometer of Gower or Oliver. The normal number of red corpuscles per cubic millimetre of blood is in the male 5,000,000, in the female, 4,500,000. The average number of leucocytes is from 6000 to 8000 per cmm. For details of the estimation, consult a recent manual of clinical methods.

The blood should be taken from the finger-tip, or the lobe of the ear, after careful cleansing of the part. The finger or ear should not be compressed.



It will be found upon examination that the various anæmias differ in the relative proportion of hæmoglobin and red blood corpuscles. In secondary anæmia, for example (following phthisis, cancer, syphilis, etc.), these two constituents may be diminished in much the same proportion, or the anæmia may be of the chlorotic type. In chlorosis the red cells are slightly defective, the hæmoglobin much more so. In pernicious anæmia there is an enormous destruction of red corpuscles, while the percentage of hæmoglobin is usually somewhat higher.

The proportion between the percentage of hæmoglobin and the percentage of corpuscles (*i.e.* the figure obtained by dividing the former by the latter), constitutes the *colour index*. In normal blood the resulting quotient is 1.0, in chlorosis it is less, and in pernicious anæmia greater than 1.0. In secondary anæmias it is 1.0 or somewhat less.

3. The red corpuscles may be altered in shape, size, or staining reaction. Alterations in shape (*poikilocytosis*) are common to all forms of anæmia, but most profound in the graver forms. Alterations in size may result in the presence of corpuscles smaller than normal (*microcytosis*), or larger (*macrocytosis*). The more frequent are such alterations, the more likely is the condition to be that of pernicious anæmia. And, especially though not exclusively in pernicious anæmia, one may find *nucleated* red corpuscles, either of ordinary size (*normoblasts*), or much larger than the normal (*megaloblasts*). Microblasts and poikiloblasts have less diagnostic importance.

In many of the anæmias some of the red corpuscles do not stain pink, like normal corpuscles, with hæmatoxylin and eosin, but take on a greyish-purple tint, *i.e.* they react to basic as well as acid stains, and are coloured by both constituents (*polychromatophilia*).

4. Increase in the number of white corpuscles (*leucocytosis*) may be—

- (1) Physiological, as after meals.
- (2) The accompaniment of inflammatory diseases and specific fevers; *but not enteric*, unless complications have arisen.
- (3) Leucocytosis is an essential feature of leukæmia, and it is possible to determine the form of leukæmia which is present by observation of the kind of leucocyte principally increased. In lymphatic leukæmia the lymphocytes are present in enormous numbers, in myelogenous or splenomedullary leukæmia the polymorphonuclear and eosinophil leucocytes.



Decrease in the number of leucocytes (*leucopenia*) is less frequent than leucocytosis, but is met with in pernicious anæmia, malaria, enteric, tuberculosis, and other conditions.

There are several varieties of leucocyte distinguishable by their staining reactions and the characters of their nuclei. They are as follows :—

- (a) Lymphocytes : small non-granular cells with a round nucleus—22-25 %.
- (b) Large mononuclear cells (large lymphocytes). The nucleus is oval, and the protoplasm non-granular—1-2 %.
- (c) Transitional cells : like the last, but with a notched or indented nucleus—1 %.
- (d) Polymorphonuclear cells : nucleus convoluted or lobed, fine neutrophil granulation—70 %.
- (e) Eosinophil cells : rounded or branched nucleus, coarse oxyphil granulation—2-4 %.
- (f) “Mast” cells : lobulated nucleus, basophil granulation— $\frac{1}{2}$  %.

Besides these cells, all of which are present in normal blood, bone-marrow cells (*myelocytes*) may be found in disease. These are large cells, with a single, large, faintly-staining nucleus. Some of them have a neutrophil, others an eosinophil granulation.

The alterations referred to under 5 and 6 do not require separate discussion here.

## ANÆMIA

In the strict sense of the word, anæmia means want of blood, but the term is employed with a much wider significance, and includes—

1. Oligæmia—deficiency of blood.
2. Hydræmia—thinness or wateriness of the blood.
3. Oligocythæmia—a diminution of the number of corpuscles.
4. Poikilocythæmia—irregularity in the shape of the corpuscles.
5. Oligochromæmia—deficiency of blood-colouring matter.

**Classification.**—Anæmias are classed as (1) *secondary* or symptomatic, and (2) *primary* or essential. Of the latter there are two varieties, (1) chlorosis, and (2) pernicious anæmia.



**General Pathology.**—It will save much recapitulation if we start with a clear idea of the general effects of anæmia upon the body. In consequence of imperfect oxygenation and deficient nutrition, there follow impairment of function, shrinkage of the tissues, softening of the blood-vessels, and, in long-standing cases, fatty degeneration of various organs. Thus begins a vicious cycle of retrograde changes, each new failure on the part of an organ increasing the original defect. In the early stages excitability of the nervous system is shown by emotional manifestations; or there may be diminished sensibility, as seen in the dilated pupils, which show the depressed condition of the retina. Œdema is common in unsupported tissues. Owing to muscular weakness dilatation of the heart is not infrequent. Hæmorrhages from any of the mucous surfaces, and especially retinal hæmorrhages, are frequent in the graver anæmias.

## SECONDARY OR SYMPTOMATIC ANÆMIA

**Causes.**—*Deficiency of food*, either through actual want, or from inability to take food, as in cancer of the œsophagus.

*Excessive discharges*—*i.e.* pyæmic abscesses, diarrhœa, albuminuria.

*Cachectic and toxic conditions*—phthisis, cancer, syphilis; poisoning by *lead*, mercury, or arsenic.

*Hæmorrhages.*—After a severe hæmorrhage the deficiency of red corpuscles and hæmoglobin is only slowly replaced, though the fluid part of the blood is soon restored. The red corpuscles show some poikilocytosis, and normoblasts may be found. There is also a moderate leucocytosis.

*Certain parasitic affections*—ankylostomiasis, bothriocephalus anæmia. In this class the symptoms may closely resemble those of pernicious anæmia.

In these cases, with the exception of those last mentioned, the anæmia is of the chlorotic type, *i.e.* the hæmoglobin is somewhat more deficient than the corpuscles, and the colour index is less than unity. Removal of the cause, when that is possible, and placing the patient under favourable hygienic conditions, effect a cure.

## CHLOROSIS

A peculiar anæmia, attended with a greenish, transparent, wax-like condition of the skin, common in females between the ages of fifteen and twenty-one, and characterised by a deficiency of hæmoglobin.



**Etiology.**—The cause of chlorosis is unknown, though there are many theoretical explanations, of which the following are the chief:—

1. The disease is due to constipation and toxic absorption from the bowel (Sir Andrew Clark).

2. The demands of puberty reveal a congenital narrowness of the arterial system, which prevents a proper supply of blood to the various tissues (Virchow).

3. The organic iron compounds of the food are broken up by excessive decomposition in the alimentary canal, and the iron thus liberated cannot be absorbed into the blood (Bunge).

4. Chlorosis is due to excessive loss of blood at the period of puberty (menorrhagia), which leads to dyspeptic conditions, anorexia, and defective absorption of iron (Stockman).

5. The disease is due not to an absolute diminution of hæmoglobin, but to an excess of plasma (Haldane and Lorrain Smith).

None of these theories covers all the cases. Constipated subjects are not always chlorotic, there may be no congenital arterial defect or excessive loss of blood, and reduction of the plasma does not cure without administration of iron. At present we can only assume that in women the adolescent period causes an increased demand upon the blood-forming organs, which in some cases they are unable to meet. Predisposing causes are defective nourishment and want of fresh air and exercise. The disease is common in girls coming from the country to domestic service or to factory life ; but it is met with in all classes.

**Symptoms.**—Languor, dyspnœa, palpitation on exertion, headache, noises in the ears, and dyspeptic symptoms, as in symptomatic anæmia. The greenish waxy pallor of the complexion is conspicuous, but may be masked by ruddiness of the cheeks. The mucous membranes are pale. The more characteristic symptoms in addition are plumpness ; systolic murmurs at the base of the heart ; “ *bruit de diable* ” at the lower end of the jugular vein ; constipation and irregular, profuse, or scanty menstruation. Mitral regurgitation is sometimes present,—the result of relative insufficiency of the valve, following upon dilatation. Thrombosis of the femoral vein, or even of the cerebral sinuses, is an occasional complication, and gastric ulcer is not uncommon.

*The blood* is pale, and the corpuscles are poorly coloured. Poikilocytosis is sometimes present, and normoblasts may be



found in severe cases. The number of red corpuscles is not greatly diminished (60-80 per cent.), but the percentage of hæmoglobin is much below normal (30-50 per cent.). The colour index is thus fractional. Leucocytes are not increased.

**Treatment** is highly satisfactory. Fresh air, gentle exercise, nitrogenous food, laxatives, and iron will cure the worst cases. Rest in bed is necessary at the outset.

An excellent combination is iron, aloes, and carbonate of potassium ; but Bland's pill, especially in the bipalatinoid form, or reduced iron, gr. ij-ijj (grm. 0·12-0·2) thrice daily after food, is the most generally useful. If there be dyspepsia, ferric salts should never be given, but the non-irritating ferrous salts should be used instead. Iron should always be given after meals. The organic preparations have little advantage over the inorganic. The iron is sometimes advantageously combined with arsenic. Manganese, often recommended, is useless. The iron acts in two ways :—

1. It combines, according to Bunge, with the intestinal sulphides, and thus prevents the decomposition of the organic iron of the food, which is therefore absorbed.

2. A small amount of the iron itself may be absorbed and assimilated.

A daily evacuation must be secured by means of aloes, cascara sagrada, or saline laxatives. Excess of carbohydrates as a rule is harmful, but in the presence of gastric ulcer the diet must be suited to the complication. Chlorotic girls should be released from all studies, and exciting literature should be kept from them.

In accordance with Lorrain Smith's view of the etiology, eliminative treatment by means of diuretics, diaphoretics, and laxatives has been tried, but has little success without the addition of iron.

## PERNICIOUS ANÆMIA

A progressive and profound anæmia, developing without any evident cause, characterised by great diminution in the number of the red corpuscles, and terminating fatally.

**Etiology.**—The disease affects males somewhat oftener than females, and is rare under twenty-five years of age. Some cases have originated during pregnancy or after child-birth. The disease, according to William Hunter, is a chronic febrile



infection, due to auto-intoxication from the gastro-intestinal tract, and produced by an organism the toxins of which give rise to hæmolysis. It must, however, be remembered that cases presenting all the clinical appearances have been found, *post mortem*, to be due to cancer of the stomach or bones, ankylostomiasis, etc. Gulland considers the essence of the disease to be a toxic affection of the bone-marrow, a mere hæmolysis being insufficient to produce the various phenomena of the disease unless the bone-marrow has undergone megaloblastic degeneration. The source of the toxin, he holds, is as yet undetermined.

**Morbid Anatomy.**—*The blood* shows a remarkable diminution of red corpuscles; they may sink to less than half a million per cubic millimetre. The total amount of hæmoglobin is also diminished, but the amount in each corpuscle, far from being diminished, is often relatively increased, so that the colour index is above unity. The corpuscles exhibit a number of various shapes, some are tailed, others crenated; often the hæmoglobin can be seen protruding prior to its escape from the stroma; some discs are extremely minute, and may be nucleated. Microcytes, macrocytes, normoblasts, and megaloblasts, may all be found. Polychromatophilia is frequent. There is no leucocytosis. The blood when shed is pale, and coagulates with difficulty. Some degree of leucopenia is usually present.

*The liver* is rich in iron, the pigment (hæmosiderin) occupying the outer and middle zones of the lobules; and the organ is generally fatty, and may be enlarged. *The spleen* and *kidney* also show excess of iron pigment; the spleen is often congested, and the kidney may be the seat of a toxic nephritis.

*The heart* is the seat of advanced fatty degeneration, which may extend to the large vessels. Hæmorrhages are common.

*The bone-marrow* is red, and contains large numbers of megaloblasts and normoblasts, while there is also an increase in myelocytes and lymphocytes; but in a few cases (*aplastic anæmia*) it shows no such reaction, but rather atrophy.

The presence of large quantities of hæmosiderin in the liver, and of pathological urobilin in the urine, are indications of the hæmolysis which is so marked a feature of the disease. Conditions of oral or gastric sepsis are frequently, though not constantly, associated with it, and, in such conditions, Hunter holds that there exists a permanent nidus for the manufacture of the microbic poison.



**Symptoms.**—The early symptoms are : “ (1) glossitic, gastric, and intestinal ; accompanied by (2) hæmolytic symptoms—lemon colour, biliousness, with or without jaundice ; (3) febrile and nervous disturbances ; (4) a remarkable degree of anæmia, out of all proportion to the severity of any symptoms present ” (Hunter). The onset is extremely insidious, but as the case progresses these symptoms recur with greater severity, but with periodic remissions at intervals of three or four weeks. In acute cases relapses are frequent and remissions brief ; in chronic cases there may be long periods of apparent improvement both in the condition of the blood and the general symptoms. Muscular weakness is marked ; vomiting is common, and attacks of moderate fever ( $100^{\circ}$ – $103^{\circ}$  F.) of a remittent type may last for a few weeks at a time, disappearing and recurring without apparent cause. Besides its lemon-yellow tint, the skin often has a peculiar velvety feeling to the touch. Hæmorrhages are frequent, especially retinal hæmorrhages and hæmoglobinuria or hæmaturia. Epistaxis or melæna may also occur. In some cases degenerative lesions of the posterior and lateral columns of the cord are present, and cause symptoms of ataxic or spastic paraplegia.

*Blood crises*, in which large numbers of nucleated red cells appear in the blood, and persist for a few days, are occasionally met with.

There may be some muscular wasting, but it is not considerable, and the subcutaneous fat is often increased ; hence wasting is not apparent, and the patient may present a fairly well-nourished appearance.

**Prognosis** is highly unfavourable, most cases dying in from six to fifteen months, though chronic cases may survive for several years.

**Treatment.**—Tonics of all kinds may be used to meet the symptoms of the individual case. Prolonged rest in bed and nourishing food are essential. The only hæmatinic of any value is *arsenic*, which must be given persistently, and gradually pushed to large doses. As much as 10 to 15 minims of Fowler’s solution may be given thrice daily in some cases. The drug is pushed till the limit of tolerance is reached, the dose being then reduced to two-thirds of the amount taken when arsenical symptoms appeared. Salvarsan has been tried with disappointing results ; nor has splenectomy proved itself of material value. Septic conditions of the mouth must be rigorously treated, and all carious teeth removed. For gastric antiseptics, after lavage



of the stomach, salicylic acid should be administered, and for intestinal antiseptis, small doses of mercurial preparations.

In cases, where, on Hunter's theory, the infection is "firmly rooted in the mucosa," the poison may be attacked in the blood. For this purpose Hunter uses injections of antistreptococcic serum, beginning with small doses (5 cc.) repeated after a few days' interval. By these means he has been able to prolong life and to maintain health for over seven years. Treatment must be resumed on the earliest appearance of a relapse.

### LEUKÆMIA (LEUCOCYTHÆMIA)

An affection characterised by persistent increase of white corpuscles, hæmorrhages, and changes in the marrow, spleen, and lymphatic glands.

There are two types of the disease, myelogenous or spleno-medullary leukæmia (myelocythæmia), in which the changes principally affect the spleen and bone-marrow, and lymphatic leukæmia, in which they fall upon the lymphatic glands.

**Etiology.**—The disease may develop at any age, but by far the greater number of cases occurs in adult life. Men are more often attacked than women. The lymphatic type is more common in the young. Leukæmia is sometimes hereditary, and it has been observed to follow injuries, malaria, and pregnancy, but its origin is entirely unknown. Its essential feature is a hyperplasia of the leucocyte-forming tissues, analogous, it has been suggested, to lymphosarcoma.

**Morbid Anatomy.**—*The blood* shows very characteristic changes. (1) In *myelogenous leukæmia* the leucocytes are enormously increased in number (300,000 or more per cmm.), and their proportion to the red cells may be as high as 1 to 6, 1 to 3, or even 1 to 1, instead of 1 to 500 or 600 as in normal blood. There is a great increase of eosinophil cells, which may also be relatively increased; while the polymorphonuclear neutrophils show a total, but not a relative, increase. "Mast" cells are present in considerable numbers; but the most characteristic feature is the presence of large numbers of myelocytes, which are not normally present in the blood. The red cells are considerably diminished, and also the hæmoglobin. Normoblasts and even occasional megaloblasts are present. (2) In *lymphatic leukæmia* only the lymphocytes are increased, and they may constitute more than ninety per cent. of the leucocytes. The type of lymphocyte varies; in some cases the large lym-



phocyte (large mononuclear cell) is predominant, and others the small lymphocyte. The total increase of leucocytes is less than in splenomedullary leukæmia, and there are rarely as many as 1 to 10 red cells. Myelocytes are absent, and nucleated red corpuscles are few.

In advanced leukæmia the blood is whitish or milky, sticky, and slow to coagulate, the clot being soft and jelly like. Colourless, elongated, octahedral crystals (Charcot-Leyden crystals) may be found in it after death.

*The spleen* is usually firm and much enlarged. In the splenomedullary form it may weigh as much as 15 or even 18 lbs., but in the lymphatic form the enlargement is less notable, and sometimes is quite slight. The organ is often bound by adhesions to the abdominal wall, the diaphragm or the stomach. The capsule is much thickened. On section, the spleen presents a pinkish appearance, appears fatty, and feels greasy to the touch. The pulp is dark, and the Malpighian tufts are usually inconspicuous. The sinuses are often distended with leucocytes. Hæmorrhagic infarctions, old and recent, are very common.

*The lymphatic glands* may be somewhat enlarged, even in the splenomedullary form, from infiltration of leucocytes. In the lymphatic form all the lymphatic glands are much enlarged and crowded with leucocytes, but they remain soft and movable.

*The marrow*.—The marrow may be dark brown in colour, or present a peculiar yellowish-red appearance. In the splenomedullary form large numbers of nucleated red corpuscles, polynuclear leucocytes, and myelocytes are present; and in the lymphatic form lymphocytes. The hyperplasia of the marrow may cause atrophy and tenderness of the bones.

The *liver* is often somewhat enlarged, and it is infiltrated with masses of leucocytes extending along the portal tracts. Its capillaries may also contain numbers of leucocytes, and there is often fatty change in the hepatic cells. The *kidneys* show similar leucocytic infiltration.

**Symptoms.**—The onset is usually insidious, although in acute lymphatic leukæmia the symptoms may be severe from the outset. The patient generally applies for medical advice on account of breathlessness, palpitation, dyspepsia, or other evidences of anæmia. Hæmorrhages of various kinds, among which are epistaxis and hæmatemesis, may be the first symptoms. When the disease has advanced, the condition of the patient is char-



acteristic. The abdomen is prominent; the countenance of a deadly white, or with a slightly sallow tint; the sclerotic of a pearly lustre, the pupils dilated, and the mucous membranes blanched. The pulse is soft and compressible, but may be full. Hæmorrhages are common under the skin, in mucous membranes, into serous sacs, and into the retina. Retinal changes are often marked, the veins being tortuous, broad, and pale, and retinitis is sometimes present. The enlargement of the spleen, or of the accessible lymphatic glands, is a striking feature of the disease. Towards the end, grave changes set in, and intermittent diarrhœa, *attacks of fever*, and œdema or general dropsy usher in a fatal issue. The urine is usually of high specific gravity, containing excess of uric acid and urea. The pallor, usually so well marked, is sometimes wanting. The special points to note are the condition of the blood and of the spleen or lymphatic glands, the marked exhaustion, the pyrexial attacks, and the tendency to hæmorrhages, and to dropsy associated with highly coloured urine.

*Characters of the enlarged spleen.*—It enlarges in the axis of the tenth rib, and therefore tends to go forward towards the right iliac fossa. If much enlarged, it is found to be present as a firm, hard tumour, having a distinct notch in its anterior border; it may reach from the flank down to or below the umbilicus. Note in contrast to enlargements of the kidney that the colon never lies in front of an enlarged spleen.

**Prognosis** is always bad, death occurring in from one to three years. *Acute leukæmia*, which is usually of the lymphatic type, may end fatally within two months.

**Treatment.**—Rest, fresh air, and a nutritious diet are of importance, and the general hygiene must be as perfect as possible. Improvement has been recorded under the use of the X-rays. Arsenic is the only drug on which reliance can be placed, and it should be used in large doses, as in pernicious anæmia. Benzol has been administered in capsules containing 5 minims of the drug along with 5 minims of olive oil. One is given thrice daily, and the dose may be gradually increased to 20 minims. It has a considerable affect in reducing the number of leucocytes, but the improvement is only temporary.

## LYMPHADENOMA (HODGKIN'S DISEASE)

An affection characterised by progressive enlargement of the lymphatic glands, destruction of red corpuscles, and secondary lymphoid growths in the various organs.



**Etiology.**—Hodgkin's disease may occur at any age, but is most common in children and young adults, and in the male sex. The etiology is unknown, though possibly syphilis and tubercle tend to predispose to the disease. It has been looked upon as an infection, bearing a doubtful relation to tuberculous adenitis on the one hand, and to leukæmia and lymphosarcoma on the other.

**Morbid Anatomy.**—The affected glands are usually much enlarged, single, painless, and non-adherent to the skin. Though they may feel to the touch hard and solid, they are far more frequently somewhat soft and elastic. *Their two great characteristics are—they tend neither to suppurate nor to caseate.* They may under certain circumstances suppurate and mass together; but such results are to be regarded as accidental. Histologically, the chief change in the glands is a general hyperplasia, with thickening and increased firmness of the capsule, proliferation of the connective tissue and of the endothelial cells, from some of which large multinuclear giant-cells (lymphadenoma cells) are developed, and formation of lymphoid and also eosinophil cells. The amount of new fibrous tissue formed governs the degree of hardness; if the lymphoid cells are abundant, the gland is soft; but when the fibrous tissue element is in excess, the glands are hard.

*The spleen* is generally enlarged, but not nearly to the same extent as in leukæmia. In consistence it is firm, and purple patches from venous congestion often appear on the surface. On section can be seen golden-yellowish masses, consisting of lymphoid cells and pigment enclosed in a fibrous reticulum. The Malpighian bodies appear as translucent yellowish suet-like masses. The general fibrous stroma is much increased, as in the lymphatic glands.

Lymphoid tissue is sometimes found in the bone-marrow, and there may be lymphoid nodules in the liver.

**Symptoms.**—1. Enlargement of the lymphatic glands.

2. Anæmia of a secondary type, with considerable decrease of red corpuscles, which may fall to 2,000,000 or less, and of hæmoglobin, and a normal or *slightly* increased number of leucocytes.

3. Symptoms pointing to the virulent nature of the disease—*i.e.* marked emaciation, cachexia, secondary deposits of lymphoid tissue.

4. Attacks of pyrexia.

5. Symptoms due to pressure from the lymphoid growths.



The glandular enlargement most frequently begins in the anterior chain of glands at the posterior border of the sterno-mastoid, and may long remain unilateral; then the axillary and inguinal groups, and finally all the glands of the body may show extensive changes. Often both sides of the neck become involved, and the growths meeting anteriorly in the middle line may compress the trachea to a dangerous extent. Osler points out that when the abdominal glands are affected, the sympathetic system is often profoundly disturbed, and pigmentation of the skin may occur. The enlarged mediastinal glands may cause severe dyspnœa, inequality of the pupils, pleurisy, etc. Very varied symptoms may result from the pressure of the enlarged glands upon nerves or veins. *The periodical elevations of temperature may be so marked as to simulate ague*, but in other cases the fever may be continuous or irregular. Lastly, note that in some cases anæmia is not prominent until marked cachexia has set in.

**Treatment.**—In a disease which appears to begin locally, excision of the set of glands first affected has naturally been practised, but it is doubtful if operation has ever retarded the course of the affection. Where a group of glands produces dangerous pressure, operation may be necessary for its relief. We may hope for benefit from the persistent use of arsenic in large doses, along with attention to the general health. Salvarsan often produces striking temporary benefit. The X-rays may be tried; but the disease is usually fatal in from two to four years.

## STATUS LYMPHATICUS (LYMPHATISM)

An unusual condition, occurring chiefly in children, in which there is hyperplasia of the lymphatic glands and lymphoid tissues throughout the body.

The internal lymphatic glands are more enlarged than the superficial, which often are of normal size. The thymus is much enlarged and soft, and there is moderate enlargement of the spleen. The bone-marrow is hyperplastic, and in the shafts of long bones it is red instead of yellow. The thyroid is often enlarged. The heart and aorta are poorly developed, and there may be evidence of rickets.

The subjects of the condition are often fat and flabby, with a pasty complexion. The tonsils are enlarged, and the superficial lymphatic glands may also be slightly affected. Dulness



over the manubrium indicates the position of the enlarged thymus, and the spleen may be palpable. The left ventricle is sometimes dilated, and the blood-pressure is low. Anæmia of the chlorotic type is present.

Sudden death from syncope is liable to occur from trifling causes, small operations, administration of anæsthetics, etc., or even without apparent cause. The reason is not well understood, but toxæmia from the secretion of the enlarged thymus, and also pressure on the trachea from the same cause, have been suggested.

Individuals suspected of this condition should not be operated upon unless for urgent reasons.

## SCURVY

A constitutional affection characterised by great debility, a spongy condition of the gums, a tendency to hæmorrhages, and anæmia.

**Etiology.**—The disease is much less common now than in former times. It is usually associated with improper and insufficient food, unhealthy hygienic surroundings, etc. It was formerly very common in sailors, and is still endemic in parts of Russia.

There are three theories as to the causation of scurvy.

1. That it depends on the presence of a specific organism. No such organism has yet been found.

2. That it is due to a defect of certain elements in the food, usually present in fresh vegetables. Defect of the potassium salts, according to Garrod, and defect of the malates, citrates, and tartrates, according to Ralfe—salts which become converted into carbonates in the blood, and tend to keep it alkaline—both these are looked upon as causal factors. Wright regards scurvy as an acid intoxication. It has been lately suggested that the defective element may be a vitamine, and the adoption of this view would bring the disease into relationship with beri-beri and rickets.

3. That it is due to decomposition of the food, giving rise to the absorption of an unknown toxin.

*Post mortem* wide-spread hæmorrhages are found. The blood has the characters of secondary anæmia.

**Symptoms** are at first insidious; the patient becomes weak, breathless, drowsy or languid, with more or less aching of the



bones and joints generally. The gums are soft and swollen, bleeding easily on the slightest pressure.

As the disease progresses, the teeth may fall out, and the breath emits an intensely foetid odour. There may be petechial hæmorrhages around the hair follicles, or large subcutaneous extravasations of blood on the extensor aspects of the limbs, popliteal spaces, etc. Epistaxis or subconjunctival hæmorrhages may occur. Hard, brawny, tender swellings of the calves are due to subcutaneous and intramuscular hæmorrhage. The heart is often irregular, and hæmic murmurs may be present at the base. The patient assumes a cachectic appearance, and is rendered quite unfit for mental or physical exertion. In some cases a peculiar form of night blindness develops, dependent not on permanent ocular changes, but on exhaustion of the retina. Death may be due to heart failure, syncope, or complications such as pneumonia.

**Treatment.**—1. General hygiene must be attended to.

2. Diet should consist of good soup, fresh milk, cream, etc. Effervescing drinks made with fresh lemon-juice are of special importance. Fresh fruits and vegetables should be liberally given.

3. Medicinal.—Calcium chloride may be given if hæmorrhage is profuse, and iron may be needed for the anæmia. Local measures for the bleeding gums, nitrate of silver, antiseptic mouth-washes, etc.

**Infantile Scurvy** sometimes occurs in children brought up on proprietary foods, and is most frequent between the sixth and the twentieth month. The symptoms are tenderness of the lower limbs and an unwillingness to move them, which may simulate paralysis; swelling in the shafts of the long bones of the legs, and sometimes crepitation at their epiphyses, which may become separated from the shaft; anæmia, debility, and in severe cases subcutaneous or internal hæmorrhages. If teeth have appeared, the gums are swollen and spongy. The swellings are due to subperiosteal hæmorrhages. Cow's milk and fresh fruit-juices form the treatment.

## HÆMOPHILIA

A constitutional and usually hereditary tendency to uncontrollable bleeding, sometimes associated with effusion into the joints.



Many varieties have been described, such as—

1. Those in which a slight traumatism is followed by excessive hæmorrhage.

2. Those which exhibit the condition after injuries of certain regions only.

3. Those in which hæmorrhages take the form of attacks of apparently spontaneous bleeding from the nose, uterus, mucous membranes, etc.

**Pathology.**—The blood is much less easily coagulable than in normal individuals; leucocytes and blood platelets are often diminished in number. The walls of the capillary vessels have been shown not to be at fault; and it has recently been demonstrated by Addis that the essential cause is a congenital deficiency of prothrombin. The diathesis is marked by being hereditary, and is transmitted through the female line, although the male is most liable to the *disease*.

**Symptoms** are sufficiently obvious, and need no detailed description. It must be remembered that in these cases death has followed the extraction of a tooth, cutting a corn too deeply, snipping a wart off, and other usually trifling operations. Often the first severe attack of bleeding occurs in infancy, or, on the other hand, it may first appear after adult age. In some instances there are hæmorrhagic effusions into the large joints, which are swollen and painful. Such attacks may be attended with fever.

**Treatment.**—The patient must be protected from injury, and is not a fit subject for operations. On the slightest appearance of hæmorrhage, energetic treatment with styptics must be at once employed. For hæmorrhages from the mouth or nose adrenalin may be used, and for external wounds compression. It may be necessary to plug the nostrils. Calcium lactate should be given in doses of gr. xv (grm. 1·0) every four hours, or a single large dose of ʒi (grm. 4·0) for an adult or gr. xv for a child may be given in solution at once. Horse serum may be injected, and in desperate cases transfusion of human blood has saved life. Obviously, the bleeder must live on a non-stimulating diet, lead as placid a life as possible, and avoid all excess, especially the ingestion of liquids which tend to raise the blood-pressure. No female of the stock should marry.



## PAROXYSMAL HÆMOGLOBINURIA

A condition manifested by the occasional passage of blood pigment in the urine.

**Etiology.**—The affection is most common in the male, and in young subjects. It is very frequently, if not always, associated with syphilis, and the attacks are determined by chill. It is found in a limited proportion of cases of Raynaud's disease. The work of Eason and others has shown that it is due to a toxin circulating in the blood, and becoming linked as an immune-body to the red cells when the blood is cooled below the normal temperature, as it is in the vessels of the skin when exposed to cold. Hæmolysis follows when the corpuscle-immune-body combination returns in the circulating blood-stream to the warmer and deeper part, where it becomes linked with normal complement.

**Symptoms.**—The attack may come on after the morning bath, muscular exertion, or exposure to cold, and is usually associated with pain in the lumbar region. Vomiting, anorexia, and jaundice are sometimes present, and fever often. The liver and spleen may be enlarged and tender. The urine is smoky, blood-coloured, or porter-coloured; it responds to the guaiacum and spectroscopic tests for blood; but on microscopic examination blood corpuscles are absent. During the paroxysm the red cell count is low, and the colour index high. An attack may last from a few hours to a few days, and may be frequently or very seldom repeated.

**Treatment.**—Preventive treatment consists in the avoidance of chill, treatment during the attack in rest, warmth, and hot drinks. Salvarsan, mercury, or the iodides should be used in all cases in which the Wassermann reaction is positive.

## PURPURA

Purpura is not a disease, but symptomatic of some grave change in either the blood or blood-vessels, whereby extravasations of blood into various tissues occur, producing red or blue patches which do not disappear on pressure or after death. In some instances, the cause of the hæmorrhages is known, and these are referred to as *symptomatic purpura*, of which the following are the chief varieties—

1. *Infective purpura*, a form associated with the malignant



fevers—*i.e.* those fevers in which the “rashes” become hæmorrhagic, as already described under typhus, measles, etc.

2. *Cachectic purpura*, a form associated with grave constitutional changes—*i.e.* syphilis, chronic Bright’s disease, cardiac diseases, scurvy, etc.

3. *Toxic purpura*, a form associated with the circulation of certain poisons, such as snake poison, quinine, antipyrin, copaiba, mercury, etc.

4. *Neurotic purpura*, a form which is met with in cases of myelitis, locomotor ataxia, severe neuralgia, purpura urticans, etc.

There are forms on the other hand of which the etiology is entirely unknown, although the condition is very probably due to the circulation of a toxin. Such are—

1. Purpura simplex, most often seen in children, unattended by fever or constitutional symptoms, and disappearing in a fortnight or less.

2. Purpura hæmorrhagica (*morbis maculosus Werlhofii*), a form which occurs without any apparent reason, and not associated with any particular condition. It is seen in two varieties:—

(a) Non-infective purpura hæmorrhagica (possibly an acute form of hæmophilia). It attacks young subjects chiefly. The eruption is severe, the mucous membranes are also affected, and there may be renal or retinal hæmorrhages. Recovery may be speedy, or increasing anæmia may lead to death.

(b) Febrile purpura hæmorrhagica, associated with intense septicæmic infection, high fever, and a septic state of the mouth. Petechiæ, ecchymoses, and bullæ are seen, also hæmorrhages from the nose and gums. The issue is fatal.

3. Arthritic purpura (*purpura rheumatica*) is probably not of rheumatic origin, although acute rheumatism may sometimes be complicated by purpuric rashes. To these the term *peliosis rheumatica* should be restricted. Arthritic purpura is accompanied by slight fever and pains in the larger joints, around which, upon the extensor surfaces, a rash appears. This is partly urticarial, and the purpuric lesions are seated upon the urticarial wheals. There may be œdema of the shins or even of the face.

**Symptoms.**—The presence of petechiæ in the skin, etc., is sufficiently obvious to demonstrate the condition. When not associated with any apparent cause, the more common symptoms



are—marked anæmia, sallow complexion, local œdema or general anasarca, and more or less severe muscular pains. Death may occur from progressive exhaustion, or from internal hæmorrhages.

Severe forms of the disease may be confused with scurvy, but the gums are not swollen, and there is no history of want of fresh air or vegetables.

**Treatment** of symptomatic purpura is the treatment of the cause. In idiopathic purpura we must aim at removing any apparent cause of ill-health, and restoring the tone of the blood-vessels. Ergot, iron, turpentine, strychnine, gallic acid, opium, etc., may be tried. Arsenic is useful in purpura simplex, but should not be given if there is diarrhœa. Adrenalin internally, or calcium lactate gr. xv-xx (grm. 1.0-1.3) every four hours are often useful. Rest in bed should be insisted on. In febrile purpura hæmorrhagica treatment is merely palliative.

## II. DISEASES OF THE THYROID GLAND

### MYXŒDEMA

A disease associated with atrophy of the thyroid gland, a mucinoid infiltration of the subcutaneous tissue, dryness and harshness of the skin, and mental hebetude.

**Etiology.**—Myxœdema is most common in women between thirty and fifty-five years of age; hereditary factors appear to be occasionally present; and the poorer classes are said to furnish the larger number of cases. It is due to atrophy of the cellular elements of the thyroid gland, associated with fibrosis. The cause of these changes is unknown, although an extreme variety of predisposing causes has been tabulated. Its onset has sometimes been preceded by goitre or exophthalmic goitre. Women are affected five times as often as men.

**Varieties.**—Loss of the thyroid function may occur—

1. In adults as the result of disease—myxœdema.
2. Congenitally or before puberty—cretinism.
3. As the result of operation—cachexia strumipriva.

**Morbid Anatomy.**—The changes are most marked in the subcutaneous tissues and thyroid gland. The *subcutaneous tissues* show :—

1. Nuclear proliferation or formation of connective tissue



around the hair follicles and sweat-glands, accounting for the loss of hair and dryness of the skin.

2. Increased deposit of subcutaneous fat.

3. Presence in the subcutaneous connective tissue of a gelatinous cement allied to mucin.

4. Formation of elastic oedematous swellings above the clavicles.

The *thyroid gland* is atrophied, often indurated, and shows scattered groups of cells, the remains of the normal vesicles.

The increase of connective tissue may also affect other glandular organs, such as the kidney, leading to interstitial nephritis, or the outer coat of arteries, or the ganglia of the central nervous system.

**Symptoms.**—The disease is of slow onset. When it is fully developed the patient presents a heavy, stolid countenance. The face is broadened, the lips thick, and there is a diffuse red flush upon the cheeks, contrasting with the yellowish pallor of the rest of the face. The hair is scanty, coarse, and brittle. The teeth become carious, and the speech is heavy, slow, and thick (leathery voice). There is great intolerance of cold. The skin is enormously thickened, *dry*, and often scaly, but does not pit on pressure. The hands and feet are broad and spade-like, and the gait is clumsy. The temperature is always subnormal. The menses are irregular, and there is often a tendency to hæmorrhages. The bowels are constipated. The mind is slow and dull, and later the mental condition may become very grave; hallucinations or delusions leading to dementia or suicidal tendencies may appear. It must not be forgotten that often towards the end albuminuria may occur, and the skin may pit on pressure. Glycosuria is an occasional symptom.

**Treatment.**—The patient must be carefully guarded against cold, and should if possible winter in a warm climate. Warm bathing is also useful. Specific treatment consists in the administration of thyroid extract, either as the *Liq. Thyroidei* (B.P.), or dry, in the form of tabellæ. Small doses should be given at first, and gradually increased up to m. x of the liquor twice daily, or gr. v (grm. 0·3) of the extract thrice daily. As the condition improves, the dose may be gradually lessened, but the patient must be told that she will have to continue the treatment all her life.

**Cretinism** is the infantile or juvenile form of myxœdema, and is due either to the congenital absence of the thyroid body, or an



absence of its functions. It is *endemic* in certain parts of Central Europe, particularly in Switzerland, where it is very common, along with goitre, in deep valleys shut off from air and sunlight. Cretins are often the children of goitrous parents, and may themselves have goitre. *Sporadic* cretinism occurs all over the continent of Europe, in Great Britain, and occasionally in America. Intemperance and insanity in the parents are believed to predispose to it. The *juvenile* form may be the result of atrophy of the thyroid after a fever.

**Symptoms and Signs.**—As regards the subcutaneous infiltration, supraclavicular swellings, and mental defects, the condition resembles an exaggerated form of myxœdema. No disease presents more strikingly characteristic features. The child is dwarfed or stunted in growth; the face is very ugly, moon-shaped, and the cheeks hang in pendulous folds; the tongue is too large for the mouth; and the voice is peculiarly harsh or squeaky, like that of Punch in the show. The hair is coarse, except over the fatty swellings between the scapulæ, which are covered by a soft down-like hair. The belly is very prominent and pendulous; umbilical and inguinal herniæ are common. The gait is clumsy, and of a waddling type. The sexual organs are rarely developed, though in female children the menses may appear once or twice; but sexual desire is never excited, even in those cases that live to adult age. Cretins are usually incapable of being taught reading or writing, and their vocabulary is always limited. They are sometimes peevish and cross, but the majority are placid and affectionate. In untreated cases death usually occurs during childhood, but a few cases reach adult life, without, however, advancing at all in intelligence.

**Treatment.**—The child should be removed from the place where the disease appeared to a more favourable climate, and should have a nutritious diet, with frequent baths and systematic exercise of the muscles. In very young children Liq. Thyroidei m. ij., or gr. i (grm. 0·065) of the extract may be given once daily, and in older children five grains (grm. 0·32) of the extract in tabloid form once daily. The younger the child the greater is the improvement. Treatment must be continued throughout life.

**Cachexia strumipriva** is a condition which sometimes follows operative removal of the thyroid gland in man, and was experimentally produced by Horsley in the monkey. It sometimes arises after even partial removal of the gland, but much more frequently when the operation is complete. There are, however,



cases of complete removal which are not followed by the cachexia, and this is to be explained by the presence of accessory thyroids (parathyroids). The symptoms and treatment are similar to those of myxœdema.

### GOITRE

A persistent enlargement of the thyroid gland, due to hypertrophy and not including new growths. The disease may be sporadic or endemic, the endemic form being prevalent in many parts of Europe, especially in deep mountain valleys, and in certain parts of England ("Derbyshire neck"), but rare in America. It affects women oftener than men, and begins most frequently in late childhood or about the age of puberty. The drinking water of the district has been almost unanimously regarded as the cause of the disease, and in an infected district those who drink from certain wells may become goitrous, while those who drink from other wells remain free. There was until recently no certainty as to the element in the water to which the goitre is due; but it has now been demonstrated by the researches of M'Carrison that it is not the water, but micro-organisms contained in it, which causes the disease; that these organisms are found in the soil, and enter the water from it; that they produce goitre-forming toxins in the alimentary tract; and that many cases can be relieved by the use of vaccines prepared from cultures of the organisms. The disease may apparently be produced by the effects of different organisms in different countries.

The changes in the gland may be (1) parenchymatous—a general hypertrophy; (2) cystic—enlargement of the vesicles; (3) vascular—dilatation of the blood-vessels, causing a pulsatile swelling.

**Symptoms.**—Fulness in the neck; thyroïdal tumour moving with deglutition, the enlargement being either uniform or irregular as the whole thyroid or only a part of it is affected; and evidence of pressure on the trachea, œsophagus, sympathetic, or recurrent laryngeal nerve. Sometimes there may be paroxysmal dyspnœa, which may prove fatal.

**Treatment.**—On the microbic theory, the internal use of thymol gr. x (grm. 0·6) night and morning may be tried. In some cases thyroid extract gr. ij (grm. 0·13) thrice daily, has proved useful, and in others iodide of potassium and the external application of iodine. The drinking water should be boiled. M'Carrison has successfully used vaccines obtained from cultures of the intestinal flora.



If endemic goitre is due to a soil-infection, good sanitation and the use of an uncontaminated water-supply should be the best prophylaxis; and in infected districts the use of such measures has already proved of value.

## EXOPHTHALMIC GOITRE

A disease characterised by enlargement of the thyroid gland, exophthalmos or protrusion of the eyeballs, increased frequency of the heart, and muscular tremors.

**Etiology.**—The disease is much more frequent in women, in whom it most commonly begins between the ages of fifteen and thirty, while it attacks men between thirty and forty-five. The patients are usually of a nervous temperament, and nervous heredity is sometimes apparent. Sudden fright, grief, or shock, are the common exciting causes; depressing diseases or prolonged overstrain may predispose to it.

**Pathology.**—The pathology of exophthalmic goitre is still unsettled, but the prevailing tendency is to regard it as due to excessive activity of the thyroid gland (*hyperthyrea*). Administration of the gland to animals is followed by many of its symptoms, and in myxœdema overdoses of thyroid extract may produce a somewhat similar condition; removal of a part of the enlarged thyroid causes subsidence of the symptoms, and if they recur it is because the remaining portion of the gland undergoes further hypertrophy; histologically, the cellular elements of the gland are found to be in a state of active proliferation; and chemically there is increase of its nucleoproteids and of its iodine content. Yet there are some who, looking to its etiology, to the ocular symptoms, and to the fact that experimental introduction of large amounts of thyroid gland substance has never caused all the symptoms of the disease, still consider it as a disturbance of the central nervous system.

**Morbid Anatomy.**—The thyroid is found to be moderately enlarged and exceptionally vascular, the arteries being dilated and tortuous. The epithelium of its vesicles is proliferated, and columnar instead of cubical; the colloid matter is diminished and more mucinoid, and new tubular spaces are formed. The fatty tissues of the orbit are increased in amount. The heart is hypertrophied. Changes in the cervical sympathetic ganglia and minute hæmorrhages in the brain have been noted.



**Symptoms.**—The cardinal symptoms are exophthalmos, thyroid hypertrophy, tachycardia, and muscular tremor. *Tachycardia* and cardiac disturbance appear early and very constantly. The pulse rate varies from 100 to 200 or more per minute. There is anæmia, and from this and the rapid, violent action of the heart, dilatation and hypertrophy may follow. Hæmic murmurs and murmurs of relative insufficiency are therefore common. Palpitation is frequent and distressing; the carotids pulsate visibly. *Exophthalmos*, or protrusion of the eyeballs, may be but slight, or so marked that the lids cannot be completely closed. It imparts a staring look of fear or horror to the face. It is sometimes unilateral. It may be caused by (1) venous congestion, or dilatation of the retro-bulbar arteries, (2) overgrowth of the orbital fat, (3) contraction of Müller's muscle. It is attended by certain classical signs—

1. Retraction of the upper lid, so that the sclerotic is seen all round the cornea. *Stellwag's sign*.
2. When the patient looks down, the upper lid follows the movement of the eye slowly and imperfectly, so that a larger area of sclerotic is seen above the cornea. *Von Graefe's sign*.

Other ocular phenomena may occur. There may be ptosis (paresis or paralysis of the branch to the levator palpebræ); imperfect convergence on near vision (*Möbius' sign*); temporary paresis or paralysis of the third, fourth, or sixth nerve, causing strabismus. The fundus is usually normal. *Thyroid enlargement* is moderate in degree, the right side being usually the larger. It may occur early or late. The gland is soft and elastic at first, later firmer and nodular. Systolic thrill may be felt over it, and systolic murmur heard. *Tremor* may affect the limbs only, or the whole body. It is most notable on holding out the hands, palms downwards, and consists of rapid, fine, rhythmic movements of the whole hand, but not the fingers separately. Any one of these symptoms may be absent at a given stage of the disease; of them all, tachycardia is the most constant.

Other symptoms that may occur are digestive disturbances—variable appetite, sometimes vomiting, often diarrhoea; wasting; polyuria, glycosuria, albuminuria; flushing of the head and face, free sweating, occasional pyrexia; cutaneous eruptions; mental alterations—depression, melancholia, or even mania.

**Prognosis.**—A considerable number of cases recover, more are much benefited by treatment but may relapse, and about



twenty-five per cent. ultimately die of complications, diarrhoea, cardiac failure, or acute mania. The duration of a fatal case is from several months to several years.

**Treatment.**—Physical and mental rest, in severe cases rest in bed, fresh air, light nutritious diet, and avoidance of stimulants are the general measures. Belladonna and bromides are useful sedatives, and digitalis in considerable doses, or the continuous application of an ice-bag, may reduce the cardiac frequency. Sodium phosphate gr. xv-xxx (grm. 1.0-2.0) thrice daily, sodium iodide, and arsenic, have all at times been beneficial, and electricity to the thyroid is often useful. Application of X-rays to the gland has been tried of late, in some instances with good results, though there is the risk that their destructive effect may induce thyroid insufficiency. Adrenalin and pituitrin have also been employed. In severe cases which are rapidly advancing, operation may be performed. Section of the cervical sympathetic, and ligature of the thyroid arteries, section of the isthmus, and partial thyroidectomy have all their advocates. Great benefit, and sometimes cure, has followed the last of these, but although improved technique has reduced the mortality, the immediate dangers of the operation are considerable. Patients should therefore be fully warned of the risk they run.

Specific therapy, as represented by antithyroidin, the serum of thyroidectomised sheep or dogs, and by rodagen, the desiccated milk of thyroidectomised goats, has not fulfilled expectations.

### III. DISEASES OF THE SUPRARENAL GLANDS

#### ADDISON'S DISEASE

A disease characterised by muscular and vascular asthenia, pigmentation of the skin, and gastro-intestinal irritation; and usually accompanied by tuberculosis of the suprarenal bodies.

**Etiology.**—Addison's disease is more frequent in men than women, and most of the cases occur between twenty and forty years of age. In eighty per cent. of them the lesion of the capsules is tuberculous, and tuberculous lesions may also be present elsewhere. In some instances a blow on the back or abdomen has preceded the onset.

**Morbid Anatomy.**—In the great majority of cases one or both capsules (usually both) are atrophic and tuberculous, and show



interstitial fibrosis, along with caseation, which may result in calcification or softening. In some instances they are affected by other diseases, such as atrophy, interstitial inflammation, or malignant growth. The semilunar ganglia are degenerated and much pigmented, and they may through cicatricial contraction become entangled in the diseased tissue of the adrenals. The nerve fibres show extensive sclerosis.

In exceptional cases the ganglia alone are diseased, and the adrenals escape.

**Pathology.**—There are two theories of the disease—(1) that it is due to loss of the internal secretion of the adrenals, or to the presence in the blood of poisons which they would normally destroy ; (2) that it is an affection of the abdominal sympathetic system, most commonly produced by suprarenal disease. All the recent evidence goes to show that defect in adrenalin, which is secreted by the medullary or chromaffin cells of the adrenalin bodies, is the essential pathogenic factor. In the exceptional cases in which the semilunar ganglia alone are affected, it is yet possible that the chromaffin cells in other parts of the sympathetic system, which also secrete adrenalin, may be involved, or, as Osler suggests, that adhesions around the semilunar ganglia may interfere with the vascular supply of the adrenal bodies.

**Symptoms.**—The onset is generally insidious, feelings of languor and weakness being first to develop. When the disease is established, the main symptoms are those of (1) *asthenia*, shown in extreme muscular prostration, feebleness of the heart, the pulse being weak and irregular, syncopal attacks, and lowness of the blood-pressure ; (2) *gastro-intestinal irritation*—nausea, vomiting, and attacks of diarrhoea ; (3) *pigmentation of the skin*. Pain in the epigastric region is not uncommon, and nervous symptoms (headache, delirium, etc.) may be present. The temperature is *subnormal* as a rule. Anæmia is often absent till the later stages, and emaciation is not marked.

The pigmentation is most marked where pigment is normally found, viz. in the areolæ, scrotum, axillæ, and on parts compressed by clothing or exposed to light. The mucous membranes are often deeply pigmented, presenting blackish patches. The marked exhaustion is altogether out of proportion to the general condition, and death may occur from a syncopal attack brought on by the slightest untoward circumstance, such as the action of a simple purge.

**Prognosis.**—The disease is usually fatal within a year or



two ; some cases die in a few weeks, and others may survive for several years. In some instances, after very slight symptoms and a still slighter amount of bronzing have been noticed for a few months, the patient sinks without any obvious cause into a semi-comatose state, which rapidly proves fatal.

**Diagnosis.**—Bronzing of the skin may occur in many other conditions, *e.g.* pregnancy, exposure to the sun, diabetes, jaundice, and a habitual verminous condition. The diagnosis should not be made in the absence of asthenia.

**Treatment.**—The patient must be treated with the extremest precautions against cold, worry, or fatigue. Complete rest in bed is necessary where asthenia is marked. Adrenalin chloride (10 minims of the 1 in 1000 solution thrice daily) or extract of suprarenal gland (one tablet thrice daily, each tablet corresponding to gr. xv (grm. 1.0) of the fresh gland) may be given, but the benefit is usually temporary. Prolonged hypodermic administration of the drug has been known to cause collapse, and also tremor and insomnia. Symptoms must be treated as they arise ; iron, arsenic, strychnine, and phosphorus have all been recommended. A few cases of improvement or cure following the use of tuberculin are on record.

#### IV. DISEASES OF THE SPLEEN

The diseases of the spleen are for the most part secondary. Thus one may meet with acute congestion in acute infections ; passive congestion in cardiac disease or engorgement of the portal circulation ; infarction, usually anæmic, but sometimes hæmorrhagic, associated with perisplenitis and consequent splenic friction ; abscess in septic conditions ; amyloid disease, in which the organ is very greatly enlarged ; and tertiary syphilitic conditions. The characters of the spleen in malaria and leukæmia have been referred to under those diseases.

#### MOVABLE SPLEEN

A condition frequently associated with general enteroptosis. The organ is usually somewhat enlarged, and may descend into the abdomen so as to be grasped by the hand and moved about. The splenic notch may be recognised. Slight pain in the left side of the abdomen is the chief symptom. A belt and carefully adjusted pad may serve to retain the spleen in its normal position, or no treatment may be required.



## SPLENIC ANÆMIA

## (Primary Splenomegaly. Banti's Disease)

A progressive enlargement of the spleen associated with anæmia, a tendency to gastric or other hæmorrhages, and in the late stages with cirrhosis of the liver.

The disease is most frequent in males. Its etiology is unknown.

**Morbid Anatomy.**—The spleen is much enlarged, its fibrous tissue being increased, and the endothelium of the blood sinuses proliferating, while the splenic pulp is atrophied. The sinuses may be choked by the proliferated endothelial cells. Perisplenitis and infarcts are also present. In the late stages the liver becomes cirrhotic, and jaundice or ascites may occur.

**Symptoms.**—The disease begins with the enlargement of the spleen, and the patient may first complain of a "lump" in the left side of the abdomen. There may be pain in the splenic region, and the tumour is found to be tender. Splenic friction may be heard. The enlargement is progressive, and may become very great, *but the lymphatic glands are not enlarged*. Anæmia usually accompanies the enlargement, but may be comparatively late. The red corpuscles may fall to 3,000,000 per cmm., the hæmoglobin being still more deficient, and the colour index therefore low. The leucocytes are reduced in number. This stage may last for several years, and is succeeded by aggravation of the anæmia, with attacks of hæmatemesis, followed by melæna. Other hæmorrhages may occur, as from the nose, gums, or kidneys. Finally the liver becomes cirrhotic, and the last stage may be marked by jaundice, ascites, and diarrhœa.

The above description applies to Banti's disease. In the allied Gaucher's disease, or primary splenomegaly, enlargement of the liver and spleen is early and great, the skin shows a greyish or brownish discolouration (not jaundice), anæmia is late, and ascites is absent. Leucopenia and hæmorrhages are common to both forms.

**Treatment.**—The splenic origin of the disease is indicated by the effects of *splenectomy*, which in the early stages often results in cure, but in the later stages is of little avail. Iron and arsenic may be of use, and the X-rays have sometimes proved beneficial, but operation is the only hopeful treatment.



## CHARACTERS OF A SPLENIC TUMOUR

1. The enlarged spleen extends *obliquely* downwards and forwards into the abdominal cavity from the left hypochondriac region. It may reach downwards to the iliac crest, and as far as the umbilicus, or even farther, to the right.
2. The fingers cannot be pushed under the ribs in the left hypochondrium.
3. The surface is usually firm and smooth, and the edges rounded. If the enlargement is sufficient, the splenic notch may be felt in the epigastric region, to the left of the middle line.
4. Dulness is uninterrupted over the area occupied by the tumour, and is continued upwards into the normal area of splenic dulness. In tumour of the left kidney, the dulness is broken by a band of tympanitic percussion, due to the position of the descending colon in front of the kidney.
5. Posteriorly the lower border of splenic dulness meets the dulness of the spinal muscles at an acute angle. In renal tumour the whole region of the loin is dull to percussion.

If, on percussing along a line drawn from the umbilicus to the apex of the left axilla, the note is found uniformly tympanitic over the abdomen, and uniformly clear over the lung, there can be no important enlargement of the spleen.

## V. DISEASES OF THE PITUITARY BODY

## ACROMEGALY

A chronic disease characterised by an excessive growth chiefly of the bones of the face and extremities, and by changes in the pituitary body.

**Etiology and Pathology.**—The disease usually begins before the age of forty, and affects women rather oftener than men. The changes in the pituitary body, according to Berthold Fischer, invariably affect its anterior *glandular* portion, which influences the development in size of the body and the growth of the bones. Acromegaly arises from a specific hyper-secretion of this part,



accompanied by an overgrowth of the glandular structure (adenoma or adeno-carcinoma), and is not produced by other forms of pituitary disease. There is a close relationship between; the pituitary body and other glands with an internal secretion; and in some instances the thyroid gland has been found enlarged and the thymus persistent.

**Symptoms.**—Rarely is a more characteristic clinical picture presented than by a confirmed case of acromegaly. The face is egg-shaped, with its broad end downwards; the lower jaw-bone particularly is much increased in size, and may cause the lower teeth, which are wide apart, to project in front of the upper; the tongue is considerably enlarged, and this may cause a similar leathery speech to that of myxœdema. The eyelids and nose are thickened and the ears hypertrophied. The extremities are markedly altered, the hands being huge and spade-shaped, and the nails broad and large. The feet show similar abnormal overgrowth, but the hypertrophy does not extend beyond the wrists and ankles, the long bones being but slightly affected. Later, the spine may become kyphotic, and the simultaneous prominence of the abdomen causes a characteristic “double hump.” The ribs and sternum are also thickened. There is much general lassitude and weakness, and usually *marked headache* and anæmia.

In women the menses are arrested. Defects of vision are often present, the most common being bitemporal hemianopia and optic atrophy. Polyuria and glycosuria may be present in some cases. Warts are frequent upon the skin, which, in marked contrast to myxœdema, is sometimes bathed in sweat.

**Treatment.**—No medicinal treatment has so far proved successful, though thyroid extract and extract of the pituitary body have both been of temporary benefit. Resection of the hypophyseal tumour has been done of recent years, in some cases with marked improvement. If symptoms of increased intracranial pressure are prominent in the late stages, a decompression operation may possibly give relief. X-ray treatment has also been tried, it is said with advantage.



## DISEASES OF THE RESPIRATORY ORGANS

IN order to be able accurately to describe the situation of morbid changes within the chest, we divide its surface into a series of small areas by a number of arbitrarily chosen vertical lines. These are, in front, the median, sternal (running down either border of the sternum), mammillary (running through the nipples), and parasternal lines, the last midway between the median and mammillary; laterally, the anterior, middle, and posterior axillary lines; and posteriorly the scapular (inner border of the scapula) and vertebral lines. Horizontal landmarks are furnished by the ribs. The chest is further divided into a series of *regions*, more or less corresponding with natural landmarks. In front we have mesially the suprasternal, sternal, and xiphisternal regions, and on either side the supraclavicular, clavicular and infraclavicular (the last extending downwards to the third rib), followed by the mammary region, and the inframammary below the sixth rib. Laterally there are the axillary region, which extends downwards to the sixth rib, and the infra-axillary below it. Posteriorly the regions are the suprascapular, scapular, infrascapular, and interscapular,—names which explain themselves.

The upper limit of the resonant percussion note over the *right lung* is found anteriorly from three-fourths to one and a half inches above the clavicle, and posteriorly at the level of the seventh cervical vertebra. The inner margin of the lung runs obliquely downwards and forwards to reach the middle line at the lower end of the manubrium, and thence vertically downwards slightly to the left of the middle line to the insertion of the sixth rib. Its lower border crosses the sixth rib in the nipple line, the eighth in the posterior axillary, and reaches the vertebral line at the level of the eleventh dorsal spinous process. The boundary between the upper and middle lobes is at the level of the fourth rib in the nipple line, and between the middle and lower at the level of the sixth. Behind, the boundary between the upper and lower lobes is at the level of the third dorsal vertebra. The middle lobe is not represented posteriorly.

In the position of its apex and the course of its inner border as far as the fourth rib, the *left lung* corresponds to the right,



except that the inner border lies a little more to the left of the middle line; but from the fourth rib this border curves outwards and downwards, and then again slightly inwards to the sixth rib, leaving uncovered a portion of the pericardium, and forming a small recurved projection (the *lingula*) before passing into the lower border, which runs a corresponding course to that of the right lower border. The left lung has but two lobes, and the boundary between them runs obliquely downwards and forwards from the third dorsal vertebra to the sixth rib in the nipple line.

The margins of the lungs move freely with respiration. The extent of their expansion is in the nipple line from three-quarters to one and a half inches, and in the mid-axillary line, where it is greatest, as much as four inches. To allow for this expansion the limits of the pleural sacs extend considerably beyond those of the lungs in quiet respiration, and hence in pleurisy the area of dulness exceeds the normal clear area of the corresponding lung, in the mammillary line by two inches, in the mid-axillary by three and a half to four inches, and in the scapular by one and a half inches.

The bifurcation of the trachea corresponds posteriorly to the fourth dorsal spine, anteriorly to the lower border of the manubrium.

## PHYSICAL EXAMINATION

### INSPECTION

The patient must be in a good light, falling evenly on either side. Attention must be given to—

#### I. FORM OF THE CHEST—

- (1) General alterations :—Deep, barrel-shaped chest of emphysema; flat or alar chest of phthisis; pigeon-breast of respiratory embarrassment in childhood; rickety or fiddle-shaped chest (vertical groove along the junction of the ribs and costal cartilages); deformed chest of spinal curvature.
- (2) Local alterations :—Local flattening due to contraction of the lung, apical in phthisis, unilateral in cirrhosis of the lung and after pleurisy; local or unilateral bulging from pleurisy, pneumothorax, tumours, aneurysm.

The *circumference* of the chest at the nipple line should also be taken (mensuration).

#### II. MOVEMENTS OF THE CHEST—

- (1) Number of respirations per minute.
- (2) General type of movement (thoracico-abdominal, abdominal, thoracic).



- (3) Rhythm and volume of respirations ; and their special character (inspiratory or expiratory dyspnœa, orthopnœa, Cheyne-Stokes breathing, etc.).
- (4) Local movements (sucking in of intercostal spaces, etc.).
- (5) Deficient expansion (over one apex, over one side, etc.).

### III. PARTS OUTSIDE THE CHEST—

- (1) Box of the larynx (its upward and downward movement).
- (2) Alæ nasi (their action in difficult breathing).
- (3) Bulging of the apices in the neck on coughing.
- (4) Action of the scaleni and other extraordinary muscles of respiration.

## PALPATION

Palpation is chiefly used in the examination of the vocal fremitus, but by its means defective movements, especially at the apex, can be made out even more accurately than by inspection. The sides should be compared by laying the finger-tips of either hand simultaneously at corresponding points. *Vocal fremitus* is the vibration imparted by the voice to a hand laid upon the chest wall. Being produced at the larynx by the vibrating cords, it is intense in those whose cords are long (deep-voiced males) and weak where they are short (women and children). Adipose tissue conducts it badly, and it is therefore weak in the obese. It is *diminished* by the interposition of air or fluid between the lung and the chest wall (pleurisy, empyema, pneumothorax) and increased by consolidation of the substance of the lung (pneumonia, phthisis). But in consolidation it may be diminished if the bronchus leading to the part is plugged by secretion. The patient should then be asked to cough, and upon expulsion of the plug the increased fremitus is restored. Corresponding points upon the two sides must be compared.

## PERCUSSION

The note obtained on percussion over a normal lung is spoken of as *resonant* or *clear*, that obtained over a solid organ or over the muscles of the thigh is *dull*, and over the stomach the note is *tympanitic*. In disease, the pulmonary note may be altered in the direction of dullness by consolidation or fluid effusion, or in the direction of tympanicity under the conditions mentioned below. A hyper-resonant and a tympanitic note differ in degree, not in kind. The following are the chief alterations, with their causes :—

### I. HYPER-RESONANCE—

- (1) Slight (emphysema).
- (2) Marked (relaxation of lung-substance, as in the upper lobe when the lower is compressed by fluid or solid from pneumonia).
- (3) Very marked—Tympanitic note (pneumothorax or large cavity).
  - (a) High-pitched.
  - (b) Medium-pitched.
  - (c) Low-pitched.

### II. DEFICIENT RESONANCE—

- (1) Slight or *relative* dullness (small areas of consolidation surrounded by air-filled lung, thin layer of pleural fluid, thickening of the pleura).
- (2) Marked dullness (massive consolidation,—pneumonia, phthisis).
- (3) Absolute dullness (large pleural effusion).



### III. MIXTURE OF DULNESS AND RESONANCE (Wooden or Boxy Note). (One of the most important signs of cavity.)

#### IV. SPECIAL QUALITY—

- (1) Cracked-pot sound (another important sign of cavity).
- (2) Metallic or amphoric resonance or bell-sound, obtained by auscultating while percussion is made with the edge of a coin upon another coin laid on the chest (pneumothorax and large cavity).

The degree of *resistance* to the percussing finger should also be noted. Increased resistance is a sign of consolidation or fluid effusion.

### SUCCUSSION

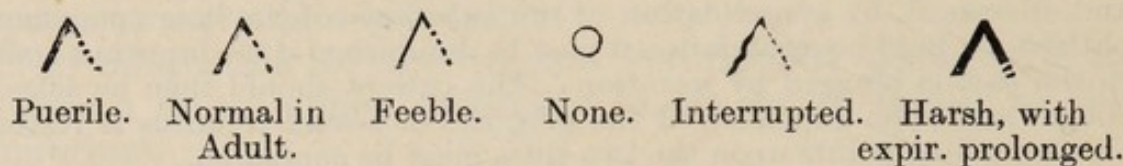
In cases of hydropneumothorax or pyopneumothorax a splashing sound (Hippocratic succussion), due to the mixture of air and fluid, can be elicited on shaking the patient.

### AUSCULTATION <sup>1</sup>

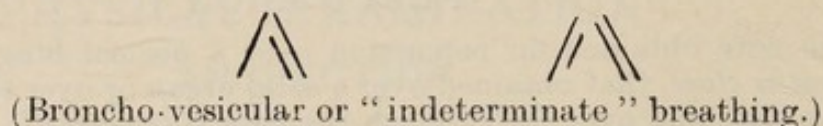
*Listen for—*(1) *Type of Breathing*, (2) *Accompaniments*, (3) *Vocal Resonance*.

#### 1. TYPES OF BREATHING.

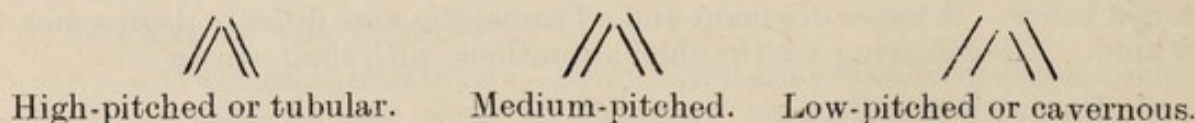
##### (1) VESICULAR OR RUSHING—



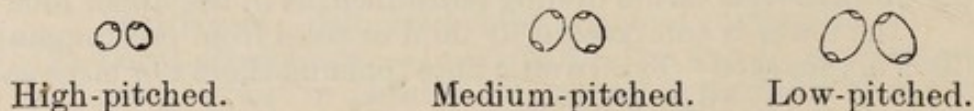
##### Transition Type—



##### (2) BRONCHIAL OR BLOWING—



##### (3) AMPHORIC—



NOTES.—(1) *Vesicular breath-sounds*.—In the auscultation of normal vesicular breathing the *inspiratory sound*, represented in the diagrams by a single line, is a fine, continuous, rushing sound, soft in the adult and loud in the child, and audible from beginning to end of the act. The *expiratory sound*, on the other hand, is thin in quality and of short duration, being

<sup>1</sup> This section is largely derived, by permission of their author, from Dr. Wyllie's *Notes on Examination of the Respiratory System*.



audible only during the earlier part of the expiratory act. In normal vesicular breathing the inspiratory sound generally passes, as represented in the diagram, directly into the expiratory without a break. Wyllie, however, believes that there is often, in perfectly normal vesicular breathing, a distinct break between the two sounds. When the breathing is quiet and easy, the expiratory sound is often totally inaudible, even in children; but in such cases it can usually be brought out by causing the patient to breathe deeply. The term "prolonged expiration" is used to signify not a prolongation of the act of expiration, but only a prolongation of the expiratory sound, resulting from the encroachment of the audible upon the inaudible part of the act. There is indeed one form of breathing, common to advanced emphysema and asthma, in which the act itself is really prolonged, being often much longer than the inspiration. In such "asthmatic" breathing, the type of respiration, primarily vesicular, is as a rule totally masked by the loud wheezing accompaniments of both inspiration and expiration.

(2) *Bronchial or tubular breathing*.—The auscultatory sound of bronchial breathing, indicated in the diagrams by a double line, can be imitated, as pointed out by Skoda, by holding the tongue in the position for the pronunciation of the guttural *ch* sound (as in the German word *Ach*, or the Scotch word *Loch*), and causing the air to pass inwards and outwards over it. The blowing sound thus produced can be made to represent the various pitches indicated in the diagram. There is always in bronchial breathing a distinct break between the sounds of inspiration and expiration, and the two sounds closely resemble each other. The higher-pitched varieties of bronchial breathing should be associated in the mind with conditions of consolidation of the lung substance, such as that of pneumonia, and the low-pitched or cavernous variety with excavation, as in phthisical cavity. *Bronchial breathing is never produced by bronchitis.*

(3) The *amphoric* type of breath-sound can be well imitated by putting the mouth in the position for whistling. Inspiratory and expiratory sounds can thus be produced by causing the air to pass inwards and outwards, and the pitch can be varied according to the variety of amphoric breathing that is being imitated. Amphoric breathing is best developed in pneumothorax, but is also sometimes met with in *very large* phthisical cavities.

(4) In the *healthy chest* the respiratory sounds are purely vesicular (without harshness of quality or prolongation of expiration) over the whole surface of the lungs, except (a) opposite the *roots of the lungs*, at the level of the third dorsal vertebra behind, and the lower part of the manubrium sterni in front, where the proximity of the large bronchi generally renders the breathing *broncho-vesicular*, by the addition of a blowing or bronchial element, most distinct during expiration; (b) over the *apex of the right lung*, especially above the clavicle and spine of the scapula, where, in health, from causes as yet imperfectly ascertained, the vesicular breath-sound has very generally a more or less prolonged, and often harsh or even somewhat blowing, expiration.

The only example of purely *bronchial* breathing that can be heard on auscultating the healthy subject, is the "*tracheal*" breathing, to be obtained by placing the stethoscope over the larynx or trachea. This is low in pitch, and if heard over the apex of the lung would be termed "cavernous."

[Unfortunately some of the great original authorities on auscultation applied the term "bronchial" to the type of breathing heard over the roots



of the lungs, but, as this is partly of bronchial and partly of vesicular origin, the term "broncho-vesicular" or "mixed" breathing is much more appropriate.]

## 2. ACCOMPANIMENTS.

(1) FRICTION sound (in pleurisy) is produced by the rubbing of the two roughened pleural surfaces one on the other. It is usually superficial, of a creaking or grating character, heard both in inspiration and in expiration (though there are exceptions to this), and modifiable by pressure with the stethoscope. When very fine it may be confused with crepitus.

(2) RHONCHI or DRY RÂLES (in bronchitis) are produced by the passage of air through bronchial tubes narrowed by tough secretion. Their pitch varies with the calibre of the tube, whistling or cooing rhonchi (*sibilus*) being produced in the bronchioles, sonorous rhonchi in the larger tubes.

(3) MUCOUS or MOIST RÂLES (bronchitis, phthisis, pneumonia, œdema) are due to the passage of air through fluid in the bronchi or alveoli. They may be large and *bubbling*, crackling (clicking or *consonating* with a metallic quality), or very small (produced in the alveoli), when they may nearly resemble pneumonic crepitus.

(4) CREPITANT RÂLES (*pneumonia*, œdema) are produced by separation of the sticky alveolar walls during inspiration. Very fine crepitus heard only towards the end of inspiration is particularly characteristic of pneumonia.

A further degree of consonating râle is the *tinkling* râle of pneumothorax.

## 3. VOCAL RESONANCE (Auscultation of the Voice).

### (1) SIMPLE INCREASE—

- (a) Slight, comparative.
- (b) Marked (bronchophony).
- (c) Very marked and *articulate* (pectoriloquy).

[The chief conditions which cause increase of vocal resonance are consolidation (bronchophony) and excavation of the lung substance (pectoriloquy).]

### (2) SIMPLE DECREASE—

- (a) Slight, comparative.
- (b) Marked decrease.
- (c) Total absence.

(Decrease of vocal resonance is most frequently due to thickening of the pleura or to pleuritic effusion.)

### (3) QUALITATIVE ALTERATIONS—

- (a) *Ægophony* (nasal timbre).
- (b) With metallic echo (amphoric resonance or *Nachklang*).

[*Ægophony* occurs in pleurisy with effusion when the layer of fluid is thin. Metallic echo is one of the signs of pneumothorax.]







## I. DISEASES OF THE LARYNX

## USE OF THE LARYNGOSCOPE

1. *Position of Patient* : sitting, body and head erect, knees together, head slightly thrown back, mouth wide open.

2. *Lamp* : in line with his ear, nine inches to the left of his head.

3. *Position of Physician* : opposite the patient with mirror properly adjusted to head and eye.

4. Reflect the light upon the fauces at the correct focal distance of the reflector.

5. Warm the laryngeal mirror over the lamp. Test it against the cheek or hand.

6. Direct the patient to protrude his tongue, and to hold it in a towel or handkerchief between the thumb and two fingers (fingers uppermost).

7. Holding the laryngeal mirror like a pen, place its back gently against the uvula, and move your hand slightly towards the patient's left side, so as to keep it out of the line of view.

8. Ask the patient to draw a deep breath, and say "ah," "ur," "eh," or "ee." Be always quiet, gentle, and encouraging ; let each examination be short, even if unsuccessful. Be careful not to burn the mouth, or to push either the uvula or the mirror against the back of the pharynx. Cocaine may be needed to lessen the sensitiveness of the fauces.

## ACUTE CATARRHAL LARYNGITIS

**Etiology.**—Contact with irritating vapours, drinking scalding water, impaction of foreign bodies, exposure to damp and cold air, extension of inflammation from other parts, and acute specific fevers.

**Morbid Anatomy.**—The changes are similar to those of bronchitis.

1. Hyperæmia, causing dryness.

2. Increase of secretion, at first mucous, afterwards mucopurulent.

3. Changes in the tissues beneath, etc., especially—

(1) Œdema of the submucous coat and rarely of the *glottis*.

(2) Changes in the neuro-muscular apparatus of the larynx.

**Symptoms.**—Soreness and dryness, with a feeling of tickling about the larynx, accompanied by hoarseness or complete loss of



voice. There is also a dry cough. The laryngoscope shows swelling of the laryngeal mucosa, and redness and swelling of the cords, which may fail to meet in the middle line.

In children the condition may be *spasmodic* from the first, or may become so after a few days (*false, spasmodic, or catarrhal croup*). The attacks come on at night, with a barking "croupy" cough, stridulous inspiration, and congestion or lividity of the face. They pass off abruptly, leaving behind them slight laryngeal symptoms, or none at all, but they are apt to recur on successive nights.

**Treatment.**—*Catarrhal Form.*—Rest in bed, steam, antiseptic inhalations of eucalyptol or benzoin. A full dose of Dover's powder often cuts the attack short. Demulcent drinks and absolute rest to the voice.

*Spasmodic Form.*—The child should be put in a hot bath, or hot sponges should be applied to the throat. An emetic dose of ipecacuanha may be given, and for some nights small doses of bromide of potassium. The bowels should be kept free.

### ŒDEMA OF THE GLOTTIS (Œdematous Laryngitis)

**Etiology.**—This condition may complicate acute laryngitis, but is much more often met with (1) as a result of extension of inflammation from adjacent parts (erysipelas, cellulitis); (2) as an occasional complication of the acute infections; (3) in syphilis or tuberculosis of the larynx; (4) in acute or chronic Bright's disease. It is rarely primary.

There is inflammatory exudation into the submucous coat, and the mucous membrane is greatly swollen.

**Symptoms.**—The disease may begin as a very acute laryngitis, but dyspnœa rapidly becomes urgent, and threatens suffocation. There are dysphagia, weakness and ultimately extinction of the voice, stridulous respiration and cough, and cyanosis of the face. The paroxysms are separated by intervals of partial relief, but death may occur in any of them.

Laryngoscopically the epiglottis is seen to be enormously swollen, and may conceal the cords. The aryepiglottidean folds may almost meet in the middle line. The mucosa is much reddened.

The swollen epiglottis can be felt with the finger.



**Treatment.**—Leeches or an ice-bag over the larynx, and ice to suck, may suffice in mild cases. If they fail, the epiglottis should be scarified, and if that fails tracheotomy must be performed.

### CHRONIC LARYNGITIS

Chronic laryngitis may be the sequel of acute laryngitis, but it is much more commonly due to *excessive use of the voice* ("dysphonia clericorum"); nasal obstruction; excessive inhalation of tobacco smoke; or chronic alcoholism.

**Morbid Anatomy.**—Congestion and fibrous thickening of the mucosa, with increased vascularity of the cords, are the principal changes. The mucosa may be studded with granulations (enlarged follicles) or superficial erosions.

**Symptoms.**—The severity of the symptoms depends on the cause. The more common are constant hawking and desire to swallow, expectoration of muco-purulent phlegm, and attacks of hoarseness or aphonia. Laryngoscopically the mucosa is reddened and may be granular, and the cords are greyish or slightly injected.

**Treatment** depends on the cause. Nervine tonics, such as strychnine and iron, are nearly always indicated, along with plenty of fresh air, rest to the voice, and *douching the throat with cold water*. Locally, we may use astringents, such as chloride of zinc 10 grains to  $\bar{3}\bar{j}$  (grm. 0.6 to cc. 30—2 per cent.), nitrate of silver (10 grains to  $\bar{3}\bar{j}$ ), chloride of ammonium vapour, and various antiseptic sprays.

### LARYNGEAL TUBERCULOSIS

This is nearly always secondary to pulmonary tuberculosis, and is due to inoculation by the sputum. It leads to infiltration and ulceration, first of the posterior part of the cords and the inter-arytenoid fold, and spreads to the epiglottis and the ventricular bands.

The **Symptoms** are those of chronic laryngitis, associated later with dyspnoea, stridor, or pain on swallowing. Coincident enlargement of the bronchial glands may cause paralysis of the recurrent laryngeal nerve.

**Treatment.**—Insufflations of morphia, curettage or excision of localised swellings, application of lactic acid (at first 50 per cent., increasing to full strength), and the use of the galvanocautery are the chief means of treatment.



## LARYNGEAL SYPHILIS

Syphilis of the larynx is usually a tertiary lesion, although hyperæmia and superficial ulceration may occur in the secondary stage. It attacks the epiglottis first, and may spread to the cords and inter-arytenoid fold. It leads to deep ulceration and necrosis of cartilages, and is followed by cicatricial contraction, causing laryngeal deformity.

The **Symptoms** are those of chronic laryngitis. Hoarseness is prominent, but there is little cough. Stridor may occur where there is laryngeal stenosis.

The **Treatment** is that of tertiary syphilis. Tracheotomy may be required for stenosis.

## TUBERCULAR ULCERATION.

1. Attacks posterior wall.
2. Advances slowly.
3. Marked thickening and infiltration.
4. Pain is commonly present.
5. General condition poor.
6. Evidences of phthisis.

## SYPHILITIC ULCERATION.

1. Attacks epiglottis.
2. Ulceration is more rapid.
3. Little thickening.
4. Little pain.
5. General condition less affected.
6. Evidences of syphilis elsewhere.

## LARYNGEAL TUMOURS

These are for the most part malignant, and epithelioma is the commonest form. At first usually limited to the vocal cords or the posterior part of the cricoid cartilage, the growth rapidly ulcerates, and the disease may spread so as to involve the whole larynx. Hæmorrhage may occur from its surface; œdema of the larynx is common; and perichondritis may lead to necrosis of the laryngeal cartilages.

The **Symptoms** are dyspnœa, dysphagia, and hoarseness, often accompanied by marked pain. In the later stages the submaxillary glands may be involved. If the disease is recognised early, operation is indicated; otherwise palliation is the only treatment.

## LARYNGISMUS STRIDULUS

## (Child-crowing)

A spasmodic disease of the larynx, occurring in infants and young children, and consisting of a temporary closure of the rima



glottidis, which causes great dyspnoea and other symptoms dependent on temporary suffocation. There is no catarrhal affection of the larynx, the condition being a pure neurosis.

**Etiology.**—Predisposing causes are malnutrition brought about by imperfect feeding; syphilis, *rickets*, which is present in the majority of cases, enlarged tonsils, or enlarged bronchial glands. The exciting causes are *reflex nervous disturbances*, brought about by gastro-intestinal irritation (worms, overloaded stomach), teething (*via* trifacial nerve), exposure to cold (vasomotor), or frights and “starts” (cortical).

It will thus be seen that the disease is essentially associated with a neurotic temperament and disturbances of the nerve centres. Escherich regards it as a latent form of tetany, to which the carpo-pedal spasms of severe cases present some analogy.

**Symptoms.**—Sometimes a slight cold precedes the attack, but often the child is apparently well on going to bed, and wakes up suddenly at night with arrested respiration or dyspnoea, and the face rapidly becomes livid. The spasm is usually worse on first awaking. After a time it passes off; air rushes into the lungs, and causes as it does so a long-drawn crowing sound. If the glottis is only partially closed by the spasm, the crowing sound occurs at each inspiration. The child is none the worse for the attack, but often a series of paroxysms takes place, at night only, or both by night and day. In severe cases *carpo-pedal spasms* may occur, the thumbs being flexed into the hands, the fingers flexed over the thumbs, and the wrists flexed, while the legs are extended, the feet bent on the legs, and the soles turned inwards. The prognosis is favourable, death in the attack being very rare.

The condition is readily distinguishable from *diphtheria*, which mothers may fear, by the absence of false membrane and marked local inflammation, and by the history.

**Treatment.**—1. In the attack, dash cold water in the face, or give an inhalation of chloroform. In milder cases give a hot bath, with cold sponging to the chest and back. This may be done twice or thrice daily if the paroxysms continue.

2. In the intervals, bromide should be given in small doses. Any constitutional disease must also be treated.



## LARYNGEAL PARALYSIS

During ordinary respiration the glottis remains partially open, being widened with every inspiration. For the production of voice, the free borders of the vocal cords must be brought almost close to each other in the middle line, only a very narrow chink being between their parallel sides. At the same time the cords must be rendered tense. The narrowing of the chink is brought about by the adductors, viz. the lateral crico-arytenoid, assisted by the arytenoid and *external* thyro-arytenoid muscles. The tightening is due to contraction of the crico-thyroid. The nerves involved are the superior laryngeal and recurrent laryngeal branches of the vagus. It will be easily understood that aphonia or loss of voice may be brought about by local muscular causes or central nervous lesions. The paralysis producing such a condition may be partial or complete, of a functional nature or due to organic lesions.

Gowers gives the following table :—

LESION.	SYMPTOMS.	POSITION OF CORDS.
<i>Total bilateral paralysis.</i>	No voice; no cough; stridor only on deep respiration.	Both cords slightly abducted and motionless.
<i>Total unilateral paralysis.</i>	Absence of stridor except on deep breathing; no cough; voice low and hoarse.	One cord motionless; the other moving freely, and <i>even beyond middle line in phonation.</i>
<i>Total abductor paralysis.</i>	The voice is little changed; cough normal; inspiration difficult and long, <i>with loud stridor.</i>	Both cords lie together, and not separated during inspiration.
<i>Unilateral abductor paralysis.</i>	Little affection of either <i>voice or cough.</i>	One cord not moving during inspiration.
<i>Adductor paralysis.</i>	No voice; perfect cough; no stridor or dyspnoea.	Cords are not brought together, but move during respiration.

### *Causes of Laryngeal Paralysis.*

#### 1. From the nerve side.

- (1) Central lesions. — Bulbar paralysis, disseminated sclerosis, etc.
- (2) Peripheral.—Aortic aneurysm, mediastinal tumour, enlarged thyroid gland, diphtheritic paralysis, all through affecting the recurrent laryngeal nerve.

2. Local lesions.—*Ulceration*, due to syphilis, tubercle, or malignant laryngeal tumours affecting the cords.



3. Functional or hysterical paralysis (nearly always affects the *adductors*).

To sum up. If there be an inability to cough or speak, suspect a serious paralysis. If the *voice is preserved* and the *cough lost* the paralysis is unilateral. If there is normal *cough*, but *no voice* (adductor paralysis), the condition is usually functional. Loud inspiratory stridor means double abductor paralysis.

**Treatment.**—Hysterical aphonia usually responds to electricity; other forms must be treated on general principles, treatment being directed to removal of the cause (syphilis, etc.) whenever possible. For details, consult a treatise on the throat.

## II. DISEASES OF THE BRONCHI

### ACUTE BRONCHITIS

**Etiology.**—Acute bronchitis occurs most frequently in winter amongst elderly people, but it is also very common amongst infants and young children, especially in connection with whooping-cough and measles; insufficient food, scanty clothing, or, on the other hand, excessive confinement in warm rooms, and too warm wrapping up, are the great predisposing causes.

*Exciting causes*—

1. Exposure to cold or wet.
2. Spreading of nasal catarrh.
3. Certain infectious diseases, particularly influenza, measles, and whooping-cough.
4. Inhalation of irritant vapours.

Many organisms may be found in the sputum, and probably more than one of them may set up bronchitis. The most common are the pyogenic cocci, the pneumococcus, and *micrococcus catarrhalis*.

**Morbid Anatomy.**—The changes in the mucous membrane are as follows :—

1. Hyperæmia.—The membrane is injected and dry, and the secretion is scanty.
2. Increase of secretion, at first mucous, later muco-purulent.
3. Swelling of the mucosa, leading in the smaller tubes to marked narrowing of their calibre; desquamation of the ciliated epithelium, and proliferation of the deeper layers.



There are also changes in the underlying tissues, namely, œdema and swelling of the submucous coat, with infiltration of leucocytes.

**Symptoms.**—Acute bronchitis is ushered in with coryza, pains or a sense of oppression about the chest, and cough. The pain is burning in character, and situated behind the sternum. The expectoration at first is frothy and scanty, but soon becomes more abundant and muco-purulent, and often is expectorated in greenish jelly-like masses; later, it may be purulent. The breathing is much embarrassed, and noisy or whistling in character; the temperature is not high as a rule, though there may be some fever, and the skin is moist. The urine is of a febrile type, scanty and high coloured. After a few days the more acute symptoms subside, and convalescence becomes rather slowly established. Death is rare in uncomplicated cases, but the disease is often fatal in the aged through extension to the bronchioles (*capillary bronchitis*) or air-cells (*broncho-pneumonia*). In such cases dyspnœa is aggravated, cyanosis is marked, the lower intercostal spaces are sucked in during inspiration, small crepitations are audible at the bases, and coma precedes death. The so-called capillary bronchitis of children may in rare instances exist as an independent disease, but even when no signs of consolidation have existed during life, broncho-pneumonia is usually found to be present at the autopsy.

**Physical Examination.**—Rhonchi and moist râles are heard all over the chest. These vary in character with the size of the tube in which they are produced. If in the large tubes, the rhonchi are deeply pitched and sonorous, and often accompanied by bronchial fremitus. If in the smaller tubes, they are sibilant or piping, the note being higher the smaller the tube. They are heard throughout both respiratory periods. There is no alteration of the percussion note in uncomplicated bronchitis, except where there are collapsed portions of lung; then there are, of course, impaired resonance and absence of breath-sounds.

**Treatment.**—In the first stage employ the bronchitis kettle, containing a solution of eucalyptus or pinol; administer a brisk saline purge, and a diaphoretic mixture such as—

R	Pot. Citratis	.	.	.	℥iij (grm. 12·0).
	Vin. Antimon.	.	.	.	℥iij (cc. 10·5).
	Spt. Æther Nit.	.	.	.	℥iij (cc. 10·5).
	Spt. Chloroformi	.	.	.	℥ij (cc. 7·0).
	Aq. ad.	.	.	.	℥viiij (cc. 240·0).

Fiat mist. A tablespoonful every three hours for an adult.



The cough may be relieved by Dover's powder, or by heroin, but opiates tend to depress the respiratory centre, and should not be used if there is much cyanosis. When expectoration has become more copious, ammonia, with senega and ipecacuan; or ammonia and iodide of potassium, with paregoric, may be given. The strength must be supported by tonics, hypophosphites, etc. Later, the mineral acids are of use in diminishing the amount of expectoration.

In the capillary form lowering measures are to be avoided. The two great dangers are—

1. Collapse of the lung.
2. Failure of the heart.

In the aged, stimulation is necessary from the outset, either by alcohol, by such drugs as the compound spirit of ether with carbonate of ammonia, or by strychnine hypodermically. In an otherwise vigorous child a brisk emetic may be of much service in clearing the bronchioles of the obstructing secretion. It should be followed at once by stimulation. *Sedatives are nearly always contra-indicated*, as they diminish the expulsive power, causing accumulation of secretion, and hence a tendency to asphyxia.

## CHRONIC BRONCHITIS

(WINTER COUGH)

Chronic bronchitis may be the result of repeated acute attacks, and is very common in the elderly, in whom it tends to recur or to be aggravated every winter. It is also apt to occur in the course of lung disease, mitral affections, disease of the kidneys, and gout. A cold, damp climate is a predisposing cause.

The organisms found in the sputum are similar to those of acute bronchitis.

**Morbid Anatomy.**—(1) The mucosa may be thinned or thickened. Its epithelium is largely destroyed.

(2) The muscular and glandular tissues atrophy, and there is fibrosis of the muscular coat.

(3) Dilatation of the tubes is common.

**Symptoms** are paroxysmal cough and copious expectoration, the sputum varying much in character. It may be mucoid and aerated, non-aerated and muco-purulent, or even entirely purulent. A *nummular* sputum, though most characteristically present in phthisis, may also be found in chronic bronchitis of long standing. Where there is emphysema, dyspnoea, or even



orthopnoea, is also present. The physical signs are rhonchi and moist râles, associated in many cases with signs of emphysema (*q.v.*).

The following varieties are described :—(1) *Bronchorrhœa*, in which the secretion is very abundant, and may be either watery or thin and purulent; (2) *Dry catarrh*, in which secretion is absent; and (3) *Fœtid or putrid bronchitis*, with an abundant fluid secretion of very offensive odour.

Chronic bronchitis must not be looked upon as a *mere bronchial* affection. When it has existed for any length of time it is accompanied by grave changes, of which the nature and results are shown by the following table :—

PATHOLOGICAL CHANGES	PHYSICAL EXAMINATION	CLINICAL SYMPTOMS
1. In the tubes themselves—rigidity of walls and subsequent dilatation (bronchiectasis).	1. Abundance of râles. Indistinct signs of cavity.	1. Copious expectoration at intervals of fœtid, pus-like secretion.
2. Dilatation of the air vesicles and absorption of their walls, with loss of elastic tissues and capillary vessels (emphysema).	2. Altered shape of chest (barrel - shape), use of auxiliary muscles of respiration, hyper-resonance, decreased breath-sounds and prolonged expiration.	2. Dyspnoea through imperfect aeration, increased on exertion.
3. Hypertrophy and dilatation of the right side of the heart.	3. Increase of cardiac dulness. Epigastric pulsation. Later, signs of tricuspid leakage— <i>i.e.</i> general venous congestion, venous pulse, and systolic murmur in tricuspid area.	3. Feeble pulse, œdema, perhaps anasarca; in fact all the troubles consequent on general venous congestion of the viscera.

**Treatment.**—Hygienic measures rank first in the treatment; proper clothing, careful dieting, and change of air do more good than drugs. Any constitutional affection must be treated. Of drugs, the iodides with ammonia are useful, terebene and other balsams (*e.g.* tolu) are frequently employed, and creosote or eucalyptol may be inhaled in putrid bronchitis. Cod-liver oil is of great value. Digitalis and strychnine are extremely beneficial when the pulmonary circulation is more sluggish than usual—in consequence of the strain thrown upon the right heart. When passive congestion is present, rest in bed and cardiac tonics are indicated.



Autogenous *vaccines*, prepared from the prevailing organisms of the sputum, have in many instances brought about marked improvement after failure of the ordinary medicinal treatment.

## BRONCHIECTASIS

Dilatation of the bronchi has already been referred to, as a complication or result of bronchial disease. We must now consider more fully how the condition is produced.

**Etiology and Morbid Anatomy.**—Bronchiectasis is occasionally a congenital defect, but it is usually produced by—

1. Primary disease of the bronchial walls.
2. Contraction of lung tissue in phthisis, etc.

Two forms occur, (1) cylindrical or fusiform, and (2) saccular.

The former is more often produced by strain from within, such as violent coughing, the rigid walls yielding to the expiratory intra-pulmonary pressure. Obstruction of a large bronchus leads to dilatation of the bronchi below the seat of obstruction. Secretion accumulates and decomposes in such bronchi, and causes an inflammatory softening of their walls.

The saccular form is most often produced by—

1. Contraction of new fibrous tissue outside the tubes.
2. Breaking down of the lung substance, causing diminished support, *and yielding of the walls at that point.*

The dilated portion is usually smooth, until ulceration from retained secretion occurs. The walls are very thin, the muscular and elastic tissue being much atrophied.

Bronchiectasis is more frequent in the lower than the upper lobe. It may be a widespread process, but sometimes there is a single saccular dilatation.

**Symptoms.**—If the dilatation is single, physical examination may reveal all the signs of a cavity. The expectoration is copious and foetid, and if allowed to stand, it separates into three layers, the lowest layer being almost pure pus; next, a thin mucoid zone; and a brownish frothy layer on the top of this. Microscopically it shows pus cells and epithelial debris, crystals of fatty acid, and micro-organisms. Elastic fibres, indicative of ulceration, may be present.

The horrible odour is due to valerianic and butyric acids,  $H_2S$ , etc. The mode of expectoration is characteristic, the patient usually bringing up a huge quantity in the morning,



or on moving after resting in a horizontal position for some time.

In other cases, where the bronchiectasis is diffuse, the physical signs are mainly those of the causative disease.

Emaciation, accompanied by night-sweats, and sometimes by fever, is frequently present. Clubbing of the fingers and toes may be marked. Slight hæmoptysis is common, and fatal hæmorrhage has occurred from rupture of an aneurysm in the walls of a cavity.

**Treatment.**—Antiseptic inhalations, such as creosote, help to check the foetor. Urotropine is also of value for this purpose, since it is excreted in the bronchial secretion. Terebene is warmly praised by some. Intratracheal injections of iodoform in olive oil, or inhalations of the *vapour* of creosote, the patient's eyes being suitably protected, have given good results; and incision and drainage of the cavities have sometimes proved successful.

## ASTHMA

A disease characterised by sudden and paroxysmal attacks of expiratory dyspnœa, with a tendency to periodic recurrences, usually in the night.

**Etiology.**—The disease sometimes occurs in children, and when it does so there is usually a history of naso-pharyngeal trouble, measles, whooping-cough, or imperfect recovery after capillary bronchitis. More frequently it is a disease of adult life, in which heredity is an important factor. There may be a family tendency to gout or phthisis, or to nervous complaints, and probably *most* cases have a neurotic origin. Asthma is much more frequent in males than in females, possibly because of their greater liability to bronchial affections generally. An attack may be induced by a variety of circumstances—change from country to town, or from town to country, an overloaded stomach, certain odours, fright or emotion. The exciting causes are either direct irritation of the bronchial mucosa, or indirect irritation through the nervous system or the blood.

**Pathology.**—All authorities agree that the affection is due to diminished calibre of the smaller bronchioles, but the cause of the diminution is still disputed. The following are the main theories of its causation, and of the three the first is the most probable:—

1. *Reflex spasm* of the bronchial muscles, associated with



hyperæmia and turgescence of the mucous membrane lining the smaller bronchioles, and the exudation of a characteristic mucus.

2. *Hyperæmic swelling of the mucous membrane*, like "nettle rash" (Clark), due to vasomotor dilatation.

3. Inflammation of the smaller bronchioles—*bronchiolitis exsudativa* (Curschmann).

**Symptoms.**—1. *Premonitory Symptoms.*—There is usually some visceral disturbance, such as flatulence, etc., but the onset may be quite sudden.

2. *The Attack* occurs most frequently during the night, the patient waking up with a feeling of great dyspnœa; he feels as if there were not sufficient air in the room, and asks for the windows and doors to be opened. The characteristic attitude of asthma is assumed—i.e. the patient grasps some support to fix the shoulder girdle, in order to bring his extraordinary muscles of respiration into play. Expiration is much prolonged, and accompanied by sibilant rhonchi, which cause a peculiar, noisy piping or wheezing. Respiration is not accelerated, though in spite of the extraordinary efforts little air enters the lung. A paroxysm of coughing and expectoration gives relief or even terminates the intense dyspnœa, and sleep may supervene, or a slight lull may be succeeded by another paroxysm.

*The sputum* is usually expelled with the greatest difficulty, and is distinctly peculiar in its composition. It consists of ball-like gelatinous masses, which can be unfolded, and are then found to represent casts of the small bronchioles. Curschmann describes its microscopic appearance as follows:—

*At first* the pellets show two forms of spiral threads (Curschmann's spirals). The one form entangles within its meshwork leucocytes, for the most part eosinophil. The other form contains a central clear filament, surrounded by a spiral network of other filaments of mucus. *Later*, the filaments disappear, and octahedral crystals of phosphates appear in the now mucopurulent expectoration (Charcot-Leyden crystals).

A paroxysm of asthma may last an hour or two, or may be prolonged for several days.

**Physical Signs.**—*During the Attack.*—The thorax is expanded and fixed. The diaphragm moves but slightly, inspiration is short, and expiration prolonged. On auscultation vesicular breathing is drowned by sibilant rhonchi, or later, by



bubbling râles. Percussion reveals marked hyper-resonance (acute emphysema), and the cardiac and hepatic dulness is diminished. This condition disappears at the end of the attack, but after many recurrences tends to merge into permanent and chronic emphysema.

*In the Intervals.*—There may be the usual signs of bronchitis over the lungs, or but little departure from the normal.

The *course* of the disease depends on the immediate cause, and on the amount of bronchitis associated with it. As a rule, the asthmatic attacks tend in time to become less severe, while the bronchial affection grows more pronounced. Death seldom or never takes place from pure asthma.

**Treatment.**—*During the Attack.*—Remove any obvious cause of reflex irritation, such as overloading of the stomach. A brisk emetic often cuts an attack short; nitrites, especially nitrite of amyl, or chloroform may be inhaled. Belladonna, stramonium, and iobelia in combination with ammonia are useful remedies; the fumes of burnt nitre papers, etc., are also recommended. A hypodermic injection of morphia may be needed; and pilocarpin or adrenalin may also be used hypodermically.

The drug which has most claim to be regarded as specific is iodide of potassium. It should be given during the intervals and steadily persevered with. It is often successful, but as often fails, the disease being very refractory to treatment.

*During the Intervals.*—Change of air, careful diet, cod-liver oil, tonics, etc. A suitable climate must be found by experiment in each case. Such sources of reflex irritation as nasal disorders, gastro-intestinal irritation, uterine or urethral disease, must be sought for and removed. The diet must be light and non-stimulating, and the general health should be kept at the highest possible level.

### III. DISEASES OF THE LUNGS

#### EMPHYSEMA

Two conditions are included under this title:—(1) Interstitial or interlobular emphysema, in which, owing to a wound of the lung, or to rupture of air vesicles by violent expiratory effort, as in whooping-cough, air escapes into the interstitial tissues. (2) Vesicular emphysema, in which the air vesicles are over-distended. The former requires no further description.



**Vesicular Emphysema** takes one of three forms :—

1. *Compensatory or Inspiratory Emphysema*.—A condition in which a portion of the lung expands to take the place of a collapsed portion ; seen in broncho-pneumonia, pleuritic adhesions, in areas of old cicatrices (phthisis), etc.

2. *Atrophic or Small-lunged Emphysema*, due to a primary senile atrophy of the lung. The chest is of course small.

3. *Hypertrophic, Substantive, or Expiratory Emphysema*, the usual form, is characterised by—

- (1) Over-distension of the vesicles.
- (2) Atrophy of their walls, with loss of the elastic tissue contained in them.
- (3) Obliteration of blood-vessels, and a consequent diminution of the oxygenating area.
- (4) Changes in the shape of the chest.
- (5) Changes in the right heart.
- (6) General changes due to imperfect exchange of gases between the blood and the air.

**Etiology**.—In violent expiratory efforts with a closed glottis, there is a great increase of intra-alveolar pressure, the strain of which is felt chiefly at the unsupported portions of the lung (apices, free margins). These tend to yield, and over-distension of the air-vesicles results. The general causes are therefore all those factors which keep up a more or less persistent high intra-alveolar tension.

1. Playing on wind instruments, and glass-blowing.
2. Occupations involving severe strain, or the lifting of heavy weights.
3. Chronic cough (bronchitis, whooping-cough).

The element of *heredity* in emphysema, first noticed by Jackson of Boston, is a striking feature. It probably depends upon congenital weakness of the elastic tissue of the lungs.

**Morbid Anatomy**.—The pleuræ are pale. The lungs pit readily on pressure, and have a peculiar soft downy feeling. The dilated vesicles are well seen on the surfaces, and also projecting from *the free margins* of the lungs. The vesicles first become dilated, then coalesce by atrophy and absorption of the septa between the neighbouring cells. The capillaries in the affected area disappear. Though the changes are principally in the air vesicles, there are always more or less marked changes in the bronchial tubes. Bronchiectasis is often present.



**Symptoms.**—Dyspnœa is the most prominent symptom, if bronchitis is absent. The presence of bronchitis causes cough and expectoration. At first present only on exertion, dyspnœa is afterwards a constant feature, and is often aggravated at night. The type is principally expiratory, inasmuch as the loss of elastic tissue prevents the normal expiratory collapse of the lung, which is in consequence accomplished only imperfectly by the voluntary muscles of respiration. But imperfect expiration means that the lung is not completely emptied; hence the entrance of sufficient fresh air is also difficult, and inspiration is impeded in its turn. Ultimately from constant elevation of the ribs, to increase the respiratory surface, the chest assumes permanently the characteristic rounded or “barrel-shaped” form. The important points to remember are—deficient aeration means *dyspnœa* and cyanosis; the retention of waste products within the blood means impure blood and defective nutrition; the loss of capillary vessels is followed by increased resistance in the pulmonary circulation, leading to hypertrophy and afterwards to dilatation of the right heart. This causes general venous congestion, dropsy, and cyanosis. Patients are often ill-nourished and emaciated.

**Physical Signs.**—Barrel-shaped chest, prominent sternum, deep sternal fossa, prolongation of the expiratory part of the respiratory murmur, and a hyper-resonant percussion note are the more common features. The auxiliary muscles in the neck, etc., are all employed in respiration to increase the air capacity of the chest. The areas of cardiac and hepatic dulness are encroached upon, and the margin of the lung becomes fixed in the position of full inspiration, from the disappearance of the elastic tissue. A zone of dilated venules along the attachment of the diaphragm is spoken of by some writers. The epigastric pulsation and altered position of the apex beat are important signs.

**Treatment.**—That of chronic bronchitis. Sudden and grave attacks of dyspnœa, with dilatation of the heart, may be treated by a mercurial purge, inhalation of oxygen, venesection, strychnine, and later digitalis.

## COLLAPSE OF THE LUNG

Collapse of the lung is not a disease, but a part of other diseases. It may be of four types—

1. *Congenital Atelectasis.*—A condition found in weakly new-



born children, when there is not sufficient inspiratory power to inflate the alveoli. The collapsed patches are of a slate colour and sink in water.

2. The form due to pressure from without (pleural or pericardial effusion, pneumothorax). The *whole* lung may collapse.

3. Collapse due to wounds of the chest-wall, and perforation of the pleura. In this form the lung at first is congested, but finally becomes anæmic.

4. Ordinary or lobular collapse from obstruction to the entrance of air, as seen in broncho-pneumonia. Two theories are advanced to explain this condition—

- (1) That a bronchus is plugged by a pellet of mucus which acts as a ball valve—*i.e.* allowing air to pass *out*, but none to *enter*.
- (2) That the plug does *not move at all*, but merely prevents access of *air* during inspiration. The alveolar air is exhausted by absorption, and none being taken in to replace it, the lobule collapses.

Collapse may occur where there is no plug, if there be paralysis of the respiratory movements. This is due to the elastic recoil of the lung tissue, aided by absorption of air by the blood-vessels.

**Morbid Anatomy.**—The collapsed portion is shrunken, and lies at a lower level than the surrounding lung. It is at first congested, and its cut surface is dark red, smooth, and glistening; later it becomes firmer, and is dry and of a bluish-grey colour.

**Symptoms.**—If the collapsed area be extensive, any already existing dyspnœa is increased, the pulse becomes rapid, and there may be cyanosis; in slight cases the symptoms are merely those of the primary disease. The physical signs are subcrepitant râles, weakened breath-sounds, and if the area be extensive, dulness and possibly tubular breathing. Collapse often occurs in measles, whooping-cough, and other conditions which give rise to broncho-pneumonia.

**Treatment** depends on the cause. Emetics or diffusible stimulants are indicated.

## ŒDEMA OF THE LUNG

**Definition.**—An accumulation of serous fluid in the air vesicles, bronchioles, and interstitial tissue of the lungs.



**Etiology.**—Œdema is frequently associated with morbid conditions of the blood, such as Bright's disease, and the anæmic disorders. It is also common in conditions which give rise to passive congestion, such as—

- |                                     |                          |
|-------------------------------------|--------------------------|
| 1. Valvular disease of the heart.   |                          |
| 2. Malignant fevers.                |                          |
| 3. Paralysis.                       | } Hypostatic congestion. |
| 4. Long-continued rest on the back. |                          |

It may occur *after the use of pilocarpin*.

**Morbid Anatomy.**—The lung is heavy and bulky. On pressure it pits, and a quantity of frothy blood-stained serum exudes.

**Physical Signs.**—The breath-sounds are deficient, and masked by fine râles. The percussion note may be resonant at first, but afterwards becomes dull at the bases. The explanation of the resonance is that a lung, when *partially collapsed*, gives forth a resonant sound, *due to diminished tension of the lung tissue*.

**Symptoms.**—Those of serious pulmonary embarrassment (already described under collapse of the lung), *plus* the abundant expectoration of frothy serum.

**Treatment** is that of the cause. Dry cupping is useful, and inhalations of oxygen may be tried. Ammonium carbonate and cardiac tonics are also indicated.

## CONGESTION OF THE LUNGS

**Active congestion** is virtually a synonym for the early stage of inflammatory affections.

**Passive congestion** may be (1) mechanical, (2) hypostatic.

**Mechanical congestion** is due to disease or relative insufficiency of the mitral valve. The resulting engorgement of the pulmonary circulation leads to dilatation and tortuosity of the capillaries, interstitial fibrosis, and hæmorrhages into the alveoli and interstitial tissue. The lung is thus dense and resistant to the knife, and of a brownish-red colour from pigmentation (*brown induration*). The symptoms are dyspnœa, cough, and expectoration. Hæmoptysis is common, and the sputum contains alveolar cells pigmented by altered hæmoglobin. Fine moist râles are audible at the bases.



**Hypostatic congestion** results from long-continued lying on the back, *combined with weakened action of the heart*. It is thus common in the severer fevers, in exhausting diseases, and in cerebral palsies. It affects the dependent portions of the lung (bases), which are gorged with dark blood, sometimes to such an extent that they are airless, and sink in water (*hypostatic pneumonia*). Slight dulness at the bases, feeble respiration, and fine moist râles are its chief signs; symptoms, in the adynamic state of the patient, may be very few.

**Treatment** in both conditions is mainly that of the cause. In mechanical congestion, if the engorgement is very severe and the heart much overloaded, bleeding may be necessary.

## HÆMOPTYSIS AND PULMONARY INFARCTION

**Hæmoptysis** or spitting of blood is a symptom of many conditions, of which the most important are the following :—

1. *Phthisis pulmonalis*.
2. Other diseases of the lungs (pneumonia, cancer, and sometimes gangrene, abscess, and bronchiectasis).
3. *Affections of the mitral valve*.
4. Ulceration of any part of the respiratory tract.
5. Aneurysm.

It may be due to injury, and severe coughing, as in bronchitis, may cause slight hæmoptysis. In rare cases it is the expression of vicarious menstruation, and it may occur in such blood diseases as purpura. It may be simulated by malingerers or in hysteria. The diagnosis between hæmoptysis and hæmatemesis has been considered at p. 172.

**Pulmonary infarction** is in most cases due to embolism of a branch of the pulmonary artery, but is not an invariable consequence, since, although the pulmonary vessels are end arteries, their capillaries are wide, and the bronchial arteries help to maintain the circulation. The infarct is a wedge-shaped mass underlying the pleura, dark red at first and airless, but later becoming whitish and finally undergoing fibrosis. The pleura over it is inflamed. Infarcts are usually multiple, and they are more common in the lower than the upper lobe. As a rule they are about the size of a walnut, but may be much larger.

The *symptoms* are dyspnœa, pleuritic pain, and hæmoptysis, the blood being dark and blackish in colour. There may be



pleuritic friction, and sometimes dulness and feeble breathing. Embolism of the main artery results in sudden death.

**Treatment of Hæmoptysis.**—Unless a large artery is opened, bleeding usually ceases spontaneously, and the patient should be reassured. Absolute rest and quiet, light food given cold, ice to suck, and injections of morphia and atropin suffice in the majority of cases. Inhalation of nitrite of amyl is a valuable measure. Ergot raises the intrapulmonary blood-pressure and may do harm. In protracted cases, saline purgatives and aromatic sulphuric acid are indicated. Adrenalin is useless.

## LOBAR PNEUMONIA

An acute specific infection associated with consolidation of one or both lungs, and with symptoms of toxæmia, and usually due to the *micrococcus lanceolatus* of Fränkel.

Lobar or croupous pneumonia is characterised pathologically by an inflammation of which the exudate is *rich in fibrin and contains large numbers of pneumococci*; and clinically by an abrupt onset, a definite course, and termination by crisis. It is usually basal, but may involve more than one lobe.

**Etiology.**—Croupous pneumonia is a most common affection, occurs frequently in winter and spring, and attacks all ages, though it is more frequent before the age of ten, and between twenty and fifty, than at other ages. Males are more often attacked than females, and there is a strong tendency to recurrence. Amongst the more common exciting causes are draughts, intemperance, exposure to inclement weather, or irritating gases. Pneumonia sometimes directly follows blows upon the chest. It frequently occurs in epidemic form, and its spread is favoured by insanitary conditions. It is most fatal in the aged, and those who are debilitated by alcohol or otherwise.

*The micrococcus lanceolatus* or *diplococcus pneumoniae* is a small capsulated diplococcus, sometimes arranged in chains. The free ends of the cocci are lancet-shaped. The organism is Gram-positive, and grows best on blood-serum or blood-agar. Other organisms occasionally found are *Friedländer's pneumobacillus*, a short capsulated rod, Gram-negative, and growing freely on ordinary agar, and various staphylococci and streptococci; but these are more definitely associated with lobular pneumonia.

The pneumococcus is very commonly present in the blood as well as in the sputum. It exists in the mouth of healthy people, but outside the body it is killed by a brief exposure to sunlight.

**Morbid Anatomy.**—It is convenient to describe four stages,



those, namely, of (1) Hyperæmia or engorgement, (2) Red hepatisation, (3) Grey hepatisation, and (4) Resolution.

*First Stage: Engorgement or Splenisation.*—The lung is injected, dark red, and heavy, and pits under the finger; on pressure, there exudes a frothy serum tinged with blood and slightly aerated. The lung still floats in water.

*Second Stage: Red Hepatisation.*—The part involved is solid and friable, presents a granular or red granite appearance, is airless, and sinks in water. The alveoli are filled with a coagulated exudation, which shows under the microscope fibrin, a few leucocytes, red corpuscles, proliferated alveolar epithelium, and pneumococci.

*Third Stage: Grey Hepatisation.*—The lobe has now the appearance of grey granite, the lung substance is softer and more friable; on pressure, a dirty purulent fluid exudes. The grey appearance is due to four factors—

1. Decolorisation of the red blood corpuscles.
2. Obliteration of the alveolar blood-vessels from pressure.
3. Fatty degeneration of the coagulated material.
4. Great infiltration of leucocytes.

A more advanced stage, in which the lung tissue is bathed in purulent fluid, is known as *purulent infiltration*. It is probably inconsistent with life.

*Fourth Stage: Resolution.*—Resolution of the inflammatory exudation is brought about principally by absorption (autolysis), but partly by liquefaction and expectoration.

Pneumonia may affect a lobe, most commonly the lower, or the whole of a lung, or it may attack both lungs. Double pneumonia occurs in about ten per cent. of cases. Different parts of the same lung may at the same time show different stages. There is always some degree of pleural inflammation over the affected area, and when pleurisy is clinically prominent, the disease is sometimes called pleuro-pneumonia.

Moderate enlargement of the *spleen* is very common.

The **Physical Signs** show a rough correspondence with the stages of the morbid process. They are as follows:—

*First Stage.*—*Percussion* yields a slight dulness, but sometimes even a slight hyper-resonance (Skodaic resonance) may be present. *Auscultation* reveals the characteristic fine crepitations, compared to the sound produced by the rubbing together of hair between the fingers. This fine crepitus is due to the separation of



the moist surfaces of the alveoli from each other. It is therefore heard towards the end of inspiration, and not during expiration.

*Second Stage.*—Gives the signs of consolidation, viz.—

*Inspection* shows diminished movement.

*Palpation* confirms this, and also demonstrates *increased* vocal fremitus.

*Auscultation* reveals the absence of vesicular breathing, but presence of typical tubular breathing. Crepitations may have entirely disappeared, or a coarser and more metallic crepitus may be heard over the consolidated area. *Vocal resonance* is increased to the pitch of bronchophony, or even whispering pectoriloquy.

*Percussion* yields a dull note (not absolutely so flat as in pleuritic effusion). In central pneumonia dulness may be absent for several days.

*Third Stage.*—This stage of grey hepatisation can scarcely be differentiated by physical examination from that of red hepatisation.

*Fourth Stage or Resolution.*—Here we have a speedy clearing up of the exudation, and a return to the normal condition. Small râles or *coarse* moist crepitation (*crepitus redux*) may be heard, and this returning crepitus is often audible both on inspiration and expiration. The dulness becomes less marked, and the movement of the side is increased.

**Symptoms.**—The incubation period is probably short. Croupous pneumonia is usually ushered in with one or more rigors, *rapid* rise of temperature, and localised pain, plus the ordinary accompaniments of the febrile state. The pain may be at first referred to the abdominal region. The fever is continued, and its average height in the adult is about 103° or 104° F. In old people it may be comparatively slight; in very acute cases it may reach 105° or even 106°. As the disease progresses, several characteristic features develop. The pain becomes less marked, but there is greater dyspnœa, and a marked disproportion *between respiration and pulse*. The former may be from thirty to seventy per minute and the pulse perhaps only 110. The cough becomes either hacking or paroxysmal in character, and there is expectoration of the *rusty, viscid phlegm, almost pathognomonic of this condition*. The pneumonic countenance



develops—*i.e.* flushed face, malar lividity, dilated pupils, and crops of herpes round the mouth. The urine is highly febrile, *chlorides are markedly diminished*, urates increased, and albuminuria is generally present. The spleen is sometimes definitely enlarged. Between the fifth and eighth day the symptoms may *abate quite suddenly*, rapid recovery taking place; but often, instead of this happy termination, the temperature increases, or perhaps falls to subnormal, the *pulse becomes more rapid*, the tongue dry and brown, the sputum less viscid and prune-coloured, the patient falls into the typhoid state, and death takes place, most frequently from heart failure. Sometimes prolonged exhaustion, or œdema of the sound lung brings about a fatal issue. A *pseudo-crisis* is common about the fourth or fifth day, the temperature falling rapidly to normal, but *the pulse does not fall with it*. In twenty-four hours the temperature may be as high as ever. At the true crisis, pulse and temperature fall together, usually about the eighth to the tenth day, but some cases may abort earlier. Along with the fall of temperature there is often profuse sweating, or there may be diarrhœa. In many cases lysis takes the place of crisis, and the fall of temperature is prolonged over several days.

In favourable cases there is a marked *leucocytosis*. Its absence makes the prognosis bad.

#### *Special Points to Note—*

1. The *viscid rusty sputum* (if it becomes prune-coloured, this is an extremely bad sign).
2. Marked disproportion between pulse and respiration.
3. Diminished chlorides in the urine.
4. The degree of leucocytosis.

As failure of the right side of the heart is a common cause of death, special attention should be paid to the state of the pulse, and to the condition of the second sound of the heart in the pulmonary area.

**Varieties.**—*Double pneumonia* much increases the gravity of the prognosis. In *wandering pneumonia*, as one lobe recovers another is involved, and the disease may cross to the other side. In the alcoholic, delirium tremens is frequent, and in these cases and in the aged the onset is insidious. In *central pneumonia* physical signs may be late in appearing, but the symptoms are characteristic. In *asthenic* or *typhoid* cases toxic symptoms are prominent from the outset. *Delayed resolution* is not uncommon, even in otherwise healthy patients.



**Complications.**—

1. Bronchitis, pleural effusion, *empyema*, rarely gangrene of the lung.
2. Endocarditis or pericarditis.
3. Meningitis.
4. Jaundice.
5. In children, otitis media.
6. Abortion.

**Prognosis.**—There is no prognosis of pneumonia; each case must be judged by itself. The most constant determining factors are the presence or absence of *alcoholism*, the age, and the degree of leucocytosis. The state of the heart, as already indicated, is also of importance. The second pulmonic sound is accentuated owing to obstruction to the intrapulmonary circulation; disappearance of this accentuation before the signs of consolidation begin to diminish suggests approaching cardiac failure. Irregularity of the heart, signs of dilatation, and cyanosis are also ominous.

**Treatment** depends entirely on the type of case, and the condition of the patient. Routine treatment is the worst of all treatments. Answer the following questions before prescribing. Is the patient full-blooded, and is there a full bounding pulse? Is the pulse feeble, irregular, or intermittent?

In the first case, in a young and previously healthy adult, if there be cyanosis, or signs of dilatation of the right heart, blood-letting to the extent of a few ounces may perhaps relieve the strain, but more generally treatment should be directed to maintaining the strength from the outset.

In the latter case we can hope for nothing from a depressing treatment, so stimulants must be resorted to, such as alcohol, ammonium carbonate, egg and brandy mixture, quinine, ether, etc. The giving or withholding of alcohol depends upon its effect upon the pulse; should the pulse rate fall and the tongue become moist it may be continued. Chronic alcoholics require free stimulation. In asthenic cases, strychnine hypodermically is necessary from the start, and normal saline may be given by the rectum or by the skin. Oxygen inhalations are used where there is cyanosis, but it is doubtful whether they have saved many lives. When there is evidence of failure of the heart (weakness of the second pulmonary sound, etc.) digitalis should be resorted to. Many prescribe it from the outset.

The diet should consist of milk, beef-tea or broths, white of



egg, and so on. The patient should be as little moved as possible, and the bed-pan must be used. Constipation must be dealt with, and in all cases the bowels should be freely opened at the beginning, preferably by a mercurial. This tends to relieve the toxæmia to which so many of the symptoms are due. As in other fevers, an airy room and good nursing are essential.

Remember that narcotics are not well borne in respiratory embarrassment as a rule. Chloral should be avoided, but *if pain be excessive* a hypodermic injection of morphia does *more good* than harm, notwithstanding that theoretically morphia is contra-indicated. It should not be given later than the first few days of the illness. Paraldehyde in doses of one to two drachms (cc. 4 to 8) is safer though less effective. It may be given by the rectum to avoid its unpleasant taste. The pain may also be relieved by poultices, which, however, are of doubtful use if carelessly made, or by application of ice. Cold packs applied to the trunk only, and frequently repeated, are very useful in relieving both pain and fever. Depressant antipyretics are to be avoided.

The results of *serum treatment* are not unequivocally encouraging, but vaccine treatment would seem to be of better promise. Where possible, an autogenous vaccine should be used.

## BRONCHOPNEUMONIA

(CATARRHAL OR LOBULAR PNEUMONIA)

Inflammation of the terminal bronchi and air vesicles.

**Etiology.**—The disease is often primary, but rather more frequently secondary, either to the *specific fevers*, among which measles, whooping-cough, and diphtheria are its most common precursors, or to the entrance of particles of food or of discharges into the larynx (*aspiration pneumonia*). It is most frequent in childhood, before the fifth year, but it is also common in old age as a complication of exhausting diseases. It may result from the extension of a simple bronchitis, it may be a part of tuberculous disease of the lung, or it may follow the inhalation of irritating gases. Rickets, diarrhoea, and malnutrition are predisposing causes; chill or exposure may determine an attack.

The organisms most commonly found are Friedländer's pneumobacillus, and the pyogenic staphylococci and streptococci. The pneumococcus is less common, but may be found in the primary form.



**Morbid Anatomy.**—The disease affects both lungs, and begins in the terminal bronchioles, spreading thence to the infundibula and alveoli. The consolidated patches have therefore a *lobular* arrangement, but if many adjacent lobules are affected the consolidation may be almost lobar. The *bronchioles* are inflamed and frequently plugged with mucus, and their walls and the surrounding interstitial tissue are infiltrated with small cells. The walls of the *air vesicles* in the consolidated area are congested, their epithelium is swollen, and their lumen is filled with proliferated epithelial cells, leucocytes, and a mucous or muco-purulent (*not fibrinous*) exudate. Many lobules are *collapsed*, but not inflamed, from plugging of the bronchioles.

When cut into, the small consolidated areas are seen to be conical in shape, with their bases towards the pleura, reddish in colour, with indefinite margins, and separated from each other by crepitant lung tissue. Adjacent lobules may be emphysematous (compensatory emphysema). The bluish-grey collapsed areas are most numerous in the lower lobes.

**Symptoms.**—In *primary* bronchopneumonia the onset is abrupt; in the *secondary* form the symptoms are at first merely those of bronchial catarrh; but the temperature rapidly becomes high, and the fever is markedly oscillating, or irregularly remittent in character. The dyspnoea becomes marked, and the pulse rapid, feeble, and sometimes irregular. The face may be flushed or cyanotic. The cough is harsh, short, and painful. The sputum, which is scanty, is *never rusty*, but may be streaked with blood. The disease ends by lysis, and frequently there are recrudescences of fever after the temperature has become normal. Loss of flesh is marked, and difficulty might be experienced in diagnosing this affection from acute tuberculosis. Death often takes place from asthenia; or recovery is followed by a rather tardy and interrupted convalescence. Aspiration pneumonia is very fatal, and may go on to gangrene. If resolution is delayed, bronchopneumonia is sometimes followed by phthisis, the softening inflammatory products forming a suitable nidus for the tubercle bacillus.

**Physical Signs** are very uncertain. The chief signs over the consolidated areas are increased vocal resonance and fremitus, with *slight* tubular breathing and *small* moist or sub-crepitant râles. There may be scattered patches of dulness, usually only relative, but seldom anything like lobar condensation. The intercostal spaces are often sucked in over the collapsed areas.



**Treatment.**—*Avoid lowering treatment.* The diet should be light and nourishing, and the bowels should be evacuated daily. Stimulant expectorants,—ammonia, senega, and ipecacuan,—are the chief drugs, and an emetic dose of ipecacuan may be needed if the tubes are blocked. Strychnine hypodermically or alcohol may be used as stimulants. Jacket poultices are very comforting, and often of much service. Immersion in a hot mustard bath for two or three minutes, during which the chest is firmly kneaded, frequently eases the respirations and lessens cyanosis. *Great care should be taken during convalescence.* Woollen clothing, cod-liver oil, hypophosphites, and malt extract, etc., must not be forgotten.

## CHRONIC INTERSTITIAL PNEUMONIA

(CIRRHOSIS OR FIBROSIS OF LUNG)

An induration of the lung tissue, characterised by overgrowth of the fibrous elements, and attended clinically with symptoms of pulmonary embarrassment.

**Etiology.**—Interstitial pneumonia is a condition secondary to other diseases of the lung. Thus it may follow—

1. Bronchopneumonia.
2. Lobar pneumonia (very rarely).
3. Chronic pleurisy (pleurogenic cirrhosis).
4. Compression of a bronchus.

It is occasionally due to syphilis.

**Morbid Anatomy.**—The overgrowth of interstitial connective tissue produces the following changes :—

1. Encroachment upon and subsequent obliteration of the air vesicles.
2. Changes in the bronchial tubes, their muscular coats being replaced by fibrous tissue. The tubes subsequently yield to intra-pulmonary traction, and form bronchiectatic cavities (*see* Bronchiectasis).
3. *Obliterative changes in the smaller pulmonary and bronchial blood-vessels.*

The lung as a whole is shrunken, often enormously, airless, and hard. The opposite lung is emphysematous, and the right ventricle is hypertrophied.



**Symptoms.**—The disease, which is usually unilateral, runs a very chronic course. Cough and dyspnœa are the chief, and may be the only symptoms complained of for a long time. Later, the symptoms are those of bronchiectasis, or those of failure of the right heart.

**Physical Signs.**—The affected side is retracted, much flattened, and the intercostal spaces more or less obliterated. The percussion note will depend on the state of the bronchi, ranging from *absolute dulness to a boxy note*; indeed, in the later stages, the physical signs may be those of small cavities with extensive bronchitis. The retraction may lead to compensatory emphysema of the opposite side, dragging of the heart out of position (to the right or to the left and upwards), dragging on the great vessels, etc.

**Treatment** consists in maintenance of the general condition, light employment in the open, and attention to bronchiectatic or cardiac conditions as they arise.

**Pneumonokoniosis** is a fibroid condition of the lung due to the inhalation of various kinds of dust. It has been recently maintained that, at all events in anthracosis, the dust may reach the lungs by way of the intestinal tract, but the work of L. Findlay shows that this is not the case. The morbid changes are very similar to those of interstitial pneumonia, but the appearance of the lung differs with the kind of dust inhaled. In *anthracosis*, due to the inhalation of carbonaceous matter (colliers), the lung is black, and the fibrosed areas are surrounded by areas of emphysema. In *silicosis*, due to inhalation of particles of stone (stonemasons, grinders, potters), the colour is a steely grey. *Siderosis* results from inhalation of oxide of iron, and so on. The main symptoms are those of bronchitis and emphysema, followed by bronchiectasis; phthisis sometimes develops at a later date.

## TUBERCULOSIS OF THE LUNGS

(PHTHISIS—*φθίνομαι*, *I waste*)

Phthisis pulmonalis may be acute or chronic. The acute form exists in two varieties, pneumonic and bronchopneumonic.



*Pneumonic phthisis*, which is uncommon, closely simulates lobar pneumonia, but usually attacks the upper lobe. The whole lobe or the greater part of it is solidified; miliary tubercles may be found closely aggregated, or few and scattered; there may be foci of early softening, or an old chronic lesion from which the acute process has sprung. The onset is abrupt, and the signs of consolidation characteristic; there may even be rusty sputum. But the fever is slightly more fluctuant than that of pneumonia, and the usual defervescence does not occur. After some time, the bacilli may be found in the sputum. The disease ends fatally within a month or two.

*Bronchopneumonic phthisis* (*galloping consumption*) has a somewhat more gradual onset, and like bronchopneumonia, may in children follow measles and whooping-cough. Both lungs are attacked, but the changes (branching areas of caseation or softening, small ragged cavities) are most advanced in the upper lobes. Dry pleurisy is constantly present. Signs of diffuse bronchitis, followed by localised (apical) dulness and friction, accompany the bronchopneumonic symptoms. The fever is hectic; sweating is free, and emaciation rapid. Tubercle bacilli and elastic fibres are to be found in the sputum. Death takes place in a few months after the onset.

**Chronic Phthisis.**—The etiology of this, the most common form of pulmonary tuberculosis, is the same as that of tuberculosis in general (*see* p. 111), but infection by inhalation assumes a special prominence. The general morbid anatomy of tuberculosis has also been discussed. It remains to be noted that the first seat of the organisms is in the alveoli and terminal bronchioles, where they set up an inflammatory reaction, and that they are disseminated by the lymph stream from these foci as centres. The early changes, then, are those of peribronchitis and lobular pneumonia, and these are followed by caseation, softening, and the formation of cavities. The bronchial glands are secondarily involved, but in children they may form the primary focus from which the lungs are infected.

An area of lung tissue infected by the tubercle bacillus passes in a typical case through three main stages:—(1) the stage of invasion, (2) that of consolidation, (3) that of softening and excavation. To these stages certain physical signs and clinical symptoms very roughly correspond, and in the following table the main features of a typical case are briefly summarised.



MORBID ANATOMY.	PHYSICAL SIGNS.	CLINICAL SYMPTOMS.
First stage. { 1. Invasion and lodgment of the bacilli.	Weakened breathing; prolonged expiration, cog-wheel inspiration; slight relative dulness at the affected apex.	Possibly no symptoms beyond a slight persistent cough, weakness, anorexia, etc. Fever is sometimes present.
Second stage. { 2. Inflammatory changes around bacilli, resulting in the formation of grey tubercles. 3. Inflammatory zone around the tubercles.	Consolidation. { Diminished movement. Slight flattening. Dull note. Vocal fremitus, and Vocal resonance increased. Bronchial or tubular breathing.	Increased weakness and cough; elevation of temperature, especially in the evening; diffuse pain in chest.
Third stage. { 4. Commencement of breaking down. 5. Formation of cavities.	Consolidation, but attended with moist sounds.  Flattening is marked; movement much diminished; boxy note or cracked-pot sound; whispering pectoriloquy; amphoric or cavernous breathing, plus adventitious sounds (large bubbling râles with metallic resonance, etc.).	Increase in the severity of above symptoms; there are now great emaciation, night-sweats, oscillating temperature, hæmoptysis, distressing bouts of coughing, characteristic sputum, diarrhoea. Evidence of lardaceous disease in other organs, especially liver.

It must, however, be remembered that cases differ widely in detail. For instance, the disease may begin with very marked symptoms of bronchial catarrh; other cases again are so insidious in their onset, that the greater part of a lung may be consolidated before any severe symptoms develop. Profuse hæmoptysis may be the first symptom, or the disease may commence as a pleurisy with effusion. Fever may be present from the outset, or may be little marked until the stage of softening is advanced.

The physical signs also vary enormously. Those given above are the typical signs of damage to the elasticity of the lung, of consolidation, and of a cavity; but such typical signs are often absent, or masked by the bronchial affection; and the student who has a clear idea how the various sounds are produced will learn more by the careful examination of a few cases than by a course of unassisted reading.

We shall now consider the various symptoms in detail.

**Onset.**—The disease usually begins in the upper lobe, slightly below the apex of the lung, by the formation of tubercles in the peribronchial tissue, but gradually deposits of tubercles form



at lower levels, especially along the anterior margins. When the lower lobe is affected, the seat of invasion is near its apex, *i.e.*, about that part of the lung corresponding to the vertebral border of the scapula, when the hand of that side is hooked over the opposite shoulder, or at a point opposite the fifth dorsal spine. Owing to the peculiar manner in which the disease begins and progresses, it is possible in *one* case to get the signs of invasion of tubercle at the *base*, consolidation in the *middle*, and excavations at the *apex*.

Phthisis is at first unilateral, but when it is at all advanced the other apex is almost invariably affected.

**Course.**—The disease may be rapid or extremely chronic. Its course may be hastened by severe complications, bad treatment, unusually suitable soil, or putrefaction of cavity contents. It may, on the other hand, be arrested or hindered by—

1. Formation of fibrous tissue encapsulating the tubercles.
2. Caseation and calcification of the tubercles or tubercular debris, thus causing cavities to dry up (natural cure). Remember, however, that the disease, having become chronic, may take on a rapid form as a consequence of some inflammatory condition elsewhere.

**Special Symptoms.**—(1) *Hæmoptysis*.—The blood is bright and frothy in the early stage, but later it may be dark from stagnation or venous congestion. During the excavation stage small aneurysmal swellings on unsupported blood-vessels *may burst and cause speedy death*. Slight or even copious hæmoptysis may be the first symptom of phthisis.

(2) *Cough* is a constant symptom; at first slight, then gradually becoming hacking, paroxysmal, and painful.

(3) *Expectoration* at first is muco-purulent, but later becomes more purulent and copious. It is non-aerated, often blood-stained, and when expectorated into water spreads out into flattened circular discs like coins (*nummular sputum*). Often little grey hard pellets are present, with abundance of elastic tissue and bacilli. In the early stages, before the lung tissue has begun to break down, tubercle bacilli may be absent, or may be found only after repeated examination; in the later stages, besides the bacilli, pyogenic cocci are also present. A case of phthisis begins as a pure tuberculosis, but ends as a “mixed infection.”

(4) *Pyrexia*.—At first the temperature is elevated only in



the evening, but towards the end the temperature may oscillate continually between 100° and 104° F. The earlier fever is produced by the tuberculous toxins, but the later hectic disturbance results from sepsis due to the presence of pyogenic organisms in the broken-down pulmonary tissues, that is, to secondary infection. It is usually accompanied by the "hectic" flush, dilated pupils, and severe night-sweats. When night-sweats occur in the *earlier* stages, they are probably due to reflex vaso-motor disturbances *and not sepsis*.

(5) *Loss of Flesh*.—This is *generally most marked*, especially in the later stages, and where pyrexia is prominent. There is often extreme muscular irritability (myoidema).

(6) *Anæmia* is prominent from an early stage, and in young girls phthisis should be remembered as a cause of pallor almost as frequent as chlorosis.

(7) *General Appearance*.—The chest is frequently flattened, the antero-posterior diameter being short. The ribs present marked obliquity, so that the epigastric angle is acute. The angles of the scapulæ may stand out prominently from the ribs (alar chest). In the late stages there are marked clubbing of the finger-tips and curvature of the nails.

**Physical Signs**.—It has already been pointed out that the typical signs given in the table are often absent or masked by various conditions. It is often difficult to say whether a cavity exists or not, owing to—

1. Its small size.
2. Its deep situation, relatively healthy lung intervening between it and the surface.
3. Thickened pleura.
4. Compensatory emphysema.
5. Breath-sounds being drowned by extensive bronchitis.
6. Blocking of the tubes leading to the cavity.
7. Irregularity of its walls.
8. Too high or too low tension in its walls.

The "cracked-pot sound" is not absolutely diagnostic; it may be heard in crying children with healthy lungs, and in some cases of pneumonia. Alteration of the pitch of the percussion note on opening the mouth (Wintrich's sign) is sometimes present, and is of considerable value; so too is an alteration of pitch when the patient changes from the recumbent to the erect posture (Gerhardt's sign). But, as will be seen, there



are many difficulties in the recognition of a cavity, and a too exclusive reliance upon physical signs may lead to an erroneous diagnosis, as *post mortem* examinations prove.

**Complications.**—1. *Dry Pleurisy* is commonly present over the affected area, but it is doubtful whether it should be looked upon as a complication. As Gairdner said: "It prevents perforation of the pleura, or even obliterates the cavity between the two layers, thus preventing empyema, pneumothorax, etc. *It is a curious and beautifully conservative arrangement* that in most cases the pleuritic adhesions are often *in advance* of the actual deposit of tubercle near the surface, and still more in advance of its softening." Pleurisy with effusion is less common, but may even precede the pulmonary signs.

2. Pneumothorax, limited to the apex or general; and its sequela, pyopneumothorax. The complication is due to the sloughing of the portion of pleura overlying a superficial cavity, and the consequent escape of air and septic matters into the pleural sac.

3. Spreading of the tubercular disease to the *larynx*, meninges, peritoneum, etc.

4. *Diarrhœa*.—This is usually a late symptom. It may be due to mere intestinal catarrh, to tuberculous ulceration of the bowel, or to amyloid disease. The two latter conditions are very intractable.

5. Amyloid disease may also, in the late stages, attack the liver and spleen.

6. Tuberculous ulceration of the bowel may lead to *hæmorrhage*, or more rarely to *perforative peritonitis*.

7. Fistula in ano.

There are many other possible complications, such as nephritis, venous thrombosis, gangrene of the lung, etc., which may occasionally present themselves. The *mental attitude* of the phthisical patient is usually optimistic almost to the end, but some cases develop melancholia and suicidal tendencies.

**Fibroid Phthisis** is a somewhat unusual form, in which the interstitial tissues of the lung are more implicated than the parenchyma. The morbid changes, the symptoms, and the physical signs are almost identical with those of interstitial pneumonia, the main difference being that in fibroid phthisis the tubercle bacillus is present in the sputum.

**Diagnosis.**—The constant presence of tubercle bacilli in the



sputum is conclusive evidence of tuberculosis of the respiratory tract ; but there are many cases of early phthisis in which they are either absent or difficult to find. In such cases the following additional means of diagnosis are available :—

1. *The injection of old tuberculin* (T.O.A.) is followed by a febrile reaction in tuberculous subjects. 0·01 cc. is the initial dose ; if there is no reaction, 0·05 cc. should be injected three days afterwards, and at a similar interval 0·1 and 0·5 cc. If the reaction remains absent, the condition is not tuberculous. This method is not advisable in febrile cases, and should not in any case be used if the diagnosis can be made in other ways, as it is not entirely free from risk.

2. *The tuberculo-opsonic index* does not, in health, fall below 0·8 or rise above 1·2. Indices persistently above or below these levels point to tuberculosis.

3. *Calmette's reaction*.—A drop of a solution of the precipitate obtained by treating tuberculin with absolute alcohol being placed in the conjunctival sac, an inflammatory reaction follows within twenty-four hours in tuberculous cases. The reaction is sometimes very severe, and if the eye itself is diseased the result may be disastrous. The test is valuable, but not infallible.

4. *Von Pirquet's cutaneous reaction*.—A drop of tuberculin is placed upon the surgically clean skin, which is scratched through it as in vaccination. In the tuberculous an inflammatory reaction follows. A control inoculation through sterile saline solution is made at the same time. The only drawback to this method is that the reaction occurs both in healed and in active tuberculous lesions ; it is therefore most valuable in children.

5. *Moro's percutaneous reaction* is a modification of the above, the tuberculin being rubbed up with an equal bulk of lanolin to form an ointment, which is rubbed into the skin.

The *agglutination test* is of little practical value.

It should be added that even in the absence of bacilli or of a positive result from the tuberculin tests, the diagnosis is justified and should be made when at one apex slight dulness is associated with the presence of *persistent râles*. In such cases X-ray examination is often helpful.

**Treatment.**—1. For the *prevention* of infection, care in the disposal of the sputum is requisite. When in the house the patient should spit into paper or rags, which should be at once burned ; outside he should carry a pocket spittoon, the contents of which



must also be burned, the spittoon itself being sterilised by boiling water.

2. *Specific treatment*.—The position of tuberculin in the treatment of phthisis is now becoming more assured. It does nothing to combat the mixed infections of the later stages, nor can it replace tissues already destroyed. It is contra-indicated in pyrexial cases, but in the *incipient* stage its cautious use has led to valuable results. The initial dose, where the opsonic index cannot be estimated, must be very small (as low as 0.00001 mgr. of T.R., or 0.000005 mgr. of the more recent *bacillary emulsion*). Mixed infections may be benefited by the appropriate staphylococcal or streptococcal vaccine.

Serum treatment has not proved successful.

3. If specific treatment is employed, it must not supersede, but aid, the more ordinary measures. The most important of these is FRESH AIR. By day the consumptive should be, short of actual fatigue, as much as possible in the open, and at night the windows should be widely open top and bottom. Where there is fever he must keep to bed; but when possible the bed should be outside, and where that is not possible, the windows must remain open in presence of fever or any other acute symptom. In ordinary circumstances he should sleep alone. A stuffy bedroom with several people in it means rapid deterioration for the patient, and infection for the rest. *Sanatorium treatment*, though except in incipient cases it often fails to cure, produces marked improvement in the majority of cases which are not too far advanced. By its means the disease is reduced to a quiescent stage, and it trains the patient in the habits he must afterwards continue.

4. *Climatic treatment*.—Under this heading are included long sea voyages and change of residence. Sea voyages undoubtedly do good in many cases of early phthisis, the comparative sterility of the air contributing to the result; but no consumptive who is not a good sailor should be sent on such a voyage, nor any one who is unable to travel in comparative comfort, or who must travel alone. In the later stages sea voyages are contra-indicated. If change of climate is decided upon, the place selected should be sunny, and should give facilities for the open-air life. Either a dry cold climate may be chosen or a warm one, according to circumstances. In the earlier stages cold dry air is best, the climates of Davos, New Mexico, and Canada offering examples. High altitudes are, however, unsuitable for those with a tendency to hæmoptysis. In the later stages, and especially



where there is much emaciation, warm climates, like those of Madeira, Algiers, Torquay, or Bournemouth are more suitable. Remember that it is sheer cruelty to send a patient far advanced in phthisis away from home and friends, only too frequently to die among strangers.

5. *Adjuvants* to the open-air treatment are *exercise* and *dietetic treatment*. The consumptive should wear wool or flannel next the skin, but should not be overloaded with heavy clothes. Tepid baths, followed by brisk rubbing, are of benefit, and much good is done by carefully graduated exercise, which promotes a regulated auto-inoculation. The food must be nourishing and varied, and ample in quantity, systematic over-feeding indeed being advocated by many. Everything must be done to combat the very common anorexia and dyspepsia.

6. *Medicinal Treatment* is (a) general, and (b) symptomatic.

(a) *General*.—Creosote or guaiacol, cod-liver oil, maltine, and tonics, such as the hypophosphites and *arsenic*, are the principal remedies. Many newer forms of treatment—nascent iodine, emetine (for pulmonary hæmorrhage), etc.—have been introduced in recent years, but their value is not yet sufficiently tested.

(b) *Symptomatic*.—The following symptoms call for special treatment :—

(1) *The cough*.—As this is a persistent and constant feature of the disease, avoid rushing at once to cough mixtures. A common exciting cause of the nightly cough is the changing from a warm room to a cold bedroom ; or again, tickling of the fauces by the uvula. A useful combination is that of morphia, spirits of chloroform, and dilute hydrocyanic acid. For laryngeal and bronchial irritation, inhalations of tincture of benzoin or creosote are of much value.

(2) *The night-sweats*.—Picrotoxin, aromatic sulphuric acid, atropin, and oxide of zinc are the favourite remedies. Atropin gr.  $\frac{1}{100}$  to  $\frac{1}{80}$  (grm. 0·00065–0·0008) in pill at night, is the most reliable.

(3) *The diarrhœa* is usually best controlled by mineral astringents, in combination with opium.

(4) *Fever* should be treated by rest, fresh air, quinine, and cold sponging, or, if need be, the cold bath. Antipyrin, etc., may be occasionally used.

*Hæmoptysis* has already been considered.



In cases which fail to improve under ordinary treatment, and in which the disease is either limited to one lung or the opposite lung is only slightly affected, much benefit often results from the establishment of an *artificial pneumothorax*, nitrogen gas being introduced into the pleural cavity by means of a trocar. The object is to produce collapse of the lung, and so to obtain for it the absolute rest which the respiratory movements forbid. The gas is at first rapidly absorbed, and hence must be re-introduced at increasing intervals. Symptoms in favourable cases are rapidly improved, connective-tissue proliferation takes place in the affected lung, and the tuberculous lesions may become obsolete. The pneumothorax must be maintained for one or two years, after which it may be abolished by permitting absorption of the gas.

For the treatment of complications see Pneumothorax, Pleurisy, etc.

## GANGRENE AND ABSCESS OF THE LUNG

**Gangrene** may arise as a rare consequence of any of the inflammatory affections of the lung, from the putrefaction of the contents of a cavity, bronchiectatic or phthisical, and from a septic or more rarely a simple embolism of the pulmonary artery. It is very apt to follow aspiration pneumonia.

Gangrene may be either diffuse or localised. In the latter and commoner case the gangrenous area, usually in the lower lobe, is sharply marked off from the surrounding tissue, which is inflamed and œdematous. It is of a greenish-brown colour, and rapidly softens, forming a ragged cavity. The pleura may slough, when pyopneumothorax follows.

The *symptoms* occur in the course of other pulmonary diseases. Intense fœtor of the breath; a sputum which is also intensely fœtid, contains fragments of stinking lung tissue, and on standing separates into three layers; moderate fever, prostration, and rapid pulse, are the most noteworthy. There may be signs of cavity.

**Abscess** may also follow inflammation (most commonly aspiration pneumonia), operations on the throat, purulent affections of the throat or nose, and cancer of the œsophagus. Metastatic abscesses, which are not clinically recognisable, occur in pyæmia. Hydatid cysts or abscesses of the liver may perforate into the lung.

An aggravation of existing symptoms, a hectic type of fever,



the physical signs of a large cavity, and an offensive sputum containing *abundance of elastic tissue* are the chief characteristics.

In either of these states *treatment* consists mainly in anti-septic inhalations and support of the strength. With definite evidence of a cavity surgical measures may be attempted.

## TUMOURS OF THE LUNG

Carcinoma is the most common form, but endothelioma and sarcoma also occur.

Primary cancer of the lung is very rare. The secondary variety occurs in the form of either *dense*, hard, irregular nodules, or soft (medullary) masses, scattered throughout the lung tissue. The original sources are mostly—

1. Cancer of the bronchial glands, which spreads inwards at the root of the lung, or
2. Cancer of the liver, which invades the diaphragm and finally the pleura and lung.

**Symptoms** are insidious and varied. Pleuritic pain, cough, dyspnoea, and a blood-stained spit, which may be like red-currant jelly, are the chief. Pressure symptoms are also common. *Physical signs* are those of consolidation or of fluid effusion. Dulness due to consolidation may be combined with feeble breathing from pressure on the bronchus.

**Diagnosis.**—When there are symptoms of malignant mischief elsewhere, *marked pulmonary symptoms point to the invasion of the lung*. Pleuritic effusion may be so well simulated that puncture is sometimes required to clear up the diagnosis. The disease is usually fatal in about six months from the onset of symptoms.

## IV. DISEASES OF THE PLEURA

### PLEURISY AND EMPYEMA

Inflammation of the pleural membranes may be primary or secondary, acute or chronic, dry or accompanied by effusion.

**Etiology.**—1. The so-called primary or idiopathic pleurisy has been shown to be, in most instances, of tuberculous origin. Certain cases arise apparently from *chill*, but even in these, tuberculous affections of the lung may subsequently develop.



2. Secondary pleurisy may arise from numerous causes :—

- (a) Pneumonia or pneumococcal infection.
- (b) Various specific fevers (particularly *rheumatism* and scarlet fever) and septic diseases.
- (c) The final stages of chronic diseases (cirrhosis of the liver, Bright's disease, cancer, etc.).
- (d) Irritation from aneurysms of the aorta, or from tumours of the lung or mediastinal glands, etc.
- (e) Traumatism, and rupture of abscesses into the pleura.

Chill is a frequent determining cause.

The organisms usually found in pleurisy are the pneumococcus, streptococcus, and tubercle bacillus.

**Morbid Anatomy.**—The following stages may be distinguished—

1. *Hyperæmia*.—Loss of lustre ; the membrane is dry and red.
2. *Exudation* of fibrin, which coagulates, and gives a shaggy appearance to the membranes. In dry or plastic pleurisy the process goes no further. The fibrin-covered surfaces adhere, and the adhesions permanently obliterate the cavity, though in some cases complete resolution may occur. Dry pleurisy is usually localised.
3. *Fluid Effusion*.—The exudate is sero-fibrinous, and yellowish-green in colour, with floating flakes of lymph. The specific gravity is 1010 to 1020 ; the albuminous fluid coagulates on boiling ; and it also contains a large amount of fibrin. It may be hæmorrhagic in tuberculous or malignant pleurisy.
4. *Resolution*, with more or less extensive permanent *adhesions*. In some instances, instead of resolving, the effusion becomes purulent, constituting an empyema. Pneumococcal pleurisies are usually purulent from the first.

*Effects of the Effusion.*—If large, it produces grave symptoms by pressure on the lung tissue and viscera near it.

1. It causes collapse of a portion, or sometimes the whole of the lung. (The lung naturally *tends* to collapse, by virtue of its own elasticity.)
2. In pleurisy of the left side the heart is often displaced to the right.
3. In extreme cases the heart, great vessels, and mediastinum are pushed to the *opposite side*.



4. The thorax bulges *forward*, and the liver or spleen are displaced *downwards*. The intercostal spaces may be slightly distended, and the diaphragm much embarrassed.

*Absorption* occurs in many cases in from ten days to three weeks. If the fluid is unusually slow of absorption, suspect empyema.

**Symptoms.**—Pleurisy is ushered in by—

1. Slight or repeated chills ; rigor is uncommon.
2. Rapid ascent of temperature (which is not so high as in pneumonia).
3. Lancinating, tearing pain in the side, rendered worse by *any respiratory act*. The pain may be referred to the epigastric or umbilical region.

As the disease advances, and effusion takes place, the severe pain becomes replaced by dyspnoea. The patient lies on the affected side to give the sound lung more freedom. The respirations are hurried, the pulse quickened, and the cough is hacking in character ; the sputum is, however, slight in amount, mucoid in character, and *never rusty*, unless pneumonia exists as a complication.

**Physical Signs.**—1. *Stage of Fibrinous Exudation.*

- (1) Auscultation reveals the characteristic friction rub, synchronous with the respiratory movements, usually leathery-creaking in character, but sometimes musical, like the sound produced by rubbing a pane of glass with a moist finger.
- (2) Palpation may yield friction fremitus.

2. *Stage of Effusion.*

A. Over the *affected area*—

- (1) Absence of breath sounds.
- (2) Absence of vocal resonance and fremitus.
- (3) Absolute dulness on percussion, and board-like resistance.
- (4) Diminished movement, and bulging of the lower intercostal spaces.

The upper boundary of the area of dulness is horizontal, falling, however, about an inch from the angle of the ribs to the spine. As the complementary pleural sinus extends beyond the lung, the area of dulness also extends beyond the normal limits



of the lung. On the left side it invades the crescentic space of Traube, and this forms a useful distinction between pleurisy and pneumonia.

*B. Just above the line of effusion.*

Here there is a small area where the voice is transmitted through (1) slightly *condensed* lung, and (2) a *thin* layer of effusion. This gives rise to a peculiar modification of the voice, termed *œgophony*—compared by some writers to the bleating of a goat.

*C. Over the lung above the effusion.*

The signs will depend on the amount of pressure. Usually, we get on—

*Inspection.*—Diminished movement.

*Palpation.*—Vocal fremitus increased. Vocal resonance increased to the extent of bronchophony.

*Percussion.*—“Skodaic resonance.” On heavy percussion, we get almost a “cracked-pot” sound.

If the amount of fluid is only moderate, the breath sounds may not be entirely absent, but they are always weakened. In such cases there may be a slightly tubular quality, due to collapse of the lung tissue around the *patent* tubes.

*Grocco's sign* is the presence of a triangular area of dullness close to the spine, and at the base of the lung *on the opposite side*. The apex of the triangle points upwards, and its base may be from 2 to 5 cm. wide. It is due to displacement of the mediastinum by the fluid.

*Signs of displacement of the organs* may also be found :—

1. Altered position of the apex beat, and displacement of the cardiac dullness to right or left according as the pleurisy is left- or right-sided.
2. Ensiform cartilage pushed aside.
3. Liver pushed downwards.

**Course.**—The fever subsides by *lysis*, sometimes within a week, sometimes not for three weeks. Absorption of the fluid is slow, and as it disappears, friction may once more be heard. The prognosis is favourable in uncomplicated pleurisy, but where there is extreme effusion, *sudden death* from syncope may take place as the result of pressure on the heart. In a certain pro-



portion of cases the fluid becomes purulent (empyema), and this may be indicated by such signs as—

1. Persistence of fever, which becomes hectic in type.
2. Rigors.
3. Night-sweats.
4. Leucocytosis.

But the only certain indication is gained from exploratory puncture.

**Diagnosis.**—The examination of the cellular contents of the fluid (*cytodiagnosis*) may help to determine the character of the pleurisy. In tuberculous pleurisies these cells are largely lymphocytes, while polymorphonuclear cells prevail in simple inflammations. For the diagnosis from pneumonia, see the table on p. 374, and from hydrothorax, see p. 378.

**Empyema.**—The main points of note in connection with purulent pleurisy are the following :—

1. Whilst an empyema is frequently due to the *pleuritic effusion* becoming purulent, it may be *primary*—i.e. purulent from the beginning.

2. When pleurisy is a *sequela* or *complication of the infective fevers or pyæmic state*, it nearly always becomes purulent.

3. If pleurisy is a result of pneumonia, it is usually *purulent from the start*.

4. In old empyema the ribs undergo the following changes :—

- (1) A crowding together of the lower ribs.
- (2) Absorption of a portion of rib (atrophy), with a deposit of new bone (hypertrophy), giving the rib on section a  $\Delta$  appearance—i.e. atrophy at the apex and hypertrophy at the base.

5. *Leucocytosis* is constant in empyema, and there are usually symptoms of sepsis.

6. The prognosis is much more unfavourable.

7. In empyema, untreated by the surgeon, *pus may burrow anywhere*. It usually ruptures into the lung, but sometimes it may open externally. In a few cases natural cure by absorption takes place.

**Hæmorrhagic Pleurisy.**—In pleurisy due to malignant disease, tuberculosis, or cachectic states, the exudate may be hæmorrhagic.



## Diagnostic Table

(MODIFIED FROM F. ROBERTS)

	BRONCHITIS.	CROUPOUS PNEUMONIA.	BRONCHOPNEUMONIA.	PLEURISY.	ACUTE PHTHISIS.
1. Mode of invasion.	Coryza, and other symptoms of "cold." No marked rigors, but only slight and repeated chills, if any.	One or more severe rigors. Often vomiting.	Generally after bronchitis, or collapse, and without distinct rigors.	Frequently none, but sometimes several, not severe rigors.	Follows pneumonia, bronchitis or bronchopneumonia, or begins with severe rigors, often repeated.
2. Sensations about the chest.	Soreness, heat, etc., behind the sternum. Muscular pains from cough.	Pain in the side at first, not stitch-like, but more dull and diffused.	Pains about the chest, but not specially localised.	Severe, stitch-like pain in side, increased on respiratory movements.	Generally pains in various parts of the chest.
3. Cough.	In paroxysms, often severe.	Hacking, or in paroxysms.	Short, hacking, and painful.	Slight, and patient tries to repress it.	Frequent and violent fits.
4. Expectoration.	Abundant, mucous, mucopurulent, etc., changing its characters as the case progresses.	Considerable; viscid, tenacious, "rusty."	Often less than before; not "rusty."	Absent, or very slight, and of no special characters.	Abundant, perhaps purulent, or nummulated; often streaked with blood.
5. Disturbance of breathing.	Sense of dyspnoea, in proportion to the extent of the disease; may be extreme. Pulse-respiration ratio not proportionately altered.	Very rapid breathing, and much perversion of pulse-respiration ratio, but not proportionate feeling of dyspnoea until the later stages, when dyspnoea is marked.	Rapidity of breathing increased when it occurs in bronchitis; dyspnoea may be marked.	Quick, shallow breathing at first, but less disturbance of pulse-respiration ratio than in pneumonia. Later on, more or less actual dyspnoea according to amount of pressure.	Great dyspnoea, and very hurried breathing.



6. Degree of pyrexia.	Often absent or slight, and temperature rarely above 100° to 102° F. Skin moist.	Considerable; temperature usually high, 103°, 104°, 105° F., or more, and runs a regular course. Skin peculiarly hot and dry.	Temperature high, but there are considerable remissions, at irregular intervals.	No regularity in course of temperature. Skin not acridly hot.	Often very high, but no regularity in temperature, <i>i.e.</i> oscillates, but usually higher in evening.
7. Aspect of patient, and general condition.	Tendency to cyanosis, if the disease be extensive. In some cases adynamic symptoms.	Marked flushing of face, often unilateral. <i>Malara</i> cyanosis. Herpes round mouth.	Face is flushed. Often much anxiety and restlessness, with loss of flesh and strength.	Nothing special. No particular prostration, or tendency to cyanosis unless dyspnoea becomes marked.	Severe prostration and weakness, with profuse night-sweats.
8. Physical signs.	Various râles and bronchial fremitus. Signs of obstruction of bronchial tubes. More or less bilateral.	At first, fine crepitations, followed by signs of consolidation, <i>viz.</i> , diminished movement, increased vocal fremitus, dulness, bronchial or tubular breathing, increased vocal resonance, etc. Usually one base is affected. The side is not notably enlarged, nor is there displacement of organs.	There may be signs of consolidation in scattered spots, with râles. Both lungs are usually involved in irregularly scattered patches.	At first friction-sound or fremitus, succeeded by signs of fluid, <i>viz.</i> , side often enlarged, movements interfered with, diminished vocal fremitus, dulness occasionally movable, weak or suppressed breathing. Usually on one side, and often displacement of organs.	At first merely signs of bronchitis, followed by consolidation, softening, or excavations in different parts. There is frequently nothing but scattered râles in very acute cases.
9. Course and termination.	Variable. No crisis. Tendency to death by apnoea or adynamia in the capillary variety.	(1) Often a marked crisis from fifth to eighth day; (2) death; (3) gangrene of lung; (4) abscess of lung.	No crisis, and course often prolonged or followed by acute phthisis.	No crisis, and course very variable.	Generally very rapid course, and fatal termination.



**Treatment.**—In *dry pleurisy* relieve pain by a blister or if need be by morphia. In *pleurisy with effusion*, fever diet and a free purge are necessary at the outset. Pain may be relieved by morphia or Dover's powder, leeches, or strapping the affected side to restrict its movements. If the exudate is only moderate in amount, an attempt may be made to promote absorption by the daily administration of a concentrated solution of magnesium sulphate (Matthew Hay), of which ℥iv to ℥vi (grm. 16·0 to 24·0) may be given before breakfast in ℥ij (cc. 60) of hot water. The free purgation induced may, however, prove exhausting to weakly patients, in whom alkaline diuretics may be substituted with advantage. Externally, liniment of iodine is often used, but inunction of mercurial ointment is more effectual. During convalescence, nourishing diet and tonics, such as arsenic or the hypophosphites, should be employed, and respiratory exercises should be begun early to aid expansion of the lung.

If the fluid is not rapidly absorbed (within two or at most three weeks), it should be removed by aspiration, which may be repeated on re-accumulation. The longer aspiration is delayed the less likely is a satisfactory expansion of the lung. Aspirate *at once* if there are signs of serious pressure on the heart or interference with breathing, and always if the dulness reaches the level of the second rib in front.

The treatment of *empyema* is surgical, and consists in free drainage. After the operation the appropriate bacterial vaccine may promote healing.

## PNEUMOTHORAX

Pneumothorax means the presence of air in the pleural cavity.

**Etiology.**—1. Traumatisms, such as punctured wounds, laceration from the end of a broken rib.

2. Causes from the lung side—

- (1) *Breaking down of a subpleural tuberculous focus*, leading to undermining and perforation of the pleura.
- (2) Gangrene or abscess of the lung.
- (3) Rupture of an empyema into a bronchus.
- (4) Rupture of emphysematous air vesicles.

The opening may be small or large, and may remain patent or rapidly heal up. In the latter case the air is absorbed.

**Symptoms.**—The symptoms of pneumothorax depend largely



upon the manner in which it was produced, and the quantity of air present. They may be summed up as follows :—

1. *Sudden* pain at the time of rupture, with or without collapse.
2. Great dyspnœa.
3. Quick and small pulse.
4. Shallow and rapid breathing.

In many cases of advanced tuberculosis, the symptoms, owing to limiting adhesions, or to extensive disease of the lung, are not very urgent, and dyspnœa is not marked.

**Physical Signs.**—*Inspection.*—Bulging may be local or general on the affected side.

*Percussion* usually yields a hyper-resonant or tympanitic note. If the valve-like opening be patent, a typical cracked-pot sound can be elicited.

*Palpation.*—Absence of vocal fremitus.

*Auscultation.*—Absence of breath-sounds. On coughing, the sounds produced are peculiarly amphoric and have a metallic ring. If the chest be struck with two coins whilst listening with the stethoscope, the characteristic bell or anvil sound is heard.

In a left-sided pneumothorax the heart may be displaced to the right, or the cardiac dulness may even be obliterated, the complementary pleural sinus being distended by air and intervening between the heart and the chest wall.

The admission of air into the pleural cavity implies the admission of organisms. Inflammatory changes are thus set up, and an effusion, either sero-fibrinous (*hydropneumothorax*) or purulent (*pyopneumothorax*), is the consequence.

**Hydropneumothorax.**—A condition where air is present above and fluid below in the pleural cavity. The physical signs are, in the upper part, those already described as diagnostic of pneumothorax, plus *hippocratic succussion* (the term applied to the splashing sound heard on shaking the patient), and below, those diagnostic of fluid already discussed under pleurisy.

**Pyopneumothorax** means the presence of air and pus in the pleural cavity. The physical signs are the same as those of hydropneumothorax.

**Treatment.**—Subdue the pain by a hypodermic injection of morphia, or by hot poultices. Meet the shock by diffusible



stimulants, etc. Paracentesis is sometimes of value, if the air is confined in the pleura under high pressure. Pyopneumothorax may be treated by incision if the lung is only slightly affected. Where there is advanced pulmonary disease, incision is not indicated.

## HYDROTHORAX

Hydrothorax, or dropsy of the pleuræ, is a term used to denote the effusion of fluid into the pleural cavity as the result of a passive exudation. It is generally due to Bright's disease, but may be of cardiac origin, and sometimes occurs in the grave anæmias.

The main points of difference between it and inflammatory effusion (pleurisy) are—

1. It is not preceded by acute symptoms of inflammation, therefore *friction is absent*.
2. It is usually bilateral.
3. The fluid gravitates with movements of the patient to a far greater extent (like ascites).
4. It is associated with signs of dropsy elsewhere.
5. Pain is absent, but dyspnœa may be greater than in pleurisy.
6. Aspiration is not likely to do more than *temporarily relieve pressure*.

## V. DISEASES OF THE MEDIASTINUM

### MEDIASTINAL TUMOURS

These not very common growths are most frequent in the male, between the ages of thirty and forty. Sarcoma and carcinoma both occur, but the latter is always secondary. Sarcomata or lymphosarcomata originate in the lymphatic glands, connective tissue, or remains of the thymus. The anterior mediastinum is the most common seat.

**Symptoms** are mainly those of pressure, the onset being insidious. There may be dyspnœa from pressure on the trachea or bronchi, cyanosis and unilateral or bilateral *œdema* of the head and arms from pressure on the large veins, distension of the external jugulars and superficial veins of the chest, pressure on the recurrent laryngeal (paralysis of the left cord), sympathetic (unequal pupils), or vagus (rapid pulse). Brassy cough is frequent, and there may be some pain in the chest,



irregular fever, and emaciation. Dulness over the manubrium is the chief physical sign, but pleural effusion may appear at any time, the heart may be displaced, and the cervical glands may be enlarged.

The *diagnosis* from aneurysm is often difficult, but there are no abnormal auscultatory phenomena, pulsation is absent, and pain is less. A case in which the symptoms have lasted over a year is unlikely to be a tumour.

The outlook is hopeless, and relief of symptoms is all that can be attempted.



# DISEASES OF THE KIDNEYS

## EXAMINATION OF THE URINE

THE examination of the urine in disease is of the utmost importance. Whenever possible, a specimen *of the urine passed in the twenty-four hours should be used*, as the urine is richer in certain constituents at certain parts of the day. Observe—

1. *Quantity*.—Normal 45 to 52 ounces (1300 cc. to 1500 cc.).
2. *Specific Gravity*.—Normal, 1015 to 1025. If above 1025, test for sugar. The total amount of solids per 1000 parts may be roughly estimated by multiplying the last two figures of the sp. gr. by 2·33.
3. *Odour*.—The normal odour is peculiar and characteristic. It becomes ammoniacal and putrid when the urine decomposes. It is honey-like when sugar is present, suggestive of chloroform in the presence of acetone, and like that of violets after the administration of turpentine.
4. *Reaction* is normally acid, except after meals, when it may be temporarily neutral or alkaline from the salts of the food. The acidity may increase for a few hours after the urine is passed; later, the reaction becomes alkaline from ammoniacal decomposition.
5. *Colour*.—Normal, pale straw to dark amber.
  - (1) If red, or reddish-brown, or smoky, suspect the presence of blood, and search for blood corpuscles; test with guaiacum and ozonic ether, and, if necessary, examine with the spectroscope.
  - (2) If greenish or yellowish-brown, suspect the presence of bile pigment; observe the colour of the froth; test for bile pigment and bile acids.
  - (3) If pale, in large quantity, and of a sp. gr. over 1025, test for sugar.

The colour may be yellow or orange from santonin or rhubarb, and green or black from carbolic acid.



6. *Deposit*.—Allow the urine to settle in a cylindrical or conical glass, and a cloud of mucus invariably forms. It is always light, and moves easily with the fluid.

A deposit of urates is common in concentrated acid urines. It is usually of a brick-dust colour, and moves easily when the vessel is inclined.

A deposit of earthy phosphates, in neutral or alkaline urines, has usually a white or dirty-white appearance, and is somewhat heavy.

Oxalates form a white dust on the surface of other sediments (powdered-wig deposit).

If the deposit is dense and slimy, it probably contains much pus.

If the urine is milky on passing, it is usually due to deposit of the earthy phosphates. This condition is common after a full meal, especially when vegetables have been freely partaken of; the urine in such a case is usually neutral or alkaline. Do not mistake this deposit for pus, nor confound *earthy* phosphates with the crystalline triple phosphates, or with urates. Urates are usually brick-red, redissolve on heating or on the addition of alkali, and are insoluble in acids; earthy phosphates are white or milky, do not dissolve on heating or on the addition of alkali, and are soluble in acids.

**Urea**.—The quantitative test for urea is of great importance; for by comparing the amount of nitrogenous material taken in with the amount of urea passed, we are enabled to gauge the state of metabolism in the body. Urea is increased during active metabolism, as in fevers, and also in diabetes mellitus and insipidus, and in chronic gout. It is decreased upon a scantily nitrogenous or non-nitrogenous diet, in all conditions which lower the metabolic functions, in Bright's disease, especially its chronic forms, in degenerative affections of the liver, in persistent vomiting or diarrhoea, and the later stages of acute febrile diseases. It must not be forgotten, however, that the formation of urea may be for a time excessive, and yet the excess may not appear in the urine through inability of the kidneys to excrete it.

*Volumetric Estimation of Urea* by the Hypobromite process depends on the fact that the solution of sodium hypobromite decomposes urea into  $\text{CO}_2$ , N, and  $\text{H}_2\text{O}$ . The  $\text{CO}_2$  combines with the free soda, and nitrogen, therefore, is the only gas given off. One gramme of urea by this method yields 371 cc. of nitrogen, therefore 37.1 nitrogen equals 1 decigramme of urea.



If we measure the amount of N given off from a known quantity of urine, we can then calculate the total amount of urea passed daily.

The *ureameter of Doremus* is a U tube of which the longer limb is graduated, while a bulb is blown on the shorter limb. The longer limb and half the bulb being filled with hypobromite solution, 1 cc. of urine, freed if need be from albumin by boiling and filtration, is drawn into a graduated pipette capped by a rubber teat. The curved lower end of the pipette is then passed through the hypobromite in the bulb, until its point is directly under the vertically placed longer limb of the tube. The urine is then slowly expelled, and the nitrogen given off rises to the upper end of the graduated arm. After the tube has stood for half an hour, the amount of urea corresponding to the nitrogen is read off upon the scale.

*Hinds's modification* of this instrument dispenses with the pipette, the urine being allowed to flow through a stopcock directly into the long arm of the tube. Loss of nitrogen is thereby avoided, and the estimation is quite sufficiently accurate for clinical purposes.

On an ordinary mixed diet, with an average amount of exercise, the daily excretion of urea for a man in good health varies between 25 and 40 grams, the average amount being about 33 grams (500 grains). This corresponds with a proteid intake of about 120 grams. But a much lower excretion with a lower proteid intake is perfectly consistent with good health, and, as has been seen, the daily excretion is much diminished in vegetarians.

**Uric Acid** appears in the urine to the extent of 7 to 12 grains (grm. 0.5–0.8) daily, combined with soda, etc., as urates. It is derived from the nucleins of the food, and in part from those set free in the body by the metabolism of the cells. It is much increased by an abundant meat diet, in acute febrile diseases, in conditions embarrassing the respiration or circulation, in diseases of the liver or spleen, and in gout after the paroxysm. In order to test for its presence, we first displace it from its base by adding an acid, HCl, or acetic acid, and letting the urine stand awhile. When uric acid is deposited it forms a cayenne-pepper or red-sand-like deposit.

*Tests for Uric Acid.*—1. *Garrod's Thread Test.*—Concentrate the urine, place 5 cc. in a watch-glass, and add ten drops of



glacial acetic acid. Place a thread in the fluid, and leave it in a cool place for twenty-four hours to allow crystals to form.

2. *Murexide Test*.—Place five drops of urine in a porcelain capsule, and add one drop of nitric acid. Evaporate *gently* nearly to dryness. Add a small drop of ammonia; a purple colour, due to murexide or purpurate of ammonia, develops.

3. *The Volumetric Test*.—The acid is estimated by Haycraft's method. Uric acid is made to combine with silver, forming a gelatinous precipitate; this is separated by filtration and made into a solution with nitric acid. The amount of silver is then tested by a colour test, and the amount of uric acid calculated by the amount of silver found.

**Chlorides**.—The amount of *chlorine* excreted daily amounts to about 100 grains, chiefly in combination with soda.

*Test*.—Silver nitrate preceded by acidulation with nitric acid.

The chlorides are markedly diminished in all cases of croupous or fibrinous inflammations; this is particularly the case in croupous pneumonia.

*Quantitative Estimation*.—

*Silver Method*.—The solutions required are—

1. Solution of *silver nitrate*, containing 29.075 grms. of the fused salt in 1000 cc. of distilled water; of this solution 1 cc. = 0.01 gm. of sodium chloride.
2. Saturated solution of *potassium chromate* (neutral).

**METHOD**.—Take 10 cc. of the urine, and dilute with 100 cc. of distilled water. Add to it a few drops of solution (2). To this mixture in a beaker allow the standard solution (1) to drop in from a burette. A precipitate of silver chloride will occur as long as any chlorine is uncombined. When the whole of the chlorine is satisfied, a reddish or pink (since there is much white precipitate present) precipitate of silver chromate appears. This indicates the time to stop the addition of the silver nitrate, and the amount of the solution which has been used is read off. This will indicate the amount of silver nitrate necessary to convert all the chlorine present in 10 cc. into silver chloride. It is known that 1 cc. of the solution = 0.01 gm. of salt, and from this the total amount of chloride present can be estimated.

**Phosphates**.—The test for phosphates generally have already been considered. The test for orthophosphoric acid is precipi-



tation by uranium acetate, the precipitate being insoluble in acetic acid.

**Albumin.**—The tests for albumins depend on their coagulability by heat and precipitation by nitric acid, picric acid, etc. The most reliable tests are—

1. *Heller's Test.*—Put a little nitric acid in a test-tube, pour the albuminous urine cautiously upon it, and an opalescent ring of coagulated albumin appears at the junction of the two fluids. If the albumin is abundant it appears quickly; if there is only a trace of albumin its appearance may be delayed for two or three minutes.

But nitric acid in the cold also precipitates *albumose*; this is distinguished from albumin by the precipitate dissolving when heated and returning when cooled. Resins are also precipitated by nitric acid.

In using Heller's test, remember that the addition of nitric acid to undiluted urine may cause a precipitate of *uric acid*; this, however, is always very scanty, and the microscope shows crystals. Further, if the urine be very concentrated the addition of nitric acid may cause a precipitate of nitrate of urea, but its appearance is very different from that of the flocculent precipitate of coagulated albumin. Nitrate of urea is not thrown down when the urine is diluted before adding the acid.

2. Boil the urine in a test-tube. A flaky precipitate falls, which may be albumin, or if the urine is only faintly acid, phosphates. Phosphates are redissolved on acidulating with acetic acid, albumin is not.

Picric acid and salicyl-sulphonic acid are also used as tests. Picric acid precipitates, besides albumin, albumose, uric acid, creatinin, and alkaloids; salicyl-sulphonic acid precipitates only albumin and albumose.

3. *The quantitative test* may be roughly performed by Esbach's albuminimeter; but since picric acid is the reagent used, it should be remembered that *all the proteids* in the urine, other than albumin, are thereby precipitated.

**METHOD.**—*Esbach's Albuminimeter* is a graduated tube for roughly estimating the percentage of albumin in urine. It cannot be successfully used if the percentage is large, and it succeeds best where the urine is diluted until its sp. gr. is not above 1010. If necessary, therefore, an equal volume or two volumes of water may require to be added to the urine, and this must be taken into account in making the calculation,



1. Fill the tube with urine up to the mark *U*.
2. Add picric and citric acid solution to mark *R*.
3. Mix thoroughly, and set aside for twenty-four hours.
4. Read the height of the coagulum on the scale. The numbers on the scale indicate grammes of albumin per litre of urine. The result is only approximative, because the bulk of the coagulum depends much upon its density.

The most accurate method of determining the exact quantity of albumin is Brandberg's modification of Sir W. Roberts's method; the process is a tedious one, and requires much practice to be accurate.

**Serum-globulin**, precipitated like serum-albumin by heat, nitric acid, and picric acid, is recognised by its precipitation when the urine is saturated with magnesium sulphate after neutralisation.

**Albumoses** are found chiefly in suppurative processes, in acute infections, and in acute inflammations, but they also occur in some cases of chronic Bright's disease. They are precipitated by nitric acid and salicyl-sulphonic acid, but redissolve on heating. They give the biuret reaction, the test being thus performed: Precipitate all albumin by heat and acetic acid, filter, and float the filtrate on the surface of the reagent, which consists of a 20 per cent. solution of caustic potash in distilled water to which a trace of sulphate of copper has been added. A rose-pink colour appears at the junction of the fluids.

A peculiar albumose is found in *Bence-Jones albumosuria*, a condition associated with certain diseases of the bone-marrow. It is coagulated by a temperature of 58° C., and redissolved on boiling. Strong hydrochloric acid in the cold produces an abundant precipitate, also redissolved on boiling.

**Sugar (Dextrose).**—The various tests used are those of Fehling, Trommer, Johnson, the phenyl-hydrazin test, the fermentation test, and the test by means of the polarising saccharometer.

Fehling's solution is an alkaline solution of potassio-tartrate of copper, and is made of such a standard strength that 1 cc. of *recently* prepared Fehling's solution is reduced by 5 milligrammes of grape sugar. It may be used to demonstrate the presence of a reducing sugar, or for the quantitative estimation; but in the latter case it is better to use the Pavy-Fehling solution, which contains ammonia in addition to the other salts. The



ammonia prevents the precipitation of cuprous oxide, and thus the complete reduction is indicated at once by the disappearance of the blue colour of the reagent.

**METHOD.**—Having removed albumin (if present) from a sample of the urine passed in the twenty-four hours—

1. Dilute the urine to 1 in 20 (5 cc. urine—95 cc. water).
2. Place the urine in a burette.
3. Measure 10 cc. Pavy-Fehling solution, and place in a porcelain basin or small flask.
4. Heat to boiling, and while boiling drop in urine until the blue colour just disappears. If the urine be dropped in too slowly, the ammonia may be driven off by the prolonged heat, and a precipitate of cuprous oxide will fall, confusing the reaction.
5. Calculate the total amount of sugar in the urine passed in twenty-four hours.

In using Fehling's solution for the qualitative test, add the urine drop by drop to the boiling reagent. If much sugar is present, a yellow or red precipitate of cuprous oxide appears at once; if little, only after some time; if very little, there may be only a greenish discolouration of the fluid. Certain other constituents of the urine (uric acid, creatinin, glycuronic acid) and certain drugs (chloral, chloroform, benzoic acid, salicylates) also reduce cupric oxide, and may give the reaction if large quantities of urine are used. More urine than reagent should never be employed, and if the sp. gr. of the urine is above 1020, it is better to use not more than half as much urine as reagent. Prolonged boiling should be avoided. Albumin should be removed by heat and filtration before the test is performed.

*Phenyl-hydrazin Test.*—If Fehling's test gives doubtful results, place in a test-tube half an inch of crystals of acetate of soda and the same quantity of phenyl-hydrazin hydrochlorate, and fill the tube half full of the suspected urine. Boil for two minutes, and allow to stand for half an hour. Then remove with a pipette a little of the yellowish sediment, and examine it under the microscope. Yellow acicular crystals of phenyl-glucosazone, arranged in sheaves, will be seen if sugar is present.

*Fermentation Test.*—Boil the urine to drive off air, and acidulate it if necessary with tartaric acid. Shake up the urine in a test-tube with a piece of German yeast the size of a pea, place the resulting emulsion in a fermentation tube, and set



aside in a warm place for twenty-four hours. The liberated carbon dioxide accumulates at the top of the tube. A control experiment should be made with yeast and water.

**Acetone.**—Add to the urine a few drops of a fresh saturated solution of sodium nitro-prusside and then a few drops of NaHO. A dark-red colour, due to creatinin, appears. Acidulate with strong acetic acid, and the red changes to crimson in the presence of acetone, but disappears if acetone is absent. Or, add to one inch of the urine in a test-tube five drops of 10 per cent. KHO solution; heat gently, and slowly add a saturated solution of iodine in potassium iodide till the urine becomes yellowish-brown; then add a little more KHO. A yellow turbidity results; crystals of iodoform are precipitated, and can be recognised by the characteristic smell.

**Diacetic Acid.**—The addition of the tincture or solution of ferric chloride produces a deep Bordeaux-red colour, not given by acetone.

**Bile.**—*Bile pigments* give from a deep orange or greenish to almost a porter-like hue to the urine, which froths easily, *the froth being tinged with the pigment*.

**TESTS.**—1. Add impure nitric acid (nitric-nitrous) cautiously to the urine in a test-tube or on a porcelain slab; at the junction of urine and acid a display of colours occurs, due to oxidation of the pigments. The characteristic colour is green. (Gmelin's test.)

2. Pour on to the surface of the urine in a test-tube a dilute solution of tincture of iodine (1 in 10). A green ring forms at the point of contact. (Maréchal's test.)

*Bile Acids* cannot be certainly detected in bilious urine without evaporating, or unless the amount of bile is excessive.

**TESTS.**—1. Sprinkle a pinch of flowers of sulphur on the surface of the urine; in the presence of bile acids the sulphur sinks, while in a normal urine it floats (Hay's test). The phenomenon is due to an alteration in the surface tension of the fluid.

2. Add a solution of cane sugar to the urine in a test-tube, shake, let a drop of  $\text{H}_2\text{SO}_4$  trickle down the tube; at the junction of the acid and froth a cherry rose-red colour will develop. (Pettenkofer's test.) The test is very fallacious.

**Blood.**—(See p. 390.)



- Pus.**—1. Examine microscopically for leucocytes.  
2. KOH added to the urine renders it stringy.  
3. Pour off the supernatant urine, and add tincture of guaiacum to the slimy sediment. A blue colour develops at the point of junction.  
4. Ozonic ether added to the urine effervesces in the presence of pus.

The chemical tests are entirely subsidiary to the microscopic recognition of the leucocytes.

**Casts** of the renal tubules are found most commonly, though not exclusively, in connection with inflammatory affections of the kidney. They probably consist of the coagulable elements of the blood, which have reached the tubules through lesions of these structures. Seen under the microscope, such a cast reproduces the outline of the lumen of the tubule, and is a structureless, non-refractile, transparent body, more easily recognisable when lightly stained with gentian violet (*hyaline* cast). But in the presence of inflammatory changes, the casts may carry on their surface any detached epithelium or blood corpuscles lying in the lumen of the tubule where they originate, and such epithelium may be either freshly shed, or degenerated in various ways. In such conditions, we may therefore find *blood casts* and *epithelial, fatty, or granular casts*. Of these, epithelial and blood casts are usually predominant in the acute inflammations, granular casts in chronic conditions, and fatty casts in "large white kidney." *Waxy* casts, structureless like the hyaline cast, but highly refractile, may also be found in chronic renal affections.

**Crystalline Deposits.**—The commonest of these are uric acid, triple phosphates, and oxalates. *Uric acid* crystallises in rhombs or prisms, which are of a red or reddish-brown colour, and may be found singly or arranged in sheaves or rosettes. *Triple phosphates* (ammoniac-magnesian phosphate), found in decomposing urine, form large colourless refractile crystals, of the familiar "knife-rest" or "coffin-lid" shape. *Oxalates* form small octahedral or dumb-bell-shaped crystals, colourless and highly refractile. When they are present in quantity, they settle as a light powdery deposit ("powdered-wig" deposit) on the surface of any other deposit which may have fallen to the bottom of the urine-glass.

Other crystals, which are occasionally found, but do not require description here, are those of crystalline urates, stellar phosphate of lime, cystin, leucin, tyrosin, and cholesterin.



## ALBUMINURIA

By albuminuria is meant the presence of serum-albumin and serum-globulin in the urine. As albumin is one of the essential constituents of the blood, an escape of it in the urine must necessarily be looked upon with suspicion, if not with anxiety. Formerly, all cases of renal albuminuria were regarded as varieties of Bright's disease, but it is now well known that albuminuria is sometimes present in individuals who are apparently in perfect health (functional or cyclic albuminuria). However, *a persistent albuminuria must always be looked upon as a grave and significant condition*. In other words, just as palpitation of the heart may exist without cardiac disease, but may on the other hand be a prominent symptom of a grave organic lesion; so with albuminuria, it may or may not be a serious omen.

Albuminuria may be either of renal origin, or dependent upon some lesion of the urinary tract below the kidney. The cause of renal or essential albuminuria is to be found mainly in the glomeruli. The epithelial cells of the glomerular tuft exert a selective influence in excretion, inasmuch as they allow the water and certain soluble salts to pass, but prevent albumin from so doing. It has also been maintained that albumin *normally* escapes through the glomeruli into the tubules, but is rapidly taken up again by the cells of the tubules, and *reabsorbed into the blood*. This is, however, very doubtful, while there is proof that the albumin escapes through the diseased glomeruli. It is, however, possible that in severe inflammations of the kidney, in which the tubular epithelium is largely shed, lymph may exude through the denuded basement membrane into the interior of the tubules, and in this way the amount of albumin may be increased. In milder inflammations the albumin is probably derived from the glomeruli alone.

*Experimentally*, we may produce albuminuria by—

1. Pressure upon (not closure of) the renal veins; the pressure in the glomeruli is increased thereby.

2. Closure of the renal artery, and subsequent re-establishment of the circulation; this interferes with the nutrition of the renal cells.

3. Ligature of the aorta *below one* kidney, and extirpation of the other.

4. Ligature of the aorta above the renal arteries.

5. Compression of the trachea; this leads to asphyxia, and consequent rise of blood-pressure (Halliburton).



*Clinically*, we get albuminuria—

1. Due to—

Morbid conditions of the kidney, such as acute and chronic inflammation, congestion, active or passive, amyloid degeneration, renal calculi, tumours, etc.

Diseases of the urinary apparatus below the kidney.

Hæmic changes—anæmia, leucocythæmia, etc.

Certain fevers, especially scarlet fever and diphtheria.

Pregnancy.

Certain poisons—cantharides, turpentine, arsenic, phosphorus, excessive use of morphia, etc.

2. After certain diets, especially in those who pass much oxalates in their urine (oxaluria).

3. In a *remittent or cyclic form*, occurring in apparently healthy people; albumin is in such cases only present in the urine at certain periods of the day, as after rising, after meals, or following severe exercise or mental emotion.

No case of albuminuria should be considered functional until the possibility of organic disease has been excluded. The urine should be examined at different periods of the day, and other abnormal ingredients (casts, renal epithelium, &c.) must be looked for in the sediment. Signs of renal disease may also be found in the condition of the vascular system (arterio-sclerosis, hypertrophied left ventricle). Only in the absence of all such signs upon repeated examination is a diagnosis of functional albuminuria justified. Should it appear that the albuminuria is due to organic disease, the source of the albumin must next be determined from the characters of the urinary sediment and from the accompanying symptoms. Do not forget that the *temporary absence* of albumin does not necessarily indicate a healthy kidney; in chronic interstitial nephritis, for example, it may occasionally be absent for days together.

The treatment of albuminuria is that of its cause.

## BLOOD IN THE URINE

The presence of blood in the urine is an important symptom. Two forms are described:—

1. Hæmaturia, or bloody urine proper.

2. Hæmoglobinuria, a condition marked by the presence of blood-pigment in the urine, but *few or no* blood corpuscles. *The blood pigment is generally methæmoglobin* (chocolate colour).



**Hæmaturia.** *Etiology.*—Diseases of the kidneys or the urinary apparatus below them.

Scurvy (especially in children).

*Malignant* forms of fevers, especially the malarial type.

Traumatisms.

Drugs which directly irritate the kidney—turpentine, cantharides, carbolic acid, etc.

Parasites, especially the *bilharzia hæmatobia*.

*Diagnosis.*—1. *By the colour of the urine*, which ranges from a slight smoky tint to deep red or even porter colour.

2. *Microscope.*—Detection of blood discs (in fresh urine).

3. *Spectroscope.*—The characteristic bands of hæmoglobin (generally the reduced Hb).

4. The ozonic ether and guaiacum test gives a blue tint. The reaction is also given by nasal mucus, saliva, and iodine or the iodides.

*If the blood comes from above the ureters*, it is usually freely mixed with the urine; if from the *ureters*, it may be clotted in the form of moulds; if *from the bladder*, it appears in greatest quantity towards the end of micturition; and if *from the urethra*, before or during the *first* part of micturition.

There are no definite means of ascertaining the site of the hæmorrhage by mere examination of the urine alone, and although in many cases the accompanying symptoms make the diagnosis clear, in others a cystoscopic or X-ray examination is necessary for certainty.

*Treatment* of hæmaturia varies with the cause. Rest in the recumbent posture is indicated in all cases. Of drugs, calcium lactate is possibly the most effective, in doses of 10 to 15 grains, (grm. 0·6–1·0) every four hours. It should not be given for more than forty-eight hours. Ergot, aromatic sulphuric acid, or extract of hamamelis may be tried. In the absence of nephritis a full dose of opium is often effective. Surgical measures are requisite in many cases; in some (*bilharzia*) parasitocides must be used.

**Hæmoglobinuria.** *Etiology.*—

The *toxic form* is due to the action of certain poisons, such as *chlorate of potassium*, nitrobenzol, arseniuretted hydrogen, carbon monoxide, bile, etc. It may also follow extensive burns, septic diseases, and severe infections, and it is the central feature of blackwater fever. It may be epidemic in the new-born. For *paroxysmal hæmoglobinuria*, see p. 311.



## URÆMIA

This is the name given to the series of toxæmic symptoms which may arise in association with inflammatory affections of the kidneys and with suppression or diminished quantity of urine. Numerous theories have been advanced to account for the toxæmia, but none of these is free from objection. The most important are—

1. The symptoms are due to retention in the blood of excess of urea. To this it is objected that the blood does not always contain excess of urea, that urea may be injected into the blood without causing uræmia, that anuria may exist without it, and that it may occur in spite of a normal elimination of urea.

2. The urea is decomposed in the blood into carbonate of ammonia, which causes the symptoms. But carbonate of ammonia may not be present in uræmic blood, and the odour of it in the breath is due to decomposition in the mouth, not in the blood.

3. The symptoms are due not to urea alone, but to the total of the normal urinary excreta. None of these except the inorganic salts is present in sufficient quantity to be toxic, and the results of experimental injection of urine are very variable.

4. The symptoms are due partly to the salts of potassium, partly to intermediate products of proteid waste which resemble alkaloids. Bouchard regards the condition as depending partly upon absorption from the intestine (constipation is very constant in nephritis), partly upon non-elimination by the kidneys.

5. The condition is due to defect or to abolition of an internal secretion of the kidneys.

6. Uræmia is produced not by the retention of poisons normally found in the body, but by poisons formed in the diseased kidneys.

No theory yet advanced is adequate to meet all the cases, and the pathogenesis of uræmia is as yet unsettled.

Uræmia may be *acute*, *chronic*, or *latent*. The acute form presents symptoms of a cerebral type; in the chronic form the symptoms are both cerebral and digestive. The latent form is associated with anuria due to obstruction of the ureters.

**Acute Uræmia : Symptoms.**—*Epileptiform convulsions*, which may come on suddenly or be preceded by headache, vertigo, or nausea. They are usually bilateral, but may be unilateral. There may be a single fit, followed by stupor, or a series of fits,



with intervening coma (*status epilepticus*). Tonic spasms or tetany, muscular tremors or twitchings may replace convulsions.

*Coma* accompanies the convulsions, but may occur independently.

The seizure may be *apoplectiform*.

*Delirium* or even *acute mania* may occur.

*Amaurosis* may accompany the convulsions, or occur independently, with no other symptom than headache. The patient finds himself suddenly totally blind in both eyes. There are no changes in the fundus oculi, and sight is perfectly recovered in a few hours or days.

**Chronic Uræmia : Symptoms.**—(A) Cerebral. Persistent headache, vertigo, and drowsiness, either continuous or intermittent, passing into a condition of apathy or stupor, and ultimately into the *typhoid state*. There may be repeated attacks of *dyspnœa*, with rapid shallow breathing and cyanosis (renal asthma), or *Cheyne-Stokes breathing* may occur. *Pruritus* is not uncommon.

(B) Digestive. *Vomiting*, at first in the morning or after meals, later independent of food. The vomitus may contain urea. *Diarrhœa* is frequent. It may be simply catarrhal, or due to ulcerative colitis. *Hiccough* may be troublesome.

For latent uræmia, see Renal Calculus, p. 408.

**Prognosis.**—The first attack of uræmia may prove fatal, but in acute nephritis complete recovery is frequent. In chronic nephritis the patient may survive the attack for several years.

**Diagnosis.**—If a patient is first seen in a comatose state, apoplexy, alcoholic poisoning, and opium poisoning must be excluded. The urine, removed by catheter, must be tested for albumin, the pulse examined for evidence of arterio-sclerosis, and the heart for hypertrophy of the left ventricle. Conjugate deviation of the eyes speaks for cerebral hæmorrhage. In opium poisoning the pupils are contracted, in alcoholic coma usually dilated, in uræmia they are variable, and may be unequal. Remember (1) that cerebral hæmorrhage is common in cirrhosis of the kidney, and (2) that though a patient's breath smells strongly of alcohol, he may yet have uræmic, and not alcoholic coma. If there be any doubt, treat the case as if it were the graver lesion.



## ACUTE BRIGHT'S DISEASE

(ACUTE PARENCHYMATOUS NEPHRITIS)

By Bright's disease we mean a non-suppurative inflammation of the kidneys. To understand the classifications that have been made, it is necessary to recognise the following structures in the kidney, which is liable to diseases in which one or the other of them may be principally involved. They are—" (1) the *tubules*, with their epithelium, forming the parenchyma of the kidney; (2) the *interstitial tissue*, very small in quantity in the healthy organ, but liable to considerable increase by inflammatory processes; (3) the *blood-vessels*, and the glomeruli, consisting of the vascular tufts, the capsule, and the epithelial cells covering the former and lining the latter " (Taylor).

It is evident that in nephritis the inflammatory changes may be most marked in the *tubules*, or in the *interstitial tissue*, or in the *blood-vessels*; but it must be equally clear that no *one* of these special tissues can be affected to any great extent *without implicating the other components of the kidney structure*. In other words, though the various types of Bright's disease are based upon the variety of tissue principally involved, still, it must be distinctly understood that *the kidney structure as a whole shares in the inflammatory changes*, in acute and still more markedly in chronic cases.

Acute inflammation of the kidneys is a disease characterised by grave changes in the urine, dropsy, and slight fever.

**Etiology.**—1. Exposure to cold, especially when the body is overheated.

2. Acute specific fevers and septic states, especially scarlet fever and ulcerative endocarditis. (Albuminuria without the other symptoms of acute nephritis is common in all fevers.)

3. Certain irritating drugs, *e.g.*—cantharides, copaiba, turpentine, etc.

4. Extensive burns involving the abdomen. The nephritis is due to septic absorption.

5. The disease sometimes occurs in pregnancy.

Males are somewhat oftener attacked than females, and acute nephritis is commoner before the age of forty than later.

**Morbid Anatomy.**—Two types are usually distinguished,—*i.e.* the ordinary or catarrhal form, and the so-called infective or glomerulo-nephritis.



1. *Catarrhal Nephritis*.—The kidneys are large, much injected, and the cortex is seen to be disproportionately enlarged. On section, the cut surfaces show up the Malpighian bodies as deep red points; here and there are patches of extravasated blood. The tubules show marked changes, especially the convoluted portions, and their epithelium undergoes cloudy swelling, followed by proliferation and detachment of the cells, which form casts or masses of granular or fatty debris, often to the extent of blocking up the tubules. It must not be forgotten, however, that though the tubules are the parts principally involved, the interstitial portion also takes part in the inflammatory process.

2. *Glomerulo-Nephritis*.—In this form, usually the result of infective diseases, and especially scarlatina, the glomeruli principally suffer. After a preliminary engorgement of the blood-vessels, extensive leucocyte emigration takes place, filling the glomerular capsule, the capillary vessels often burst, and the blood flows into the tubules. The epithelium of Bowman's capsules often proliferates to such an extent that the tufts become obliterated by the pressure, and in time the latter may be actually changed into fibrous nodules. Of course the tubules and interstitial tissue are also involved, though not to the same extent.

**Symptoms** are, at first, chilliness, pain in the back, vomiting, and pain over the brows; the temperature is raised (but may never be high); and the characteristic œdema rapidly appears, at first in the eyelids, cheeks, and ankles, and later it may become general. The pulse is usually quick and of *high tension* from the beginning. The urine is voided frequently, but is scanty in quantity, and indeed may be suppressed for a time. It is dark from the presence of blood, exhibits the well-known smoky hue, and generally gives a copious deposit of urates, blood-discs, granular debris, epithelial and hyaline casts, and blood-casts, and *a large amount of albumin*. Though the urine is of high specific gravity, the total amount of urea passed daily is much decreased. Dropsy may be very slight, or may go on to anasarca; there may be dangerous dropsy of the serous sacs, œdema of the base of the lung or of the glottis. Anæmia is marked. The bowels become constipated, the tongue dry, and there is great thirst. Hypertrophy of the heart is seldom marked in acute cases, although acute dilatation of the heart may occur. If improvement does not quickly take place, grave dangers arise from the development of uræmia; and intense



headache, convulsions, and coma often usher in a fatal termination. Most cases, however, with care, recover; others pass into the subacute stage or "large white kidney."

In cases of acute nephritis complicating the specific fevers, oedema may be but little marked. Scarlatinal nephritis is an exception to this rule.

The more important complications to remember are—

1. Excessive dilatation of the heart in debilitated patients.
2. Oedema of the lungs or glottis; hydropericardium or hydrothorax.
3. Uræmia.

Albuminuric retinitis is not present unless the acute attack arises as an exacerbation of pre-existing chronic disease.

**Treatment.**—Absolute rest in bed, the patient being laid between blankets, occasional purgation by compound jalap powder  $\mathfrak{zss}$ – $\mathfrak{zj}$  (grm. 2·0–4·0), or by salines, diluents, such as Potus Imperialis—bitartrate of potassium  $\mathfrak{zj}$  (grm. 4·0), syrup of lemon  $\mathfrak{ziss}$  (cc. 45), water to  $\mathfrak{zxx}$  (cc. 568)—and a *non-nitrogenous* diet, form the routine treatment. If there is much blood in the urine, dry cupping or hot fomentations should be applied over the loins. The diet should, if possible, be of milk only. Remember that nitrogenous extractives are highly irritating to an inflamed kidney, *hence the necessity of diminishing the quantity of proteids taken*, and of withholding such preparations as beef-tea. Acetate of potash and nitrous ether with hyoscyamus form a useful mixture, as we have at once a *diuretic*, a *dilator of the peripheral arterioles*, and a *sedative to the urinary tract*. But if there is excessive renal congestion causing marked diminution or suppression of urine, diuretics should not be given till the congestion is relieved by cupping, hot air-baths, or leeching, and it may even be advisable to allow nothing but sips of water for the first few days. During convalescence the anæmia should be treated by iron.

*Excessive Dropsy* should be treated by hot packs or hot air-baths, and free purgation by compound jalap powder. Elaterium is seldom necessary. Should these measures fail, pilocarpin gr.  $\frac{1}{8}$ – $\frac{1}{4}$  (grm. 0·008–0·016) may be injected, but only if there is no oedema of the lungs, and if the bronchioles are free. The fluid may also be drawn off from the legs by Southey's tubes, or from the internal cavities by tapping. The diet should be deprived of salt.



In *uræmia*, treatment is the same as for excessive dropsy, *plus* chloroform during the convulsions, and wet cupping. Venesection is a valuable measure in patients otherwise robust.

In *severe dyspnœa* nitrite of amyl is of great service.

## CHRONIC BRIGHT'S DISEASE

Chronic Bright's disease may be divided into two forms—

1. Chronic parenchymatous or tubal nephritis (large white kidney, sometimes leading in the later stages to contraction (small white kidney).
2. Chronic interstitial nephritis (small red or gouty kidney, cirrhosis of the kidney, granular kidney).

Chronic parenchymatous or tubal nephritis may either follow an attack of acute nephritis or arise independently. It is a disease of comparatively early adult life. Chronic interstitial nephritis is rare before the age of forty.

Lardaceous or waxy disease of the kidney (pale waxy kidney), although it has been described as a form of Bright's disease, is really a degeneration, and not at all of an inflammatory nature. But it is often *accompanied* by interstitial nephritis.

Before describing these varieties in detail, we must consider some *general* facts in reference to chronic renal disease. The consequences of defective renal secretion are as follows :—

1. Retention of waste and poisonous products in the blood.
2. Damage to the vascular walls by those poisonous products.
3. Disturbed metabolism ; leading to imperfect nutrition of *all the tissues and organs in the body*. One of its chief expressions is *wasting*, often masked by accompanying œdema. Anæmia is also prominent.

The main changes may be described under six heads :—

1. *Changes in the kidney structure*, involving all its elements, interstitial tissue, tubules, and blood-vessels.

2. *Changes in the urine*, both as regards quantity and quality. The main pathological constituents are albumin, and fatty, granular, or hyaline casts. Blood is often present in chronic parenchymatous, seldom in interstitial nephritis. The excretion of urea is always defective.



3. *Changes in the circulatory system*; hypertrophy of the heart, arterio-sclerosis, and a hard *pulse of high blood-pressure* and prolonged tension (see Fig. 13), in which the dicrotic wave is almost obliterated.

The cause of the cardiac hypertrophy is still unsettled. It has been thought to be secondary to the arterio-sclerosis, and to the increased peripheral tension which occurs quite early in even acute Bright's disease. Herringham considers that it follows



FIG. 13.—Pulse of high blood-pressure, from a case of Bright's disease.

degenerative changes in the great vessels, which lose their elasticity, the resistance in the smaller vessels being thus increased. In Cohnheim's view, the obliteration of the capillary areas in numerous glomeruli demands an increase of pressure, in order that as much blood as formerly passed through the whole kidney should pass through the now restricted field, and thus accounts for the hypertrophy. The arterio-sclerosis itself is often secondary to the renal affection, but in one form of interstitial nephritis it is primary.

4. *Ocular changes.* Albuminuric retinitis is characterised by—  
Oedema of the retina, causing opacity and swelling.

White glistening patches due to fatty degeneration of Müller's fibres.

Flame-shaped extravasations of blood.

Slight optic neuritis, sometimes followed by atrophy.

It occurs in about fifteen per cent. of the cases, and adds to the gravity of the prognosis.

5. In chronic parenchymatous nephritis, *dropsy* is first apparent, as in acute nephritis, in the eyelids and feet: later, general anasarca, and dropsy of the serous cavities—ascites, hydrothorax, or immense dropsy of the scrotum—may occur. The position of the fluid is influenced by gravitation. In interstitial nephritis dropsy is not at first prominent, and later assumes the cardiac type.

The dropsy, which is at first most apparent in the morning, is due to a combination of causes, including—



- (1) The anæmic and hydræmic state of the blood.
- (2) Degeneration of the vascular walls.
- (3) Deficient secretion of urine.
- (4) Defective excretion of salts, especially chlorides.

6. *Tendency to Uræmia*.—Uræmia has already been fully discussed.

When it follows an acute attack, chronic inflammation of the kidney, as Grainger Stewart pointed out, differs in no way from that of other organs. Given a slowly progressing inflammatory action, we should expect at first a stage of enlargement, and finally a state of atrophy. The chronic inflammation must also be preceded by an *intermediate or subacute stage*. But the relations of chronic to acute inflammation are by no means always so simple as this. Chronic Bright's disease may arise independently of the acute disease, "large white kidney" is not necessarily followed by "small white kidney," and the causes of interstitial nephritis are, as will be seen immediately, very different from those of the parenchymatous form.

**Chronic Parenchymatous Nephritis** is often a sequel of an acute attack, but may develop insidiously after infective diseases or in the course of chronic suppurations or syphilis. It leads either to fatty degeneration of the kidney substance ("large white kidney"), in which the capsule is non-adherent, the cortex yellowish or mottled, and the pyramids injected, or, later, to fibrous degeneration ("small white kidney"). In this stage the capsule is adherent, the surface granular, the cortex narrow and pale.

In its early stages ("subacute nephritis") the *symptoms* approximate to those of the acute form. The urine is scanty, highly albuminous, contains blood, and is defective in urea. Fatty casts are present in quantity. Dropsy is prominent (Fagge quotes the saying that "large white kidneys make a large white body"), and affects the internal cavities even more frequently than in acute nephritis. In the later stages, the urine is increased in quantity, blood and albumin are less, the casts tend to become granular or hyaline, and dropsy is less prominent. Cardiovascular changes are more pronounced.

The relations of acute nephritis to large white kidney and to secondary contraction are still a matter of dispute. The older view that these affections represent consecutive stages of a single disease is widely combated. Herringham's conception is that both large white kidney and secondary contraction may arise out of acute nephritis, not as consecutive events, but as radii



## Differential Diagnosis of the Forms of Bright's Disease

CHRONIC NEPHRITIS.					
ETIOLOGY.	ACUTE NEPHRITIS.	CHRONIC PARENCHY- MATOUS NEPHRITIS: LARGE WHITE KIDNEY.		SECONDARY CONTRACTED KIDNEY: SMALL WHITE KIDNEY.	CHRONIC INTERSTITIAL NEPHRITIS; PRIMARY CON- TRACTED OR SMALL RED KIDNEY; CIRRHOTIC OR GRANULAR KIDNEY.
		Acute nephritis; prolonged influence of cold and damp; malaria, cardiac lesions, phthisis.			Gout, lead-poisoning, excess in alcohol or nitrogenous diet (uric acid).
CHARACTERS OF URINE.	Quantity.	Scanty.	Rather less than nor- mal.	Not less than nor- mal; usually increased.	Very abundant.
	Colour.	Turbid, pale red or smoky to deep red.	Turbid, resembling meat infusion.	Fairly clear.	Clear, pale.
	Specific gravity.	High.	Somewhat raised, may be normal.	A little below nor- mal.	Low.
	Blood.	Abundant.	Commonly present.	In small quantity.	Usually <i>absent</i> .
	Albumin.	Abundant.	Abundant.	In moderate quan- tity.	In very small quantity; may be absent for some time.



Sediment.	Abundant; white and red corpuscles; blood-casts, epithelial and granular casts; urates.	Abundant. White and red corpuscles; numerous casts, especially <i>fatty</i> .	In moderate quantity. Casts fairly numerous, granular and hyaline chiefly.	Very scanty. Casts few, chiefly hyaline.
	Salts and Urea.	Diminution of urea, etc.	Marked diminution of urea, etc.	Marked diminution of urea, etc.
Cardiac Hypertrophy.	Marked diminution of urea, chlorides and phosphates.	Sometimes present.	Usually present.	Almost always present, and very considerable.
Dropsy.	Usually <i>absent</i> , unless in acute exacerbations of chronic disease.	Marked; <i>dropsy of internal cavities</i> .	Moderate; both subcutaneous and internal.	Usually absent; later dropsy of cardiac type when heart fails.
Uræmia.	Marked; shifting from place to place with position of patient.	Fairly frequent.	Frequent; both chronic and acute types.	Very frequent; both chronic and acute types.
Associated symptoms.	Frequent.	Marked pallor of skin, retinitis, bronchitis, etc., inflammations of internal organs.		
Death results from—	Those of infective diseases or intoxications.	Uræmia, or oftener internal inflammations.	Uræmia, cerebral hæmorrhage, cardiac failure, internal inflammation.	
	Uræmia, pulmonary œdema, internal inflammations.			

Modified from Leube.



diverging from a common centre. If the tubules are chiefly affected in the acute stage, the tendency will be to large white kidney; if the interstitial tissue has been much involved, contraction will follow. There are intermediate forms, as any number of radii may be interposed between two given radii, but each pursues its own line of development. Cases beginning as large white kidney end as such, in his view, with much dropsy, little arterial change, and death by asthenia; while in contracted kidney cardiovascular changes are marked, œdema is slight, and death may be due to uræmia, cerebral hæmorrhage, or cardiac failure.

**Chronic Interstitial Nephritis** (*granular kidney, cirrhosis of the kidney*) has two forms, one primary, the other secondary to a general arteriosclerosis (*arteriosclerotic kidney*). Its most frequent causes are gout, lead-poisoning, chronic alcoholism, or excessively nitrogenous diet, associated with the less active metabolism of middle age. Heredity is sometimes a factor. The kidney is small, the capsule firmly adherent, the surface purplish-red, uneven, and studded with small cysts. The cortex is narrow and fibrosis is very marked. Arterio-sclerosis is pronounced.

In the arterio-sclerotic form, most common in America (Osler), the kidney is comparatively little contracted.

*Symptoms.*—Chronic interstitial nephritis is a disease of middle and later life. Its onset is very insidious, and often there is nothing to attract the patient's attention to such a serious condition, beyond languor, frequency of micturition, *especially at night*, possibly dyspeptic symptoms, diffuse headache, and thirst. The urine is pale, clear, and abundant, but albuminuria may be absent for a considerable time. Casts are very few, and chiefly granular or hyaline. Occasionally there may be an intercurrent attack of hæmaturia. When the disease is well marked, and cardiac hypertrophy begins to fail, the symptoms become very pronounced, and are such as pallor, breathlessness, attacks of uræmic asthma, dimness of vision, and dropsy of the cardiac type. Pulmonary complications are frequent. It is very essential that the condition should be early diagnosed, and any of the early symptoms mentioned above, occurring in a middle-aged man, demands a careful examination of the pulse, heart, retina, and urine.

The foregoing remarks, along with the diagnostic table on pp. 401–2 appended to them, should enable the beginner to have a clear idea of the elementary facts of chronic Bright's disease. An important feature, more notable in interstitial than in



chronic parenchymatous nephritis, but common to both, is the liability to death by *cerebral hæmorrhage*. In both, too, internal inflammations are frequent causes of death; and in interstitial nephritis the long-standing cardiac hypertrophy may end in dilatation, and in death from cardiac failure, sooner or later.

The course of chronic nephritis may be interrupted by inter-current acute attacks.

**Treatment of chronic Bright's disease** must be considered under two heads—

1. General treatment.

2. Symptomatic treatment.

*General Treatment.*—The indications vary with the stage of the disease. In the stage of large white kidney, treatment must evidently be upon much the same lines as in acute nephritis, but in the subsequent contracted kidney, and also in interstitial nephritis, confinement to bed is undesirable. The patient should be placed in the most favourable hygienic conditions, and guarded from cold by woollen underclothing. The action of the skin should be promoted by tepid baths and friction; he should take gentle exercise in the open, and should live on a varied diet, containing only a limited quantity of nitrogenous food. Meat should not be allowed oftener than once a day, and alcohol should be forbidden. Strychnine may be used as a general tonic, and iron, or iron and arsenic, for the anæmia. The bowels should be kept free by the use of saline laxatives, and although mercurials are sometimes badly borne in renal affections, there can be no doubt as to the efficacy of an occasional dose of pil. hydrarg. or calomel, given at night-time, and followed by an aperient water in the morning. Hepatic depletion by brisk cathartics is often more efficacious than any other treatment in allaying urgent symptoms.

*Symptomatic Treatment.*—High blood-pressure, when moderate, should not be interfered with, but if it is excessive and causes troublesome symptoms, saline laxatives are indicated, and also nitroglycerin or nitrate of sodium.

Headache is best relieved by caffeine. When the tension is very high, a good purge and a few whiffs of nitrite of amyl are often promptly effective.

*Cardiac failure* is to be met by strychnine and digitalis.

*Dropsy*, in large white kidney, requires the measures detailed under Acute Nephritis.



*Threatened Uræmia* requires hot packs, smart purgation or pilocarpin, the last only in the absence of pulmonary complications.

*Albuminuria* cannot be effectively controlled by any known drug.

### LARDACEOUS (AMYLOID) DISEASE OF THE KIDNEY

This affection, which has been sometimes described as a form of Bright's disease, is not an inflammatory lesion, but a degenerative change. It is, however, frequently associated with interstitial nephritis.

**Etiology.**—The disease follows upon prolonged suppuration, especially that due to phthisis or to caries of bone, and upon long-standing syphilis, whether associated with suppuration or not. It occurs, but rarely, in cases of malarial cachexia.

**Morbid Anatomy.**—The kidney is large (it may be contracted if interstitial nephritis co-exists), pale and smooth on the surface, and firm, the capsule stripping off easily. On section, the cortex has a glistening waxy appearance, the medulla being injected. The lardaceous tissues stain mahogany brown with tincture of iodine.

The changes affect first the walls of the vessels (glomeruli and arterioles), spreading thence to the basement membrane of the tubules.

**Symptoms** vary considerably, according as interstitial nephritis is or is not present. Where it exists, the urinary changes are those already tabulated on pp. 400–1. Where it is absent, the urine is variable in quantity, pale yellow, *clear*, of normal or low specific gravity, and free from blood. Albumin is sometimes absent or scanty, but usually abundant; sediment is absent, though a few hyaline or waxy casts may be found. *Urea is not diminished.* Dropsy is very considerable, but cardiac hypertrophy, retinitis, and uræmia are all absent unless there are inflammatory complications. Death occurs from exhaustion, or from the causative disease.

**Diagnosis** is easy if, in the presence of a definite cause, and with evidence of amyloid disease elsewhere (spleen, *liver*, intestine), the above symptoms are to be found. It is difficult when the amyloid change is superadded to an interstitial



nephritis. The kidney is never sufficiently enlarged to be accessible to physical examination.

**Treatment** is that of the causative disease. Cases not too far advanced may recover completely where the cause can be removed (tuberculosis of joints, etc.).

### PYELITIS, PYELONEPHRITIS, AND PYONEPHROSIS

By pyelitis is meant an inflammation limited to the pelvis of the kidney. The inflammation may extend to the substance of the kidney, and there very often leads to suppuration (*pyelonephritis*). Should a large collection of pus form within the pelvis, distending that structure and the calyces, and flattening out the renal substance, the condition is called *pyonephrosis*.

**Etiology.**—The exciting cause of these conditions is almost invariably microbic, the bacillus coli being the commonest organism, although others, such as the tubercle bacillus or staphylococci, may also be found. There are many predisposing causes, among which are—

1. Morbid states of the blood, such as acute infections, diabetes, Bright's disease, anæmia.
  2. Extension of inflammation from below ("surgical kidney").
  3. Calculi, blood-clots, parasites, tumours.
  4. Certain drugs—cantharides, etc.
  5. Pressure upon the ureter, causing dilatation of the pelvis.
- Decomposition of the retained urine may follow.
6. Injuries to the spinal cord.
  7. Pregnancy.

**Morbid Anatomy.**—If the pelvis alone is inflamed, its mucosa is opaque and thickened, showing punctiform hæmorrhages or superficial ulcers. Pus is present in the pelvic cavity. In pyelonephritis linear areas of suppuration are found extending between the pyramids towards the cortex of the kidney, in which small abscesses are present. In pyonephrosis the wall of the sac is formed by the kidney substance, which is flattened as the result of pressure, and becomes fibrotic from coincident nephritis.

**Symptoms** depend upon the cause, the stage, and the complications. A simple pyelitis gives rise to but few definite symptoms; a localised pain in the lumbar region may be the



chief or only complaint on the part of the patient, or perhaps he seeks advice on account of frequent passage of urine, which, on examination, may be found to contain pelvic epithelia or blood. When suppuration commences, the symptoms are much more definite: the urine now contains blood, pus cells, and albumin, but, if the *bacillus coli* is the infecting organism, remains acid in reaction. There may be occasional attacks of pyrexia, resembling ague. As the kidney *substance* becomes involved, the symptoms of more or less acute nephritis appear. If the suppurative process is pronounced, there are usually rigors, sweating, wasting, and fluctuating temperature. The urine may become ammoniacal, viscid, and "ropy," if the pyelitis has followed upon cystitis. Hæmorrhage is usually slight, but in calculous pyelitis there may be intermittent profuse hæmaturia.

Sometimes the debris chokes up the ureter, or it may be occluded by a calculus, and the urine which escapes by the free ureter will be clear for a short time, only to become purulent again when the plug moves away. If the pus be pent up, it causes a cystic swelling of the renal pelvis (pyonephrosis). It will of course be easily understood that, if such a condition occurred in both kidneys, death would speedily result from uræmia.

The physical signs of pyonephrosis are those of a tumour in the flank, which (when large) causes distinct bulging in front and behind. Such a tumour—

Can be separated from the liver or spleen ;

Is always crossed by the colon ;

Yields a dull note on percussion ; and is *tender* on pressure.

Tenderness in the lumbar region is often present in the absence of tumour.

**Prognosis.**—Cases occurring in the course of the acute fevers usually recover spontaneously. In other forms the condition, if left alone, is frequently fatal, but sometimes, even after pyonephrosis has occurred, the pus dries up, the walls of the cyst become coated with patchy layers of phosphatic deposits, and the cavity in time contracts. In other cases the abscess may burst through the capsule of the kidney into the peritoneum, or so as to form a retroperitoneal abscess, which in turn may burrow in various directions, leading to subphrenic abscess, perforation of the diaphragm, etc. Death may also be brought



about by extensive lardaceous disease, by cachexia, or by uræmia (typhoid state).

**Treatment.**—Our treatment should aim to—

1. Remove the cause if possible. In simple pyelitis operative treatment is unnecessary. Rest, light diet, diluent drinks, and urinary antiseptics, such as urotropin, are sufficient.

2. Relieve the inflamed kidney by rest, cupping, and the administration of sedative salines, such as citrate of potassium with hyoscyamus, and the free drinking of liquids.

Vaccines may be of use in cases due to bacillus coli infection.

Pyonephrosis demands surgical treatment, either by emptying and draining the sac, or in some cases by nephrectomy.

## HYDRONEPHROSIS

A condition due to distension of the pelvis and calyces of the kidney by urine. It may be congenital or acquired.

**Etiology.**—Congenital hydronephrosis is usually double, being caused by imperforate urethra. The kidneys may be so dilated as to impede delivery. The acquired form may be single or double. Where it is single it is due to obstruction in the ureter however caused (stricture, compression by cicatricial bands, impacted calculus, floating kidney, etc.); where it is double, to obstruction lower down, which is usually partial. Thus urethral stricture, enlarged prostate, villous tumours of the bladder, and in the female displacements or tumours of the uterus, may cause it. In such cases death often ensues from uræmia before the renal tumour becomes large enough for recognition during life. In unilateral hydronephrosis the other kidney carries on the renal function. Symptoms dangerous to life do not follow, and the tumour may grow to a very great size.

**Morbid Anatomy.**—Dilatation of the ureter and pelvis are followed by pressure on the pyramids, with flattening and atrophy of the kidney. The sac is divided into loculi by septa formed of the connective tissue surrounding the inter-pyramidal vessels. There are corresponding bulgings on its surface. The sac contains a pale watery fluid, albuminous, but presenting only traces of urea or other urinary constituents.

**Symptoms.**—In bilateral cases with incomplete obstruction, frequent passage of very dilute urine, followed later by



symptoms of chronic uræmia, and death in the typhoid state. In unilateral cases, weight and pain in the loin, and if the tumour be large, nausea, vomiting, or constipation from pressure on the colon. If the obstruction be intermittent, history of sudden passage of a large amount of clear urine, followed by disappearance of the tumour, and gradual re-accumulation. The physical signs are those of renal tumour in general.

**Treatment.**—Do not interfere if there be no discomfort. Sometimes the sac has been emptied by massage, but operation is usually required. Repeated aspiration may result in cure, but it may be necessary to resort to nephrotomy and drainage, or even to nephrectomy. Make certain first (*a*) that there is another kidney, (*b*) that it is functionally active.

## RENAL CALCULUS

(NEPHROLITHIASIS. STONE IN THE KIDNEY)

Renal calculi are due to the deposition of certain solid constituents of the urine in the substance of the kidney or in the renal pelvis. They may be of any size, forming granules of “sand” or “gravel,” or large concretions. They may be solitary or very numerous.

**Characters.**—Calculi of appreciable size are made up of concentric layers arranged around a nucleus often of quite different composition. The nucleus may consist of crystals of uric acid or oxalate of lime; or of colloid bodies such as mucin, epithelia, or tube casts; or of micro-organisms. The laminae are composed of uric acid (the commonest form), oxalate of lime, alternate layers of uric acid and oxalate, or sometimes phosphate of lime and triple phosphates deposited upon a nucleus of one of the other crystals. This occurs in the presence of ammoniacal decomposition of the urine in the pelvis, as sometimes in pyonephrosis. Other forms of calculi (cystin, xanthin, etc.) are very rare.

Uric acid calculi are of a reddish colour, hard, smooth, and, if they are multiple, faceted on the opposing surfaces. Oxalate calculi are very hard, greyish, smooth, and rounded while small, but when they become larger presenting a rough tuberculated surface (“mulberry calculi”). Phosphatic calculi may grow to a great size by continual deposition of phosphates from the alkaline urine. They tend to assume the shape of the pelvis and calyces, and form “dendritic” masses.



**Etiology.**—The deposition of uric acid is favoured by increased acidity of the urine, and diminution of the salts, especially phosphates, which tend to hold it in solution. Oxalates tend to be deposited when there is excess of them in the urine. But neither uric acid nor oxalate crystals agglomerate into calculi without some additional factor, which is supplied by the presence in the pelvis of colloid material, such as mucus, albumin, or blood. These may be present as the result of slight catarrhal pyelitis or congestion of the pelvis. That such local causes are at work is shown by the frequency with which calculus is unilateral.

**Morbid Anatomy.**—A calculus generally sets up some degree of pyelitis, which may be suppurative, and lead to pyonephrosis. If the ureter be obstructed, the kidney may atrophy, or hydronephrosis may follow. Malignant tumours have been attributed to the continued irritation of the calculus. If both ureters be obstructed, anuria and the symptoms of latent uræmia will follow.

**Symptoms** may be latent throughout life. The most characteristic is renal colic, but between the attacks there may be (1) pain in the lumbar region, aching or dragging, and made worse by movement; (2) some degree of tenderness on deep pressure in the lumbar region; (3) hæmaturia, recurring at intervals, often without other symptoms, aggravated or induced by movements especially of a jolting nature (riding, jumping, etc.), and relieved or removed by rest; (4) marked acidity of the urine, which may contain uric acid or oxalate crystals, and, if there be pyelitis, pus.

*Renal colic* results from the passage or attempted passage of a calculus down the ureter to the bladder. It is characterised by excruciating pain beginning in the loin and radiating over the anterior aspect of the abdomen on the affected side, and downwards to the testicle, which is retracted, and to the inner side of the thigh. The skin is hyperæsthetic over the affected abdominal area, but the scrotal skin is not so, having a different nerve supply. The patient is faint, and sweats profusely. The pulse is small and rapid; there are nausea and vomiting; and pregnant women may abort. The paroxysm may last a few hours or a few days. It ends suddenly if the stone slips into the bladder; more gradually if it becomes impacted. During the attack urine is passed frequently and in small quantity. It is usually scanty, high-coloured, and blood-stained. It may be suppressed.



Where the opposite ureter is already obstructed, death may ensue from *obstructive suppression*, the symptoms being those of *latent uræmia*. There is anuria, without marked symptoms beyond lassitude and insomnia for about a week. Then muscular twitchings appear, the pupils become contracted, and there is gradually increasing dyspnœa, which leads to death within a day or two. The mind remains clear till just before death.

When the *diagnosis* is doubtful, examination under the X-rays is of much service.

**Treatment.**—Uric acid calculi are soluble in alkaline solutions. The urine should therefore be rendered alkaline by salts of potassium, such as the citrate or acetate, ʒj-ʒij (grm. 4·0-8·0) every three hours, and the treatment should be kept up for three months or more, but should be stopped if the urine becomes ammoniacal. The diet should be light, as little nitrogenous as possible, and stimulants should be avoided. Plenty of fluid should be taken. Oxalate calculi are not affected by this treatment. In them, and in uric acid calculi where the solvent treatment has failed, operation (nephrotomy or nephrectomy) may be required.

The treatment of renal colic is to be directed to the relief of pain, by hot fomentations, hot baths, morphia, and, if necessary, chloroform.

## RENAL TUBERCULOSIS

This condition may occur in the form of miliary tubercles in the course of a general infection, or may be primary. The disease may begin in the kidney and spread downwards, or may extend to the kidney from the lower urinary tract, in which case both kidneys are usually affected.

**Morbid Anatomy.**—The primary form of the disease begins by the formation of tubercles in the medulla, which coalesce and lead to caseation and softening. Cavities are thus formed, separated from each other by inter-pyramidal septa of fibrous tissue. The whole renal substance may be ultimately destroyed. The pus is discharged in the urine, causing tuberculous pyelitis, extension of the process to the ureter, and consequently partial or complete occlusion of that structure, leading to pyonephrosis.

**Symptoms** are chiefly those of pyelitis, and consist of dull aching pain in the loin, frequent micturition, constant presence



of pus in an acid urine, proportionate albuminuria, and often hæmaturia, much less readily influenced by rest than that of calculus. Tubercle bacilli *may* be found in the urine. There are the usual general symptoms of wasting, fever, and sweating; later, pulmonary or peritoneal tuberculosis may appear. If pyonephrosis is present, there may be evidence of renal tumour; tuberculosis *per se* does not materially increase the size of the kidney.

**Diagnosis** depends upon the presence of bacilli in the urine, the existence of tuberculosis elsewhere, or the positive result of the tuberculin tests (*see* p. 365). An examination by the cystoscope is called for to determine whether the bladder is affected.

**Treatment**, medicinal and hygienic, is that for tuberculosis in general, with the addition of such urinary antiseptics as salol, urotropin, or benzoate of soda. Nephrectomy can only be thought of where the disease is unilateral and the bladder is unaffected; nephrotomy and drainage may sometimes be of use.

## RENAL TUMOURS

These are almost invariably malignant. The so-called sarcomata are congenital tumours of complex structure, often growing to an enormous size, and running a brief course to death. The tumours of adult life are either epitheliomata originating in the pelvis, or adeno-carcinomata originating in the cortex. Many of these renal tumours are supposed to be of suprarenal origin, developing in portions of suprarenal tissue included in the kidney substance. They are therefore called *hypernephromata*. The epitheliomata cause uniform enlargement of the kidney, the adeno-carcinomata nodular outgrowths. There may be direct extension through the capsule of the kidney to the retroperitoneal tissue; or extension may take place by the veins (the renal vein or inferior cava may be blocked by the growth) to the lungs, or by the lymphatics to the liver and lumbar glands.

**Symptoms** are (1) lumbar *pain*, dull and aching, but sometimes, where blood-clot is present in the ureter, like that of renal colic; (2) *tenderness* on pressure; (3) recurrent *hæmaturia*, often profuse; (4) sometimes *varicocele*, from pressure on the spermatic vein; (5) evidence of tumour; (6) the general symptoms of malignant disease.

**Characters of a Renal Tumour.**—(1) The tumour fills up the loin, and bulges forwards. Its shape is rounded, and its



edges blunt. Its direction is vertical. (2) The fingers can be slipped beneath the ribs above it (distinction from liver or spleen), and beneath the iliac crest below (distinction from ovarian tumour). (3) It is only slightly movable on respiration. (4) The colon may be traced in front of it by palpation or percussion (see that the bowel is emptied before examining). (5) The dulness is continued posteriorly to the middle line (distinction from spleen). (6) It may cause *pressure symptoms*—(a) varicocele, (b) ascites from pressure on the portal vein, (c) enlargement of superficial veins from pressure on the vena cava, (d) intestinal obstruction.

**Treatment**, as far as it is not surgical, is purely palliative.

### MOVABLE OR FLOATING KIDNEY

The kidney may be unduly mobile from laxity of the subperitoneal fascia. The peritoneum may nearly surround it, forming a false mesonephron. This condition may be part of a general enteroptosis, or may be the result of laxity and pendulousness of the abdominal wall. Tight lacing is said to cause it; it is more frequent in the female than the male; and the right kidney is much more commonly affected than the left.

**Symptoms** may be entirely absent. There is often dragging pain in the loin, worse on exertion. Paroxysmal crises of pain like that of renal colic, attended by rigor, vomiting, and collapse, may occur (Dietl's crises), and neurasthenia and dyspepsia are frequent. Intermittent hydronephrosis may occur from temporary kinking of the ureter. A freely movable tumour of the size and shape of the kidney is to be felt. It may occupy almost any position in the abdominal cavity. Squeezing it causes a feeling of nausea. It can be pushed back into the loin by palpation.

**Treatment.**—If a displaced kidney causes no symptoms, and is found by accident in the course of an abdominal examination, the patient should not be told of its existence. No treatment may be required, but in other cases a carefully adjusted pad and bandage may retain the kidney in position. Sometimes it is necessary to stitch it to the posterior wall.



# DISEASES OF THE NERVOUS SYSTEM

## I. INTRODUCTORY

BEFORE commencing the study of diseases of the nervous system, the reader is strongly advised to digest the following account of the anatomy and physiology of the motor and sensory tracts.

### THE NEURONE

The primary elements of the nervous system are the neurones and the neuroglia, the former being the discharging and conducting structures, and the latter the supporting. The **neurone** is the essential element of the nervous system, which may be regarded as built up of an enormous number of them arranged in series, and occupying definite tracts. A neurone consists of a nerve cell and its branches (axon and dendrons or dendrites). The **dendrons** belonging to a single cell may be many, or there may be but one; in either case they are short processes, soon subdivided into many terminal branches forming an arborisation. They are made up of fibrillæ, and of granular matter lying between the fibrillæ, which are continuous, through the body of the cell itself, with the fibrillæ of other dendrons or of the **axon**. This process, entirely composed of fibrillæ, gives off collaterals at right angles to its course, and these, like the axon itself, often end in an arborisation around the dendrons of another nerve cell. These places of linkage of one neurone with another are called **synapses**. An axon may be either long or short; in the former case it does not branch for a considerable distance, becomes surrounded with myelin, and passes as a nerve fibre into the white matter; in the latter it breaks up into branches close to its cell, which is known as a "link cell," and is confined to the grey matter alone. The dendrons conduct impulses *towards* the cell, the axons *away* from it.

The **nerve cell** itself, besides its nucleus and nucleolus and the fibrillæ which traverse it, contains a series of angular granules



(Nissl's bodies) similar to those of the dendron. They stain deeply with methylene blue, and are an index of the state of health or degeneration of the cell. The multiplication of nerve cells ceases shortly after birth, but their growth is active, and they have great reparative power.

The **neuroglia** is composed of a network of delicate interlacing fibrils containing a number of nucleated cells (Deiter's cells) embedded in it. It everywhere interpenetrates the nervous elements, but is most abundant round the central canal of the cord and the ventricles of the brain, and in the substantia gelatinosa of Rolando, which lies at the tip of the posterior cornu of the cord. In the various "scleroses," it is increased at the expense of the nervous elements.

A neurone is "efferent" or "afferent," according as its axon carries impulses from the central nervous system towards the periphery, or from the periphery towards the centre. By the superposition of one efferent neurone upon another, or of one afferent neurone upon another, efferent, descending, or *motor*, and afferent, ascending, or *sensory paths* are built up, which occupy definite positions in the spinal cord and brain. A brief account of the anatomy of the cord is necessary for the comprehension of these paths.

## THE SPINAL CORD

**The Spinal Cord** consists of a tubular prolongation of the brain enclosed in three membranes, the dura mater externally, the arachnoid, and internally the pia mater. Between the arachnoid and the pia mater is the sub-arachnoidal space, filled with cerebro-spinal fluid, and *continuous*, through the foramen of Majendie, *with the ventricles of the brain*. The blood vessels enter the spinal cord in trabeculæ derived from the pia mater, which closely invests it. The substance of the cord consists of white matter externally, and grey matter internally.

**The Grey Matter**, which is more vascular than the white, is composed of a network of medullated and non-medullated fibrils (axons and dendrons of the cells, and commissural fibres), and of nerve cells, supported by a framework of neuroglia. The nerve cells are arranged for the most part in groups, of which the following are the most important:—

In the *Anterior Horn*.

1. Median or internal group, close to the anterior column of white matter.
2. Vento-lateral group, in the outer part of the horn.



In the *Intermedio-lateral Tract*.

3. Lateral group, most prominent in the thoracic region.

In the *Posterior Horn*.

4. *Vesicular Column of Clarke*, at the junction of the posterior horn with the grey commissure. These cells send their axons into the white matter of Flechsig's tract.
5. Smaller groups at the margins of the horn, and in the substantia gelatinosa; also solitary cells throughout the horn.

The anterior cells are multipolar and give origin to motor nerve-roots; they also act as *trophic centres* for motor fibres in the nerve trunk. The cells of the column of Clarke are important through their implication in the transmission of sensory impulses from the posterior root-fibres.

**The White Matter**, though apparently homogeneous, is really divisible into columns or tracts which have been recognised by the study of degenerations, and each of which is specially concerned in the transference of impulses up to or from the brain, or in some instances between different parts of the cord. The chief tracts are:—

#### I. Efferent or Descending (see Fig. 14).

- (a) *The Crossed Pyramidal Tract*, in the posterior part of the lateral column, between the posterior horn and the direct cerebellar tract of Flechsig.

- (b) *The Direct Pyramidal Tract*, on either side of the anterior median fissure. Both these tracts are formed of the axons of cells in the motor area of the cortex. The fibres of the crossed pyramidal tract, which is much the larger, decussate in the medulla, passing to the opposite side, while those of the direct pyramidal tract

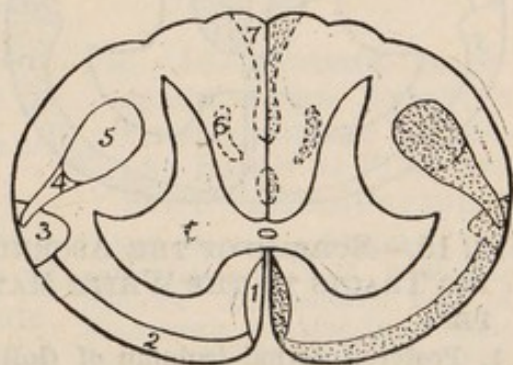


FIG. 14.—SCHEME OF THE DESCENDING TRACTS IN THE WHITE MATTER.

1, Direct pyramidal (Türck); 2, descending cerebellar or vestibulo-spinal (Löwenthal); 3, tract of Helweg; 4, pre-pyramidal or rubro-spinal (Monakow); 5, crossed pyramidal (Türck); 6, comma tract; 7, association tracts, containing both ascending and descending fibres.



continue on the same side as their cells, probably decussating lower down in the white commissure.

- (c) *The Rubro-spinal or Pre-pyramidal Tract*, a crossed tract descending from the red nucleus in the cerebral peduncle.
- (d) *The Descending Cerebellar or Vestibulo-spinal Tract*, uncrossed, coming from Deiter's nucleus in the medulla, and through the vestibular division of the auditory nerve connecting cerebellar nuclei with the grey matter of the anterior cornu.

## II. Afferent or Ascending (see Fig. 15).

- (a) *The Postero-internal Tract (column of Goll)* next the posterior median fissure.
- (b) *The Postero-external Tract (column of Burdach)* between the column of Goll and the posterior horn.

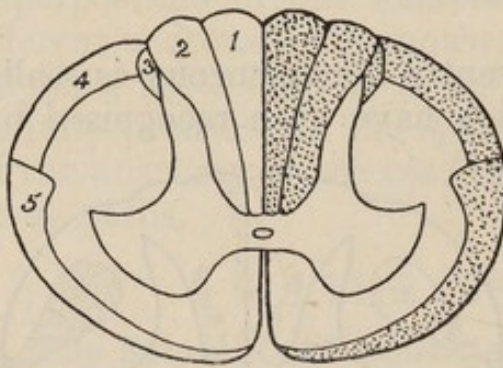


FIG. 15.—SCHEME OF THE ASCENDING TRACTS IN THE WHITE MATTER.

1, Postero-internal (column of Goll); 2, postero-external (column of Burdach); 3, marginal (Lissauer); 4, postero-lateral ascending cerebellar (Flechsig); 5, antero-lateral ascending cerebellar (Gowers).

*of the cord* (cells of the posterior horn and of Clarke's column).

- (e) *The Marginal Tract of Lissauer*, a small area just external to the posterior roots. It contains fibres from the outer bundle of posterior root-fibres.

Of these five tracts the first four run the whole length of the cord, and are known as the *long ascending tracts*. Among other tracts of secondary importance are a tract from the anterior corpora quadrigemina to the opposite anterior columns, and the small comma tract of the posterior column, which, though *descending*, is composed of *sensory* fibres.



## THE MOTOR PATH

The motor path is composed of two sections :—(1) An upper or cerebro-spinal efferent neurone (the pyramidal system, Wyllie's *first trophic realm*), comprising the cortical motor cell, its dendrons, and its axon, which passes downwards through the cord to terminate in an arborisation round a motor cell of the anterior grey cornu ; (2) a lower or spino-peripheral neurone (second trophic realm), comprising the motor cell of the anterior cornu, its dendrons, and its axon passing downwards in a peripheral nerve to the muscle, and ending on the muscular fibres.

Tracing them from above downwards, the motor fibres, originating in the cells of the cortex, principally around the *fissure of Rolando*, traverse—

1. The *corona radiata*.

2. The *genu* and the anterior two-thirds of the *posterior limb* of the internal capsule. The fibres in the genu go to the eye, head, tongue, and mouth ; those in the posterior limb to the limbs and trunk. (See Fig. 16.)

3. The *crusta* of the *crus cerebri*, where the fibres going to the nucleus of the third nerve cross to the opposite side.

4. The *pons Varolii*. Here the fibres for the face cross over, and end in arborisations round the nucleus of the seventh nerve, the cells and axons of which (fibres of the nerve) constitute the lower neurone.

5. The *medulla oblongata*. At the lower part of the medulla the bulk of the fibres crosses (decussation of the pyramids, Fig. 17) to the opposite side, and passes down the cord as the *crossed pyramidal tract*, and thence (possibly by way of a short intermediate dendron linking the posterior cornual cells with those of the anterior cornu, possibly direct) to a terminal arborisation round the cells of the anterior horn. A few fibres pass down from the medulla without decussation, and form the direct pyramidal tract.

Subsidiary motor tracts, by which in the event of damage to

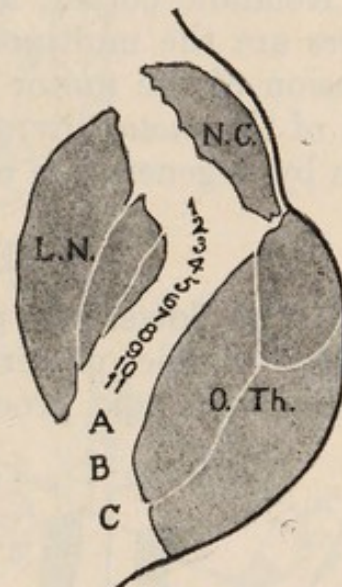


FIG. 16.—SCHEME ILLUSTRATING THE RELATIVE POSITION OF THE DIFFERENT BANDS OF FIBRES IN THE INTERNAL CAPSULE.

N.C., Nucleus caudatus; O. Th., optic thalamus; L.N., lenticular nucleus. The anterior limb of the capsule contains, in front the fibres of the fronto-pontine projection system, and posteriorly some of the fibres of the pyramidal system—1, fibres to head; 2, head; 3, tongue. The posterior limb contains—4, mouth; 5, arm; 6, hand; 7, trunk; 8, hip; 9, knee; 10, leg; 11, toes. In the posterior part we have sensory fibres A, the temporo-pontine projection fibres B, and the visual radiating fibres C.



the pyramidal tract, motor impulses may be to some extent conducted, are the rubro-spinal and the vestibulo-spinal. The former appears also to convey inhibitory impulses, the latter impulses increasing motor tone.

Such is the course of the upper neurone, and that of the lower or spino-peripheral neurone has already been indicated. It will be seen that the trophic centre for the *motor tracts* is situated in the Rolandic cortex, and that the trophic centres for the *motor nerves* are the multipolar cells in the anterior horn of the cord. A lesion of the motor cortex is therefore followed by degeneration of the *motor* (pyramidal) *tracts*, and a lesion of the anterior horn by degeneration of motor *nerves*.

### THE SENSORY PATH

1. The **lower or peripheral neurone** has its cells in the ganglia of the posterior nerve-root. These cells give off an apparently single process, which shortly divides like a T into

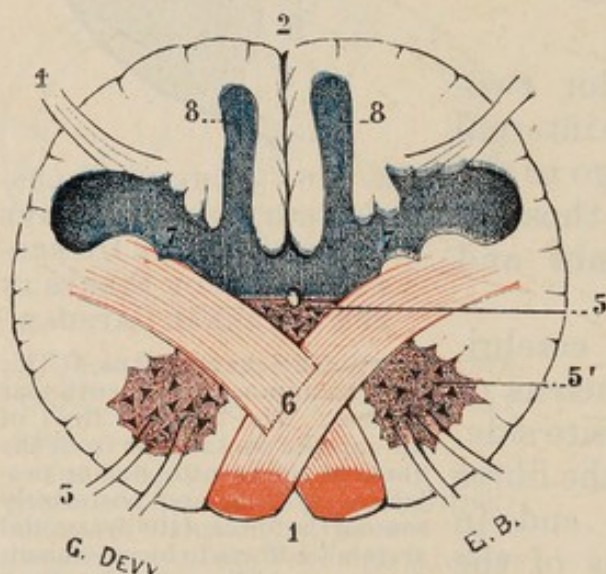


FIG.17—DIAGRAMMATIC SECTION THROUGH THE LOWER PART OF THE MEDULLA (AT THE INFERIOR OR MOTOR DECUSSATION) (TESTUT).

1, Antero-median fissure; 2, postero-median fissure; 6, pyramidal decussation; 3, separated anterior cornua; 5, central canal; 7, posterior cornua of grey matter; 8, beginning of the nucleus gracilis.

cells of the grey matter. Some of these are reflex collaterals, arborising round cells of the anterior horn.

The descending branches run a short course, some of them in the comma tract of the posterior column, and then turn into the grey matter.

The ascending branches of Lissauer's tract arborise around

two limbs, one, which corresponds physiologically to an elongated dendron, communicating with the peripheral sense-organ, and the other, which forms the axon, passing into the cord. These axonic processes enter the cord in two bundles, of which the mesial bundle passes into the *posterior column*, and the lateral into the *marginal tract* of Lissauer.

The processes of both bundles divide on entering the cord into *ascending* and *descending* branches, both of which, as the diagram (Fig. 18) more fully explains, give off collaterals to various



cells in the substantia gelatinosa of Rolando, while those of the posterior column run upwards in the columns of Goll and Burdach, entering the grey matter at various levels, or continuing upwards until they end in the nucleus gracilis and nucleus cuneatus of the medulla.

With the doubtful exception that some of the collaterals may pass by the posterior grey commissure to the posterior horn of the opposite side, *the peripheral neurone is uncrossed*.

2. The **central neurones** are composed of two orders of neurones, one above the other, *and are crossed*. The *second order of neurones* commences partly in the cord and partly in the medulla oblongata.

A. Some of the fibres of the posterior roots arborise, as we have seen, round cells of the posterior horn. The axons of these cells decussate *in the cord* (lower sensory decussation), and pass by the anterior commissure to run upwards in the opposite antero-lateral tract of Gowers. Part of the fibres of this tract goes to the cerebellum, part to the pons, and a third part through the fillet and tegmentum of the crus to the *optic thalamus*. It is this third set of fibres which carries the sensations of *pain and temperature*, and most of the *touch fibres* follow this route.

B. The fibres of the posterior columns which we have traced to the gracile and cuneate nuclei of the medulla arborise there around the cells of the second order of neurones. The axons of these cells cross to the opposite side (*upper sensory decussation*, decus-

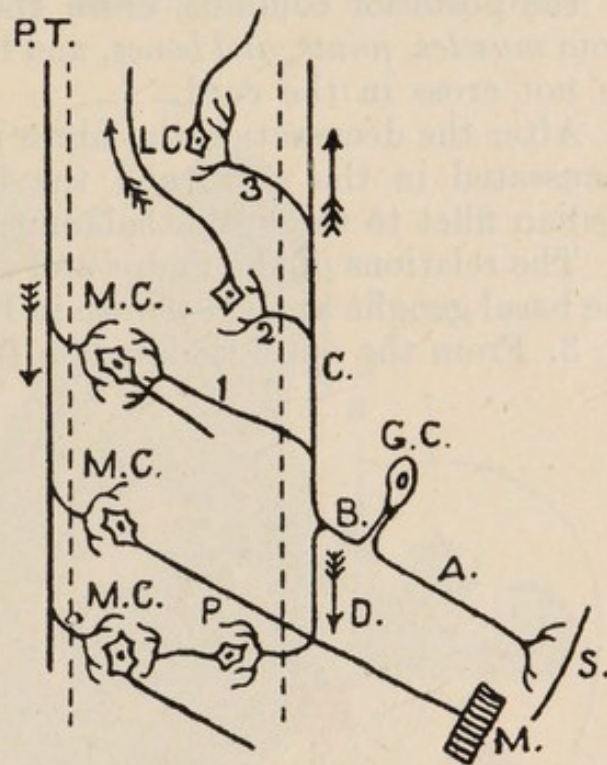


FIG. 18.—CONNECTIONS OF NERVE FIBRES IN THE SPINAL CORD.

S., Skin, from which the fibre A., of a peripheral sensory nerve leads to G.C., the ganglion cell of the posterior root. B., Axon of the ganglion cell, dividing into a short descending branch D., which turns into the grey matter and arborises round a cell P. of the posterior horn; and an ascending branch C., which runs upwards to the medulla oblongata, giving off the following collaterals—1, to a cell of the anterior horn (reflex collateral); 2, to a cell of the posterior horn, the axon of which crosses to the antero-lateral tract of the opposite side; 3, to a cell (L.C.) of Lockhart Clarke's column, the axon of which enters the direct cerebellar tract. P.T., fibre of pyramidal tract, sending collaterals to the motor cells (M.C.) of the anterior horn, the axons of which end in muscle fibres M.

The dotted lines represent the boundaries of the grey matter. (Modified from Schäfer.)



sation of the fillet, Fig. 19) at a higher level than the decussation of the pyramids, and continue upwards in the fillet through the pons and tegmentum of the crus to the optic thalamus. In this upper sensory decussation, the continuation of the tracts of the posterior columns, cross the fibres conveying sensations from *muscles, joints, and bones*, and those of the touch fibres which do not cross in the cord.

After the decussation the fibres join those which have already decussated in the cord, and the two sets run together in the median fillet to the optic thalamus.

The relations of the motor and sensory tracts of the crus with the basal ganglia are well shown in Fig. 20.

3. From the optic thalamus a **third order of neurones** carries

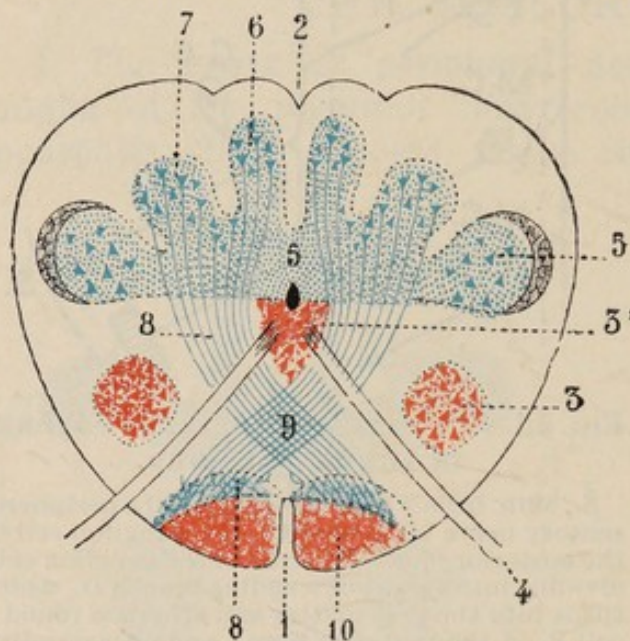


FIG. 19.—SCHEME OF THE STRUCTURE OF THE MEDULLA AT THE LEVEL OF THE SUPERIOR OR SENSORY DECUSSATION (TESTUT).

1, Anterior fissure; 2, posterior fissure; 3, antero-lateral nucleus; 4, hypoglossal; 5, central canal; 6, nucleus gracilis; 7, nucleus cuneatus; 8, nucleus of Rolando capped by the descending root of the fifth nerve; 9, formatio reticularis.

by means of its axons the impressions derived from the second order, through the posterior third of the posterior limb of the internal capsule and the corona radiata, to the cortical sensory centres in the post-central gyrus of the parietal lobe.

Fibres for *co-ordination of muscular movement* ascend in the *direct cerebellar tract* of Flechsig from cells of Clarke's column to the cerebellum, but remain *uncrossed*. Some of the cells of Clarke's column may possibly send axons into the antero-lateral tract.

Fig. 21 illustrates these connections of the posterior root-fibres with the cells of the cord.

**The Internal Capsule** (Fig. 16 and Fig. 20), through which the motor and sensory fibres pass, is the band of white matter between the basal ganglia, its boundaries being—

Externally :— Lenticular nucleus.

Internally :— { Caudate nucleus (anteriorly).  
                  { Optic thalamus (posteriorly).



In the middle of the internal capsule is a bend termed the "genu," or knee; the portion in front of the knee is called the anterior limb, and the portion behind, the posterior limb. The anterior limb contains commissural fibres of various kinds, the genu contains descending fibres passing to the cranial nuclei of

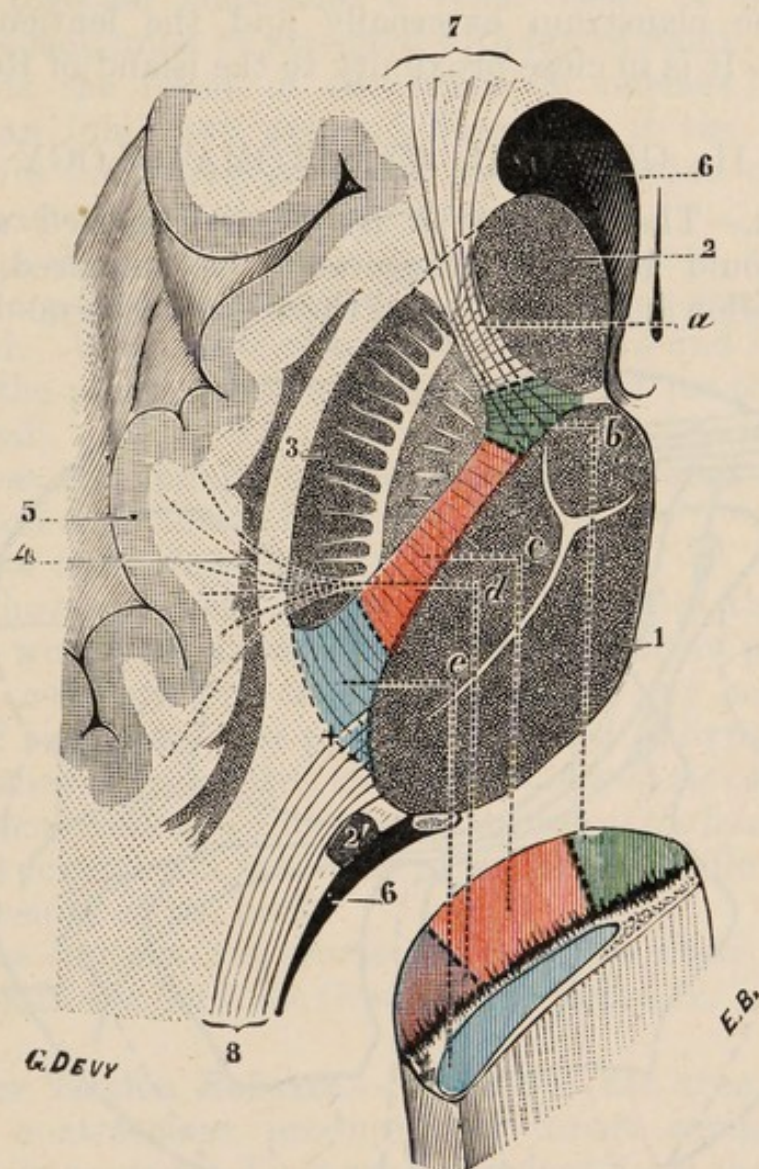


FIG. 20.—SCHEME OF THE CONNECTIONS OF THE CRUS WITH THE BASAL GANGLIA (TESTUT).

1, Optic thalamus; 2, caudate nucleus; 3, lenticular nucleus; 4, claustrum; 5, grey matter of the island of Reil; *a*, fronto-pontine projection system; *b*, genu of internal capsule-cortico. bulbar projection system; *c*, pyramidal projection system; *d*, temporo-pontine projection system; *e*, sensory fibres; 7, frontal fibres; 8, optic radiation.

the opposite side, whilst the *posterior* limb contains, as we have already seen, the *sensory and motor fibres* from the spinal cord.

A lesion of the internal capsule involving the motor fibres must therefore cause motor paralysis of the side of the face and of the limbs, on the opposite side of the body.

In cases that survive the original shock, sensation is not



usually so much affected. It will be readily seen that a lesion, extensive enough to cause both *sensory and motor* paralysis, would probably be fatal in the comatose stage; but hemi-anæsthesia, usually transitory, does occasionally occur.

**The External Capsule** is that portion of the cerebrum lying between the claustrum externally and the lenticular nucleus internally. It is in close proximity to the island of Reil.

## II. GENERAL SYMPTOMATOLOGY

**Reflexes.**—The student is frequently puzzled as to when reflexes should be lost, exaggerated, or impaired, but if he bears the following facts in mind there should be no difficulty.

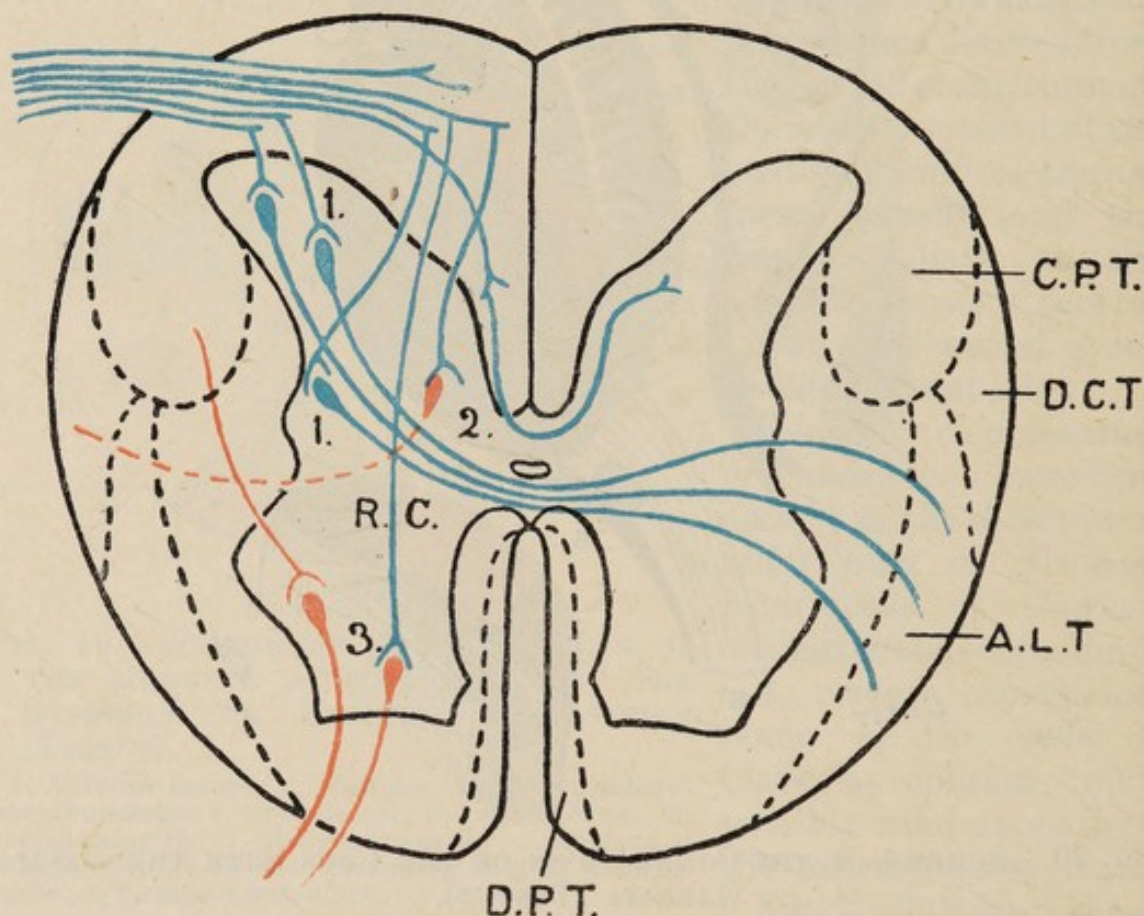


FIG. 21.—SHOWING THE MAIN CONNECTIONS OF THE POSTERIOR ROOT-FIBRES WITH CELLS OF THE GREY MATTER OF THE CORD.

1, Cells of the posterior horn, sending their axons to A.L.T., the antero lateral tract of the opposite side (lower sensory decussation); 2, Cell of Clarke's column, its axon going to the direct cerebellar tract (D.C.T.) of the same side; 3, Motor cells of the anterior cornu, R.C., reflex collateral. Sensory cells and fibres blue, motor red. Clarke's column dotted red line.

A reflex act requires a continuity between an *afferent* nerve and an *efferent* nerve. The parts usually involved are—

1. A receiving surface (as skin).



2. A sensory nerve (afferent fibre), receiving station (cells of posterior ganglion), and transmitting fibres (reflex collateral).
3. A discharging station (cells in anterior horn).
4. A motor nerve (efferent fibre) and end station in muscle.

These together constitute a *reflex loop*. A break in *any* part of this loop must be attended with *loss* of reflex.

What is the effect of the brain on reflexes? The brain exercises an inhibitive action; therefore, if the cord be cut off from its inhibitive influence, as by disease of the upper efferent neurone, reflexes must be *exaggerated*.

Imagine the cord to be built up of segments, each segment giving off a pair of spinal nerves; then take a transverse lesion of the cord. What state of reflexes should we find?

1. At the point of lesion—reflexes are lost (as the reflex loop is destroyed).
2. *Below* the lesion—reflexes are exaggerated (as the inhibitive action of the brain is cut off).
3. *Above* the lesion—reflexes are normal.

Note, however, that at the upper border of the lesion, the dead part would act as an irritant to the healthy portion of the cord, and consequently cause irritation of any sensory nerves coming off that area, thus producing a band of hyperæsthesia.

*This painful zone, taken in conjunction with the condition of the reflexes, often enables an accurate localisation of the lesion to be made.*

The hyperæsthetic zone also explains the girdle pain present in many lesions of the cord.

For the various cutaneous reflexes and their approximate localisation in the cord, see Gowers' Table, p. 432.

**Deep or Tendon Reflexes.**—These are not true reflexes, but muscular contractions produced by direct excitation of the muscle or its tendon. They are dependent upon the tonus of the muscle, which in its turn depends upon the integrity of the reflex arc. Thus, disease affecting the afferent or efferent parts of the loop, or the receiving station in the cord, abolishes them; and they are exaggerated when the cerebral inhibitory action is interfered with.

The *knee-jerk* is present in health. It is obtained by tapping the patellar tendon when the quadriceps femoris is slightly stretched. The patient may cross one leg over the other, or the leg may hang over the edge of the bed, or the thigh may be supported by the observer's hand. It corresponds to the second, third, and fourth lumbar segments of the cord.



The *Achilles-tendon jerk* is obtained by getting the patient to place his leg in a kneeling position on a chair with his foot hanging over the edge, and tapping smartly on the Achilles tendon. It is present in health, and corresponds to the first sacral segment.

*Knee-clonus* is obtained by pulling down the patella as the patient lies upon his back, and delivering a tap upon its upper edge while the tendon is stretched. It is not present in health.

*Ankle-clonus* is obtained by supporting the calf of the patient's leg upon one hand, and thrusting the foot upwards with the other hand placed upon the ball of the toes. It is also absent in health.

In the arm the most important tendon reflexes are the *triceps* tendon jerk, obtained by tapping the tendon above the elbow, and corresponding to the sixth and seventh cervical segments; and the *supinator* jerk, obtained by tapping the lower end of the radius while the forearm is semi-pronated, and corresponding to the fifth cervical segment. These jerks are often absent or very feeble in healthy persons.

**Degenerations.**—When nerve fibres are cut off from their trophic centres, they undergo disintegration, known as secondary degeneration—viz., in the case of sensory fibres, ascending degeneration, and in the case of motor fibres, descending degeneration.

In the case of sensory fibres, the trophic cells are situated peripherally, and the direction of the axon is centripetal. Hence in the cord sensory degeneration starts from below and passes upwards towards the cerebrum. In the motor tracts the reverse is the case, as the trophic cells have a central situation. Degeneration therefore occurs from the cerebrum downwards through the cord, or from the anterior cornu along peripheral nerve fibres.

Such degenerative changes in the lower motor neurone express themselves in an alteration of the electrical reactions of the muscles governed by the affected nervous area, and also by alterations in the reactions of the implicated nerves. These are known as the *reaction of degeneration*, or briefly, R.D. The changes on applying the electrodes to the *muscle* are as follows:—

1. At first, increase of excitability to galvanic and faradic currents; this quickly passes off, and we get—
2. *Decreased* response to faradic, *but increased to galvanic*; next—
3. Decrease to *both* galvanic and faradic.



4. Loss of response to both, in cases where the nervous disease is permanent. In other cases, the response to faradism and to galvanism gradually returns.

These peculiar changes are accompanied by "polar" alterations.

In health we get in response to a minimum current—

1. K.C.C., or kathodal closing contraction, the strongest ;  
next—
2. A.O.C., or anodal opening contraction
3. A.C.C., or anodal closing contraction } equal.
4. K.O.C., or kathodal opening contraction, the weakest.

But in disease—

*A.C.C.*, or anodal closing contraction, may be stronger than the *K.C.C.* (kathodal closing contraction)—*i.e.* a reversed condition to that seen in health.

The main point, however, to remember is, that a *muscle* cut off from its nerve, or supplied by a *diseased* nerve, undergoes—

1. Increase of excitability.
2. Decrease of excitability, failing ultimately to respond to any electrical stimulus.

**Morbid Changes of Degeneration.**—The nerve structure shows—

1. Breaking up of the medullary sheath into myeloid globules.
2. Proliferation of the nuclei under the sheath.
3. Disappearance of the axon, and transformation of the nerve substance into a fibrous cord.

*The muscle structure shows* atrophy of the muscle fibres, which lose their transverse striation and become granular, and great increase of the fibrous tissue.

**Spasm and Rigidity.**—When a motor nerve is irritated, the muscles supplied by that nerve contract. If the irritation be kept up, the contractions are more or less constant, and cause "spasm." Spasmodic muscular contractions may be of two kinds, *tonic*, when the contractions are continuous, and *clonic*, when they are intermittent. Spastic contractions of the muscles of the lower limbs, especially when the patient is falling asleep, and cerebral control is thereby relaxed, are frequent in early degenerative lesions of the motor tract. There also exists in such conditions a permanent hypertonicity of the muscles (the spastic



state), manifested clinically by a peculiar gait (*see* p. 427), by exaggerated reflexes, and by rigidity. When due to irritation of motor nerves, rigidity may take one of several forms. When the motor tracts are injured, let us say by a severe hæmorrhage in the brain, there is not only paralysis, but also rigidity of the limbs. This may come on at once, and pass off in a few hours (*initial* rigidity, due to irritation of the lesion); or more commonly, it comes on after a few days (*early* rigidity) and lasts for a few weeks. It is then due to irritation of the motor fibres by inflammatory products.

Suppose the patient recovers from a fit of apoplexy, with, however, a *resulting hemiplegia*, it will be noted that later a permanent rigidity takes place (*late* rigidity). This has been supposed to be due to the irritation produced by the ensuing secondary descending degeneration. The true explanation is that the rigidity is due to the increased muscular tonus caused by impulses from the subsidiary descending tracts, particularly the vestibulo-spinal, which are left undamaged when the pyramidal fibres are destroyed in the brain. Still later, degenerative changes leading to contraction take place in the muscles themselves.

Rigidity of a similar nature may also occur independently of cerebral disease, when the motor tracts in the cord are affected by sclerosis (lateral sclerosis, disseminated sclerosis).

**Inco-ordination.**—By co-ordination is meant the harmonious action of muscles involved in the carrying out of complicated movements. *Inco-ordination* means failure of this harmony.

The cerebellum is one of the *chief* centres for co-ordinated movements, though there are subsidiary centres. The centres seem to act through afferent impressions derived from—

1. The muscular sense (afferent muscle-nerve fibres in the columns of Goll and Burdach and the direct cerebellar tract of Flechsig).
2. Sight (optic nerves).
3. Auditory organs (semicircular canals).

Lesions interfering with these afferent fibres are attended with more or less inco-ordination. Inco-ordination may, therefore, be due to—

1. Disease of the *centres* rendering them powerless to emit the necessary influence.
2. Disease cutting off the means (afferent fibres) by which the centres are stimulated.



**Paralysis.**—When paralysis is due to a cerebral lesion it is unilateral (*hemiplegia*), and when due to a lesion of the cord it is bilateral (*paraplegia*). When one limb only is affected, the condition is called *monoplegia*. Paralysis may be *spastic* or *flaccid*. In the former variety, due to disease of the upper motor neurone, rigidity of the affected limbs is present, and is attended with exaggerated reflexes and later with contracture; in the latter, in which the lower motor neurone is involved, the muscles are flaccid, *wasting* is marked, R.D. is present, and the deep reflexes disappear. Where the lower motor neurone is not implicated, any atrophy that may exist is slight, and due merely to disuse of the affected limbs. A complete transverse lesion of the cord (transverse myelitis) leads to motor and sensory paralysis of the parts below the lesion, and, in addition, the *urethral* and anal sphincters are usually affected.

**Gait.**—In the various forms of chronic disease of the spinal cord, whether the lesion be confined to the tracts in the cord itself or is more extensive, there are certain peculiarities of gait which help to create a typical picture of the disease. These may, therefore, be briefly characterised in this place:—

1. *The Ataxic Gait.*—In locomotor ataxia, as the patient walks, his feet are separated from each other, his heels are brought down first in a stamping fashion, he watches his feet to correct by sight the imperfect muscular sense, and more power is used than is necessary for progression, so that the feet are, as it were, flung out beyond the line of progress. If he stands with the feet together he tends to sway; if he tries to turn sharply he may reel or fall.

2. *The Spastic Gait.*—In lesions such as spastic paraplegia, while the patient can still walk, he drags the rearward leg slowly forward, the toes scraping the ground. The knee and ankle are very slightly flexed, and hence there may be “circumduction.” There is often strong adductor spasm, so that the feet may cross each other. When the spastic element is prominent, the gait may assume a “hopping” character.

In *disseminated sclerosis* (*q.v.*), besides the spastic gait, there may be tremor on exertion (“intention tremor”).

**Sclerosis.**—This is a term used to express the pathological changes which take place in *chronic degenerative lesions* of the nervous system. The following summary of its features will save repetition in the description of particular diseases.



*Changes in the Grey Matter.*

1. Increase of neuroglia.
2. Atrophy of *proper nerve-cells*.
3. *Thickening of the vessels*, some of which are obliterated.
4. Extravasation of pigment, and small patches of fatty degeneration.

*Changes in the White Matter.*

1. Grey discolouration.
2. Wasting or disappearance of axis-cylinders; absence of medullary sheaths.
3. Great increase of neuroglia.

*The Nerve-roots*, when affected, exhibit similar changes.

**To understand the Diseases of the Spinal Cord**, where many of the chronic degenerative lesions tend to involve one or more clearly defined nervous tracts, it is important to grasp what fibres are interrupted, or what cells are destroyed, by lesions of those tracts. The student should therefore construct for himself such diagrams as those on pp. 415, 416, and mark upon them the sites of various lesions in relation to the particular tracts involved. He will then be able to reason what must happen in any given instance.

### III. DISEASES OF THE SPINAL CORD AND ITS MEMBRANES

It is no longer possible rigidly to distinguish between diseases of the cord, diseases of the brain, and diseases of the peripheral nerves, although for clinical purposes and in connection with acute and focal lesions the distinction may still be roughly preserved. But the morbid changes of chronic degenerative lesions affect one or other neurone *as a whole*, and thus the anatomical seat of such diseases occupies both the brain and cord, or both the cord and the peripheral nerves at the same time. Such degenerative lesions, known as "*system diseases*," although it may be more convenient to describe them according as their symptoms are mainly cerebral, spinal, or peripheral, are more rationally classified, as in the following scheme of Aldren Turner's, according to the neuronie system primarily affected.

#### I. *Diseases of the Upper Efferent Neurone* (pyramidal system).

General paralysis (spastic form), lateral sclerosis, old-standing hemiplegia, spastic paraplegia and diplegia of children, disseminated sclerosis (paraplegic form).



II. *Diseases of the Lower Efferent Neurone.*

Local and multiple neuritis; acute anterior poliomyelitis, acute bulbar paralysis, acute ophthalmoplegia; Landry's paralysis; chronic anterior poliomyelitis, chronic bulbar paralysis, ophthalmoplegia externa.

III. *Diseases affecting both Efferent Neurones simultaneously.*

Amyotrophic lateral sclerosis, general paralysis (advanced stage).

IV. *Diseases of the Lowest Afferent Neurone* (posterior ganglionic system).

Locomotor ataxy, general paralysis (ataxic form).

V. *Diseases of Afferent and Efferent Neurones simultaneously.*

Friedreich's ataxia, ataxic paraplegia, disseminated sclerosis (advanced stage).

## SPINAL MENINGITIS

Inflammation of the meninges may be acute or chronic, diffuse or localised. When the dura mater is principally affected, the disease is termed pachymeningitis; when the pia mater is most involved, leptomeningitis. This distinction is convenient when we have to deal with a *slow* process, as in *chronic meningitis*; but in acute inflammation, though one or other membrane may be primarily affected, the disease quickly spreads, and involves the whole three membranes equally.

## I. ACUTE SPINAL MENINGITIS

The process may begin in the cellular tissue *outside* the dura mater, *i.e.* external meningitis; or within the sheath, internal meningitis.

**External Meningitis** is most commonly local, but is sometimes diffuse. Internal meningitis (leptomeningitis), although it begins in the pia arachnoid, tends also to involve the dura.

The *diffuse form of external meningitis* differs from leptomeningitis mainly in its causes (extension of contiguous disease, especially caries or other disease of the vertebræ), and very little in its symptoms. It is to be distinguished by evidence of its arising secondarily to such conditions.

The seat of the *local form* can be determined by a study of the distribution of the painful or anæsthetic areas, and the behaviour of the reflexes. The table of Gowers (p. 432) will be of assistance in this localisation.

**Internal Meningitis (Leptomeningitis)** may, although rarely, be due to extension of an external or of a cerebral meningitis. It forms part of the changes of epidemic cerebro-spinal fever, and occasionally of tuberculous cerebral meningitis. It also arises in connection with other acute infections, such as septi-



cæmia and especially *pneumonia*. It may follow injuries to the spine, or, in rare instances, exposure to cold. Syphilis of the cord causes a meningo-myelitis, rather than an acute meningitis; but it may give rise to a *chronic* localised meningitis (*v. infra*).

The organisms found are those of the associated disease.

*Morbid Anatomy.*—The meninges are widely involved, and the internal membranes suffer early. The arachnoid is frequently wholly disorganised, the pia deeply injected, and the dura bulged outwards from the accumulation of turbid cerebro-spinal fluid or even pus. In severe cases, the substance of the cord may be softened; and the nerve-roots are often swollen.

*Symptoms* set in with rigor and pyrexia. There are severe and constant pain along the spine, increased by pressure, and paroxysmal pain in the distribution of the implicated nerve-roots. The muscles of the back are rigid; there may be retraction of the head, and sometimes opisthotonos; and there may be cramps in the muscles of the abdomen or limbs. Constipation and retention of urine are frequent. There is marked cutaneous hyperæsthesia, and Kernig's sign (*see p. 52*) may be present. Later, the signs of irritation yield to those of paralysis, and the reflexes, at first exaggerated, are now lost. Death may occur from bedsores or secondary renal affections, or recovery may follow, either complete or more commonly partial, some degree of paralysis or anæsthesia being left behind.

*Diagnosis* may be much aided by lumbar puncture, the nature of the meningitis being determined by bacteriological examination of the fluid.

*Treatment* consists in absolute rest in a darkened room, morphia or chloral and bromide for the pain and spasm, local depletion by dry cupping or leeching, saline purgation, and the promotion of diaphoresis by warm baths and packs. Gowers advises, in addition, inunction of oleate of mercury until the gums are slightly affected. The spinal ice-bag may help to relieve the spasm.

## II. CHRONIC INTERNAL MENINGITIS

Sometimes a sequel to the acute affection, and therefore dependent on the same causes, this disease may also be chronic from the beginning. In such cases it may follow injury or disease of the bones, or form an extension of chronic diseases of the cord. Alcoholism, syphilis, and repeated exposure to cold and wet are also among the causes.



**Morbid Anatomy.**—In *chronic leptomeningitis* the changes vary from a mere thickening and cloudiness of the membrane with *increase* of cerebro-spinal fluid, to obliteration of the sub-arachnoidal space by complete organisation of the inflammatory lymph, which mats the pia mater and dura mater together. The *nerve-roots*, at first swollen and injected, become fibrous and atrophied. The *spinal cord* suffers in proportion to—

1. The amount of compression from without.
2. The amount of thickening of the processes of the pia mater which *run into* the cord.

*Hypertrophic internal pachymeningitis* affects the inner surface of the dura, usually in the cervical region, and causes great thickening, with pressure upon the nerve-roots and the cord, which may be softened.

**Symptoms** are those of irritation, followed by destruction of the nerve-roots, and of damage to the cord. At first from irritation of sensory fibres, we have—

1. Pain in the back, increased on pressure, and with some stiffness.
2. Radiating or excentric paroxysmal pains in the distribution of the nerve-roots.

Later, when the roots are destroyed, anæsthesia with weakness, wasting of muscles, R.D., and loss of reflexes replace the pain; and ultimately the parts below the lesion are paralysed from implication of the cord.

**Treatment.**—Rest, the prone position, sedatives, and *counter-irritation* are the usual measures. If syphilis is the cause, the Wassermann reaction will be positive in the blood or cerebro-spinal fluid, and the latter may show a considerable leucocytosis. In such cases specific treatment should be adopted; and mercurial inunction is useful, apart from syphilis. When the disease has begun outside the dura, laminectomy may relieve the pressure on the cord.

## MENINGEAL HÆMORRHAGE

### (HÆMATORRHACHIS)

Like meningitis, this condition may be extradural or intradural. It is frequently due to injury, and may occur in severe convulsions, and also in hæmorrhagic disorders. Extradural hæmorrhage may be caused by the bursting of an aortic aneurysm which has eroded the vertebræ.



**GOWERS' TABLE.**—Showing the approximate relation to the spinal nerves of the various motor, sensory, and reflex functions of the spinal cord.

MOTOR. (Nerves).	MOTOR.	SENSORY.	REFLEX.
St.-mastoid, Upper neck muscles, Upper part of Trapezius	C 1 } Small rotators of head.	1 } Scalp.	1
	2 } Depressors of hyoid.	2 } Neck and upper part of chest.	2
	3 } Lev. angulæ scapulæ.	3 } Shoulder.	3
	4 } Diaphragm.	4 } Arm, outer side.	4
	5 } Serratus. Flex. of elbow. Supinators.	5 } Radial side, forearm and hand; thumb.	5
Lower neck muscles, Middle part of Trapezius	6 } Ext. wrist and fingers.	6 } Arm, inner side, ulnar side of forearm and hand; tips of fingers.	6
	7 } Ext. of elbow. Flex. wrist and fingers. Pronators.	7 } Scapular.	7
	8 } Muscles of hand.	8 } Abdomen (Umbilicus, tenth).	8
	D 1	1	1
	2	2	2
Lower part of Trapezius and dorsal muscles	3	3	3
	4	4	4
	5	5	5
	6 } Intercostals.	6 } Front of thorax.	6
	7 } Abdominal muscles.	7 } Eusiform area.	7
Lumbar muscles	8	8	8
	9	9	9
	10	10	10
	11	11	11
	12	12	12
Peroneus, l. Flex. of ankle, Ext. of ankle.	L 1	1 } Buttock, upper part.	1
	2 } Cremaster. Flexors of hip.	2 } Groin and scrotum (front).	2
	3 } Extensors of knee. Adductors of hip.	3 } Thigh. { outer side.	3
	4 } Ext. and abduct. of hip.	4 } { front.	4
	5 } Flexors of knee.	5 } { inner side.	5
S 1	1 } Intrinsic muscles of foot.	1 } Leg, inner side.	1
	2 } Perineal and anal muscles.	2 } Buttock, lower part.	2
	3 } Perineal and anal muscles.	3 } Back of thigh.	3
	4 } Perineal and anal muscles.	4 } Leg and foot } except inner part.	4
	5 } Co.	5 } Perinæum and anus.	5
		co } Skin from coccyx to anus.	co



**Symptoms** (pain in the back and in the course of the nerves, with spasm, sometimes retention, etc.) are very similar to those of meningitis, and are due to irritation of the nerve-roots. They are to be distinguished from meningitis by the absolute *suddenness* of their onset, and by the absence, at first, of *fever*. Later the effused blood sets up a secondary meningitis, and fever follows. When the hæmorrhage is into the *substance of the cord* (hæmatomyelia), the symptoms are those of sudden spinal paralysis, and pain is not prominent.

**Treatment** consists in absolute rest in the prone posture, venesection, leeches or cupping, ergotin, and morphia for the pain. Where death threatens in spite of these steps, or symptoms of compression of the cord are prominent, laminectomy may be performed.

## ACUTE MYELITIS

A localised or extensive inflammation of the cord substance, affecting either the grey matter only, or grey and white together. The disease may be acute or subacute, the difference consisting largely in the rapidity of onset. The so-called "chronic" forms are really more of the nature of degenerations than of inflammation.

**Etiology.**—Myelitis is most common in the male. Its main causes are *infection* and *intoxication*. The disease frequently develops after acute infections of the most varied kind; and it may itself be a primary infection, the organisms reaching the cord by the blood or lymph. In other cases it may be secondary to thrombotic changes in the vessels, usually due to syphilitic endarteritis. Some cases directly follow chill, others injury or disease of the vertebræ, and general debility predisposes to an attack.

**Morbid Anatomy.**—The cord is swollen and softened at the affected part, and may be hæmorrhagic. The pia mater is injected, and the meninges occasionally share in the inflammation (*meningo-myelitis*). The lower dorsal region is most frequently attacked.

**Symptoms.**—If meningitis is also present, there may be symptoms of irritation of nerve-roots. Usually paralytic phenomena are more prominent. The onset is rapid, a feeling of heaviness in the legs being succeeded in a few hours, or in the course of a day or two, by complete paraplegia. There is usually fever, and, in the diffuse form, hyperpyrexia may precede death. In the case of myelitis affecting the lower dorsal region, the



following are the symptoms indicative of a total transverse lesion *after paralysis has become established*. Above the lesion there are no disturbances of sensation, movement, or reflexes, but at a point corresponding to the upper margin of the lesion there is a zone of hyperæsthesia, often accompanied by "girdle sensation." Below the level of the lesion there is complete paralysis (paraplegia) of both limbs, which in the early stages may be flaccid, but rapidly become rigid; there is anæsthesia, complete or partial, to all forms of sensation; the reflexes, both superficial and deep, are exaggerated, after, it may be, a temporary absence; and there is paralysis of the bladder and rectum. There is no wasting of muscles. Where anæsthesia is complete, trophic and vasomotor disturbances are often present; crops of herpes may appear, and bedsores are frequent. In the later stages descending degeneration affects the pyramidal tracts; rigidity becomes more pronounced, and painful contractures may be a distressing symptom.

If the lesion affects the cervical or lumbar enlargement, there is rapid wasting of the muscles of the arms or legs following destruction of the cells of the anterior horns. Involvement of the phrenic nerves, in the former case, will lead to rapid death from respiratory paralysis. Occasionally the myelitis is diffuse, affecting the grey matter in a great part of its length, and here, too, rapid wasting of muscles may be found. In some cases it takes an ascending form, beginning with paralysis, anæsthesia, and atrophy of the lower limbs, involving the sphincters, causing bedsores, and implicating the muscles of the trunk and arms.

The condition of the sphincters varies with the seat of the lesion. The *rectal centre* may be below the lesion or may be involved in it, and there follows at first constipation, from paralysis of the bowel; later, incontinence, due, if the disease is above the centre, to involuntary action of the sphincter, if it involves the centre, to inaction. Similarly with the *vesical centre*; when it is involved, there is incontinence from the first, when it is below the lesion, there is at first retention, and afterwards an overflow incontinence. Priapism may occur as a reflex phenomenon.

In consequence of the increased muscular tonus and exaggerated reflex response, the slightest touch of the bedclothes, the passage of a catheter, etc., may cause severe spasm. The temperature of the affected limbs is often raised at first, but afterwards it falls below that of the healthy parts.

The course is variable. In cases that recover, sensation returns first, motor power much later, and usually imperfectly.



The disease may extend upwards, causing respiratory paralysis and death, or death may follow bedsores or ascending nephritis.

**Syphilitic Meningo-Myelitis** is usually of slower onset, and takes the form of an incomplete transverse myelitis associated with pain in the distribution of the affected nerve-roots, and also with partial anæsthesia from implication of the posterior columns. In some instances the onset is rapid, and the symptoms those of acute myelitis.

**Treatment.**—Absolute rest, if possible on the side or in the prone position; leeches, dry cupping, or fomentations to the spine; mercurial inunction. A water-bed should be used. Prevent bedsores, relieve urinary retention. Later, tonics, massage, and electricity to maintain the muscular nutrition. In syphilitic cases, vigorous specific treatment, mercury being preferable to salvarsan.

## SYRINGOMYELIA

A disease due to a slowly progressive gliomatous new growth about the central canal of the cord, and leading, by breaking down of tissue, to the formation of cavities, which may extend the whole length of the cord, but usually occupy the cervical and dorsal regions. The cavities give rise to pressure upon the grey matter, and later to ascending and descending degenerations. The cervical part of the cord is much enlarged. In a few cases the condition is apparently due to a congenital defect of development, leading to a progressive dilatation of the central canal.

The disease usually affects males, and begins before the age of thirty. Its chief symptoms are *dissociated anæsthesia* and *muscular atrophy*. (1) The anæsthesia is to sensations of temperature and pain, while touch is unaffected. Cigarette smokers often burn their finger-tips. The neck, upper part of the trunk, and the upper limbs are most affected, but the disease may spread to the lower limbs, and touch may also suffer later on. (2) Muscular atrophy (pressure on the anterior cornua) begins in the small muscles of the hand, and slowly spreads up the arms to the trunk. Other *trophic symptoms* are painless whitlows, necrosis of the finger bones, and affections of the joints. Secondary degenerations may in time lead to spastic paraplegia or ataxia, and if the disease spreads



upwards, laryngeal paralysis, dysphagia, or nystagmus may occur.

The course is very chronic, and the sole treatment consists in attention to the general health and special symptoms.

## TUMOURS OF THE CORD

The most common varieties are, in the membranes, sarcomata, gummata, and myxomata; in the substance of the cord, solitary tubercle and gummata, though many other tumours may be found. The symptoms are due to irritation of nerve-roots and to pressure on the cord. Pain in the course of the affected nerves is persistent, and may be agonising; it is usually *at first unilateral*. Rigidity may also be present; and paralysis is constant. It takes the form of paraplegia, at first more pronounced on one side, and of gradual onset and slow course. Anæsthesia is at first partial, but later becomes complete. The muscles supplied by the damaged nerve-roots waste. The site of the tumour can be determined with the help of Gowers' Table (p. 432).

The **treatment** varies with the cause. A few cases are open to surgery; tuberculous cases require the treatment for tuberculosis in general; and energetic specific treatment is necessary for gummata.

## SYSTEM DISEASES OF THE CORD. ACUTE ANTERIOR POLIOMYELITIS

### (INFANTILE PARALYSIS)

An acute infection, of which the morbid changes principally affect the lower efferent neurone, occurring sporadically in children and in occasional epidemics in children and adults.

**Etiology.**—Sporadic cases are most common in childhood, usually before the age of four. Chills and falls have coincided with the onset, and cases are most numerous in the hot months. The epidemic form attacks adults and children, and is often fatal. The virus is found in the spinal cord and brain, the mesenteric lymph-glands, and the mucous membranes of the naso-pharynx and gastro-intestinal tract and their secretions. The disease is communicable through the nasal secretion, direct and apparently by carriers, and the nasal mucous membrane would seem to be its portal of entry. It can be produced in monkeys by injecting



emulsions of the cord. An organism has recently been described by Flexner and Noguchi. It grows anaërobically in ascitic fluid, to which a fragment of sterile fresh tissue is added, and it takes the form of minute "globoid bodies" measuring from  $0.15\ \mu$  to  $0.3\ \mu$  in diameter, and arranged in pairs, chains, or masses. Inoculation into monkeys reproduces the features of the disease. The virus reaches the cord through the lymphatics, not the blood, and the predominance of vascular changes is explained by the fact that the vessels of the central nervous system are surrounded by lymph-sheaths. There is evidence that in some instances the stable-fly (*Stomoxys calcitrans*) may act as carrier. The incubation period is probably from one to fourteen days.

**Morbid Anatomy.**—The first notable changes are in the *vessels* of the anterior horn (anterior spinal artery), which are congested, distended, and surrounded by small-celled infiltration. Thrombosis is common, but not necessarily present. Secondary interstitial changes take place in the grey matter, and its multipolar cells undergo cloudy swelling and ultimate destruction. Degenerative changes can be traced into the anterior roots. Later, the motor nerve trunks show marked change, the fibres being smaller and fewer in number. The neuroglia becomes increased, and the *anterior horn* as a whole is *sclerosed* and shrunken (see Sclerosis, p. 427). The *muscles* are pale and flabby; atrophy begins early and is well marked; and microscopically, they show the changes already referred to (p. 425), as the result of destruction of their trophic centre.

In the epidemic form the lesions are more extensive. The posterior horn may suffer, the white matter may be implicated, and there may be vascular irritation and cellular infiltration in the higher centres, but the *ganglion cells* of the medulla, pons, and optic thalamus usually escape.

Flexner describes extensive hyperplasia of lymphoid tissues, and necrosis of small groups of liver cells.

**Symptoms.**—We have here to do with an interference with the reflex loop, destruction of motor cells, and loss of motor trophic influence. The symptoms must therefore be absence of reflexes, paralysis, and atrophy; the extent and severity of these symptoms will vary with the extent of the lesion. The onset is sudden, and after two or three days of pain in the limbs, feverishness, and sometimes vomiting or convulsions, the paralysis becomes established. It may be unilateral or bilateral, and is at first widespread, affecting usually the *lower* limbs, but all four limbs or only one, or only *a group of muscles*



may be implicated. In a short time the paralysis passes away, except from a group or groups of muscles in which atrophy, loss of reflexes, and reaction of degeneration (p. ), quickly supervene. There is no rigidity or spasm, and pain may or may not be present. Note that muscle wasting may be concealed by fat.

*The bladder and rectum usually escape.*

In the epidemic form, the average death-rate is about eight per cent. The sporadic form ends in recovery, but some permanent paralysis is generally left behind. The following are among the permanent results :—

1. Defective nutrition of the bones, leading to arrest of growth.

2. Permanent deformities produced by—

- (1) Want of antagonism or unresisted contraction.
- (2) Extension due to the weight of the foot, and maintained by contraction of the unopposed gastrocnemius (talipes equinus).
- (3) Curvature of the spine due to shortening of one leg.

In exceptionally favourable cases there may be a complete recovery. In a few instances the bulbar nuclei may be affected, in others the paralysis may assume an ascending type. Both these forms are of unfavourable omen.

**Treatment.**—Rest, warmth to the affected part of the spine, fever diet, warm baths, and laxatives. Urotropin, which can be found after administration in the cerebro-spinal fluid, may be given for its bactericidal effect. Later, strychnine, cod-liver oil, lacto-phosphate of iron and lime, electricity, and massage. Mechanical appliances may be required to prevent deformities, and surgical measures to correct them. No further benefit from electricity is to be looked for after a year, though the increased movements of the patient may lead to slow growth of the muscles. During the first weeks of the disease patients should be isolated, and all secretions and excretions should be disinfected.

With the exception of the rare *Landry's paralysis*—an acute ascending paralysis beginning in the legs, rapidly involving the trunk, diaphragm, and arms, ending fatally, and probably due to a toxic affection of the lower motor neurone—the remaining



system diseases of the cord are chronic. They may be classified as follows (Ormerod):—

1. Degeneration of the Afferent Neurones: Posterior Sclerosis; *Locomotor Ataxy*.
2. Degeneration of the upper Efferent Neurones: Lateral Sclerosis; *Primary Spastic Paraplegia*.
3. Combined Degeneration of Afferent and Efferent Neurones: Posterolateral Sclerosis; (1) *Ataxic Paraplegia*; (2) *Hereditary Ataxia*.
4. Degeneration of the lower Efferent Neurones: Progressive Muscular Atrophies of Spinal Origin. (1) *Chronic Anterior Poliomyelitis* or *Progressive Muscular Atrophy*, or (2) combined with Degeneration of the upper Efferent Neurone; *Amyotrophic Lateral Sclerosis*.

We shall first consider those diseases in which the degeneration is confined to one system.

## LOCOMOTOR ATAXIA

(TABES DORSALIS)

**Definition.**—A degenerative disease of the lower afferent neurone, characterised by an indefinite onset, and a chronic but progressive course, and attended with marked symptoms of inco-ordination, trophic changes, and disturbances of special sense.

**Etiology.**—The disease is most common in men between the ages of twenty and fifty. It may occur before the age of twenty (juvenile tabes), chiefly in congenital syphilitics. Sexual excesses, long-continued exposure, and injury may act as determining causes, but syphilis is the essential factor in the etiology. Until quite recently it was taught that locomotor ataxia was a *para-syphilitic* disease, i.e. that the syphilitic toxæmia led to a change in the structures of the spinal cord, not directly syphilitic, but favouring degenerative change. But the application of the Wassermann reaction to the cerebro-spinal fluid, and still more emphatically the discovery of spirochætes in the affected nervous tissues, have shown that the disease is directly due to syphilis in the vast majority of cases, and must be regarded as a parenchymatous form of syphilis of the nervous system. It is a late manifestation, the interval between infection and the onset of symptoms being usually from five to fifteen years.

**Morbid Anatomy.**—The changes are those of sclerosis (*see* p. 427) affecting—

1. The posterior nerve-roots and posterior root ganglia. The primary degeneration is in the nerve fibres, and the thickening of the neuroglia is secondary to it.



Sensory fibres of the peripheral nerves may also degenerate. Some degree of thickening of the meninges is frequently present.

2. The posterior columns, beginning in Burdach's and affecting secondarily the column of Goll. Lissauer's tract often suffers.

The disease begins, as a rule, in the lower levels of the cord, and extends upwards. Thus, below, the whole of the posterior columns are affected, while above, the lesion may be limited to the column of Goll.

In advanced cases, the cells of Clarke's column and the fibres of the ascending antero-lateral and direct cerebellar tracts may be implicated. *In the brain*, the most common change is optic nerve atrophy, but the nuclei of other cranial nerves may also be affected.

Infection of the cord probably takes place by the lymph stream ascending along the peripheral nerves and nerve-roots towards the central nervous system.

**Symptoms.**—Consideration of the seat of the morbid changes will show that while the symptoms may vary much in individual cases, sensory changes, inco-ordination, and changes in the visual apparatus must be common to all.

For descriptive purposes the disease may be divided into three stages—

1. Pre-ataxic.
2. Ataxic (stage of inco-ordination).
3. Paralytic.

### PRE-ATAXIC STAGE

The onset of symptoms is often insidious. Taking a typical case, the following symptoms are to be found :—

1. *Changes due to irritation of sensory roots*—

- (1) Lightning pains over the body: hot, burning, and tingling in character; lasting a few seconds; and usually affecting the lower limbs.
- (2) Girdle pains (from the upper margin of sclerosis), the patient complaining of constriction, as if an iron band were around him.
- (3) Paræsthesiæ, such as numbness of the feet, may occur, but are often a later symptom.

2. Early loss of knee-jerks and of Achilles-tendon jerks. The latter may be absent while the knee-jerks are still preserved.



### 3. Symptoms in connection with the optical apparatus—

- (1) Argyll-Robertson pupil (reflex iridoplegia). The pupil fails to react to light, but accommodation remains unaffected.
- (2) Ptosis, double or single.
- (3) Primary optic atrophy.
- (4) Paralysis of ocular muscles, causing diplopia.
- (5) Extreme contraction of the pupils (myosis).

May occur early or late.

4. Priapism is occasionally present in the early stages; *later, sexual desire is abolished.*

5. Not infrequently imperfect control of micturition, or retention.

It should be noted that the early appearance of optic atrophy means late development or absence of ataxia. The ptosis and other ocular paralyses are often transient.

### ATAXIC STAGE

Sometimes the first sign of inco-ordination is tumbling forward into the basin on closing the eyes during the morning wash, while there is no unsteadiness when the eyes are open. Difficulty in walking in an imperfect light may follow, and once begun, the inco-ordination often rapidly develops into the characteristic "Ataxic gait." The patient feels and is unsteady on turning round or standing with his eyes shut, and fails to walk on a straight line, etc. (*See Gait, p. 427.*)

*Romberg's sign* is the usual test for ataxia. If the patient is made to stand with his feet together (heels and toes), and to close his eyes, he sways from side to side, and may fall if not supported.

Anæsthesia of the soles of the feet soon comes on. There is complaint of numbness, or of a sensation of walking on wool or indiarubber. The anæsthesia may be tactile, thermal, muscular, or painful. Thus the patient may fail to distinguish with his feet the difference between a hot and cold body, or between heavy and light weights, provided they are similar in appearance. *Delayed sensation* is common.

Ataxia may affect the hands as well as the feet, making it difficult to unfasten the clothing, etc.

It should be noted particularly that the muscular *power* is not diminished, for the sufferer can resist movement or push away a heavy weight. There is, however, a condition of muscular



*hypotonus*, permitting an increased range of movement of the joints which may lead to subluxation.

Often at this period occur peculiar visceral disturbances, termed crises. The best-known are—

1. Gastric crisis—intense epigastric pain, hyperacidity, and vomiting.
2. Laryngeal crisis—noisy stridulous breathing, with great dyspnoea. This is, however, rare.
3. Vesical crisis—severe renal and vesical pain, often with dysuria.
4. Rectal crisis—tenesmus, etc.

The other most marked changes are the so-called “trophic” alterations:—

1. The skin becomes dry, or shiny and glossy with absence of hair; nails crack, etc.
2. *Joints*.—Charcot’s disease (tabetic arthropathy). The cartilages are eroded, the ends of the bones wasted, and the ligaments ossified. There may or may not be effusion into the joint.
3. Perforating ulcer of the foot.
4. Brittleness of bones, ulceration of cartilage, etc.

### PARALYTIC STAGE

The ataxia increases to such a degree that the patient can neither walk nor stand. Though real paraplegia is absent, he is emaciated and weak, and ultimately becomes bedridden, and liable to grave intercurrent diseases. Hemiplegia, pneumonia, or gangrene, etc., ushers in a fatal ending. Vesical troubles are aggravated; cystitis may set in, and end in an ascending pyelonephritis.

Some cases run an extremely lengthened, others a more rapid course. There is no distinct margin between the stages, though for descriptive purposes it is convenient to separate them. In many cases the disease becomes apparently arrested before the third stage is reached. When optic atrophy is an early symptom, ataxia often develops late or imperfectly.

**Treatment.**—The recent demonstration of the syphilitic nature of locomotor ataxy tends to strengthen the position of those who advocate specific therapy in its treatment. But even the most modern methods of administering salvarsan (intrathecal injection of salvarsanised serum) appear at best to have only a temporary effect, and to show little advantage over efficient



mercurial inunction. The best cases for specific therapy are those in which the ataxia has rapidly followed the initial lesion (two years or so), and where the case is seen early. Other methods of treatment are largely symptomatic. The general health must be maintained by tonics, especially iron; diet should be nutritious; and rest, or exercise stopping short of fatigue, must be ordered according to circumstances. Lightning pains may be treated with antipyrin, phenacetin, or aspirin; chloride of aluminium, three to four grains (grm. 0.2-0.3) thrice daily may be given where these fail, and morphia is often necessary. Similar treatment must be used in the various crises, and in the gastric form gastric sedatives may also have some effect. Where they make life intolerable, division of the appropriate posterior nerve-roots has been practised with some success. For the ataxy the best treatment is a systematic re-education of the muscles under the control of sight, on the system introduced by Fränkel, beginning with the simplest exercises and movements, and passing gradually to the more complex. Massage is also of considerable use.

## PRIMARY SPASTIC PARAPLEGIA

### (PRIMARY LATERAL SCLEROSIS)

A paralysis attended with spasm and rigidity, resulting from sclerosis of the anterior pyramidal and crossed pyramidal tracts (upper efferent neurone).

Let us consider what we must expect from interruption of these two tracts.

1. *The brain being cut off from the motor nerves, two conditions are brought about:—*

(1) *Voluntary* motion must be imperfect, according to the extent of the lesion.

(2) Reflexes must also be exaggerated, as they are cut off from the inhibitive influence of the brain, but the reflex loops are not interfered with.

2. *The motor tracts, being cut off from the first trophic realm in the cortex*, will undergo secondary degeneration. The uncontrolled impulses transmitted by the vestibulo-spinal (descending cerebellar) tract will cause increased muscular tonus, and hence *spasm and rigidity*.

3. The anterior horn not being affected, there will be no interference with the second trophic realm, and consequently no atrophy of muscles.



**Etiology.**—The disease is comparatively rare, but is most common between the ages of twenty and forty. It is possibly due to nutritional changes in the motor cells of the cerebral cortex, leading to descending degeneration, but the cause of these changes is unknown. Nervous heredity, concussion of the spine, exposure to cold and wet, and possibly influenza are predisposing causes. Syphilis has been present in a small minority of cases. Spastic paraplegia is sometimes congenital, owing to bilateral meningeal hæmorrhage over the central convolutions following injury during birth (*see Cerebral Palsies of Infancy*). A *family* or *hereditary* form is also known.

**Morbid Anatomy.**—Unless other tracts are implicated, the disease does not cause death. Changes limited to the upper efferent neurone are therefore seldom found.

**Symptoms.**—

1. Weakness and stiffness of the lower limbs.
2. Exaggerated knee-jerk, and presence of ankle clonus.

*Babinski's sign* is generally present, *i.e.* when the sole of the foot is stroked by a pointed instrument, the toes, especially the great toe, are *over-extended* towards the dorsum of the foot, and abducted one from the other.

3. Spasm and rigidity, causing forcible *adduction* and extension of the limbs, and rendering the gait characteristic. (*See Gait*, p. 427.)

The rigidity is nearly continuous, and when relaxations take place, the slightest stimulus causes spasm.

Though the legs are the limbs usually attacked, still the muscles of the trunk, and the arms occasionally, may be first involved.

The disease runs an extremely chronic course, as, until it becomes widely spread, the muscles remain plump, sensation unaffected, and the sphincters unimpaired. Finally, there may be complete paralysis of the affected parts, or the lesion may extend to other tracts of the cord.

**Diagnosis.**—The *syphilitic spinal paralysis* of Erb presents similar symptoms, but other tracts are involved at the same time; the bladder suffers, and there may be disordered sensation. For diagnosis from other spinal diseases, *see Table*, p. 451. It should be remembered that similar symptoms may be secondary



to disease or injury of the vertebræ, or to myelitis; and that many cases beginning as spastic paraplegia develop other symptoms of disseminated sclerosis at a later stage.

**Treatment.**—Spastic paraplegia is to be treated mainly by general tonics such as arsenic and iron. Strychnine is unsuitable because it tends to increase spasm and rigidity. If syphilis is suspected, specific treatment may be employed. Massage and passive movements are valuable in the treatment of rigidity, and warm baths also promote relaxation of spasm. Electricity applied to the limbs tends to increase spasm, but galvanism to the spine may be of benefit. Excess of every kind must be avoided.

## PROGRESSIVE MUSCULAR ATROPHY

Progressive muscular atrophy, sometimes called chronic anterior poliomyelitis, is *the* example of a *chronic* degeneration of the anterior grey horn and motor roots (lower efferent neurone). The site of the lesion leads us to expect abolition of reflexes, atrophy, and deficient muscular power; and these are the principal symptoms. But in many cases, the changes come to implicate the upper as well as the lower neurone, and the disease then becomes identical with amyotrophic lateral sclerosis. It is homologous with bulbar paralysis, in which the lower efferent neurone (nuclei of cranial nerves) is affected at a higher level.

**Etiology.**—Males are more often affected than females. The disease is one of adult life. It is often associated with exposure to cold and wet, but is not limited to the labouring classes. Often no cause can be found.

**Morbid Anatomy.**—Sclerosis of the anterior horn, anterior roots and nerve trunks, with changes in groups of muscles.

1. The anterior grey horns are pale, but not much altered in shape.
2. Great increase of neuroglia.
3. Obliteration of nerve cells.
4. Later, in many instances, sclerosis of the pyramidal tracts.

*Anterior Nerve roots* are markedly atrophied.

*Nerve Trunks.*—Changes are not so marked as in the roots, for the sensory fibres in the mixed nerve trunk are unaffected, and frequently *some* of the motor fibres escape.



**Muscles.**—Pale and flabby, as in acute anterior poliomyelitis. Fatty and granular degenerations are also present.

**Symptoms.**—The disease usually begins in one or both of the upper extremities with atrophy of—

1. Thenar and hypothenar eminences.
2. Interossei.
3. Forearm muscles, and those of the shoulder.

Paralysis of the interossei, with unopposed action of their opponents, produces the claw-like hand (*main en griffe*)—i.e. the first phalanges are *hyper-extended*, while the middle and distal phalanges are flexed on the first. The muscles of the back are early affected, but the upper third of the trapezius escapes until quite late. After this, the extension is extremely gradual, and years may pass before both arms, both legs, intercostals, or diaphragm are attacked. The legs are usually affected late, but the disease may commence in them. The affected muscles generally exhibit the peculiar *fibrillary twitchings* of dying or exhausted muscle. These twitches can be readily produced by a gentle tapping; they are often visible to the patient, lasting two or three minutes, and starting without any apparent stimulus. A partial reaction of degeneration is often present, i.e. ACC is greater than KCC, but the galvanic response is not increased. It may be absent in many instances, owing to the escape of some of the fibres of the motor nerve, so that degenerated and healthy muscle fibres are intermixed. There is then diminished irritability, both to faradism and galvanism. The disease may cease spontaneously, usually at a late stage, or may end fatally from complication with bulbar paralysis or intercurrent pneumonia, etc.

The *peroneal type* of muscular atrophy differs from the above description. It is a family or hereditary disease, beginning in late childhood, affecting first the peronei and the muscles of the feet, and spreading to those of the thighs. The knee-jerks are lost. Club-foot is a common consequence. After some years, the disease spreads to the arms, commencing in the intrinsic muscles of the hands. Degenerative changes have been found in various sections of the peripheral motor neurone.

**Differential Diagnosis.** (See Table, p. 451.)

**Treatment.**—The disease is progressive and incurable, but its advance may be retarded by maintaining the nutrition of the muscles by means of gentle massage, moderate electrical stimula-



tion, and avoidance of fatigue. The only drug of much value is strychnine, given hypodermically in doses of  $\frac{1}{80}$ th to  $\frac{1}{40}$ th of a grain (grm. 0·001–0·0015) once daily. It is contra-indicated if spastic symptoms are present, as in amyotrophic lateral sclerosis. General tonics may also be used.

We have in the foregoing pages studied lesions limited to one system of the cord, and we have found them to present the following characteristics:—

Posterior affection—inco-ordination, paræsthesia, and loss of reflexes.

Lateral affection—spasm, rigidity, and increased reflexes.

Anterior affection—atrophy and loss of reflexes.

But system diseases are not always so strictly limited, and the degenerative process may “overflow” from one system to another, so that “mixed” lesions are the result. Of these the following are the most common forms:—

1. Amyotrophic lateral sclerosis.
2. Ataxic paraplegia.
3. Hereditary ataxia.

## AMYOTROPHIC LATERAL SCLEROSIS

Although this disease was described by Charcot as distinct from progressive muscular atrophy, the lesions in the cord are in the two affections practically identical, the pyramidal fibres being affected in both cases. The difference between them is that, in the one case, the degeneration affects first and principally the lower motor neurone, thereby preventing the development of spastic symptoms, while, in the other, the affection of the upper (cortico-spinal) neurone is more prominent, and colours the clinical picture. There are many cases which present intermediate stages between the two typical forms, which may be regarded as “extreme examples at the opposite ends of a series.” Provided the essential pathological similarity is understood, it is still desirable to describe separately the two sharply contrasted types.

**Etiology.**—The causes—exposure to cold and wet, fatigue, nervous worry—are similar to those of progressive muscular atrophy. The age of onset is from twenty-five to fifty, and both sexes are liable. Syphilis is not a factor.



**Morbid Anatomy.**—Sclerotic degeneration of—

1. The crossed pyramidal tracts, extending upwards sometimes to the pons and medulla, sometimes even to the cortex.
2. The anterior cornua, with their nerve cells, the anterior roots, and the motor nerve fibres. The muscles also degenerate.

**Symptoms.**—1. Weakness and wasting of the *upper* extremities coming on very slowly. Pain and disordered sensation may be present, but not anæsthesia. Electrical excitability of the muscles is lessened for both galvanic and faradic stimuli.

2. *Increase of reflexes with spasm* (until the wasting is profound; then, the anterior multipolar cells being extensively diseased, the reflexes *may be abolished*). In typical cases, the spasm and rigidity cause a characteristic deformity, with the following features:—

The arm is extended close to the body.

The forearm is semi-flexed and pronated.

The wrist is strongly flexed; and the fingers are bent into the palms.

3. Later, in the second stage, spastic paralysis commences in the *lower extremities*. It is accompanied by exaggeration of the reflexes, and followed by atrophy. The sphincters are not affected.

4. Finally, in the third stage, there occur symptoms dependent on *extension of the disease to the motor nuclei of the medulla*. The tongue or lips may be affected, the palate paralysed, the speech nasal. Dysphagia may follow. Septic pneumonia, or cardiac or respiratory paralysis, may end the scene.

The disease runs a quicker course than the chronic lesions of the cord already described, and death usually occurs in from one to three years.

The beginner is frequently puzzled to understand why, in this disease where *the anterior horn* is involved, reflexes are increased instead of lost. The explanation is, that this affection *begins*, in many cases, in the upper efferent neurone. Cerebral inhibition is therefore cut off, and as the reflex loop is still intact, there is exaggeration of reflexes. When the anterior horn becomes affected the reflexes corresponding to the site of the lesion disappear.

**Treatment** is similar to that of progressive muscular atrophy, but strychnine is contra-indicated.



## ATAXIC PARAPLEGIA

**Etiology.**—This condition occurs most frequently in males, beginning in early middle age. There is rarely any connection with syphilis, and the etiology is largely conjectural. Exposure to cold, sexual excess, etc., are blamed, as in all chronic spinal affections. Pernicious anæmia and other exhausting diseases are sometimes complicated by a combined sclerosis of similar type, the origin of which is evidently toxic.

**Morbid Anatomy.**—Sclerosis of—

1. The lateral columns (direct and crossed pyramidal tracts, antero-lateral, and direct cerebellar tracts).
2. The posterior columns (but not the roots). The thoracic part of the cord is more affected than the lumbar.

**Symptoms.**—The onset is gradual, and the first symptoms may resemble those of lateral sclerosis.

1. The patient complains of tiredness and weakness of the limbs, with some degree of stiffness, but no lightning pains.
2. Inco-ordination; the patient reels or sways if the eyes be closed, and he fails to walk on a straight chalked line.
3. *Increase* of knee-jerk (ankle clonus is often present). Babinski's sign may be present. The exaggeration of the reflexes is due to the interference with cerebral inhibition and the integrity of the reflex arc, the posterior root-zone not being affected.
4. As the disease progresses, the tendency is to become *more spastic*, and less ataxic in character—*i.e.* inco-ordination does not increase, but *spasm and rigidity* become markedly increased. Both ataxy and spastic weakness may extend to the upper limbs.

It should be noted that there is no anæsthesia, lightning pain, or alteration of the optical apparatus; points which distinguish it from locomotor ataxia; and this is what we should expect, seeing there is no involvement of the posterior *nerve roots*, or *cranial nerves*. It must also be remembered that similar symptoms may be produced by disseminated sclerosis.

**Treatment** consists in maintenance of the general health, and in the treatment of any anæmia or other constitutional affection which may be causative.



## HEREDITARY ATAXIA

(FRIEDREICH'S DISEASE.)

**Etiology.**—The disease may or may not be hereditary. It is strictly rather a family disease, tending to affect several brothers or sisters. It begins as a rule in childhood or about the age of puberty, and affects both sexes. There is seldom any obvious cause.

**Morbid Anatomy.**—Sclerosis involving principally the neuroglial element in—

1. The lateral columns—The direct cerebellar tract, the antero-lateral tract of Gowers, and part of the crossed pyramidal tract.
2. The posterior columns, and the posterior nerve-roots (partially or completely).
3. Clarke's columns in the grey matter (to a slight degree).
4. Certain cranial nuclei.

**Symptoms.**—

1. Inco-ordination of a jerky, staggering kind, affecting first the lower extremities.
2. Inco-ordination of the *arms* (somewhat later).
3. Hesitation in speaking, or a jerky manner in delivery of speech.
4. Unsteadiness of the *head*, and *oscillatory movements of the eyeballs* (nystagmus).
5. Impairment of sensation—the anæsthesia is not marked in most cases.
6. Absence of knee-jerk, but often presence of Babinski's sign.
7. *Talipes equinus and other deformities*, especially curvatures of the spine.

Note *absence* of lightning pains, gastric crises, trophic changes, and the Argyll-Robertson pupil, and also the early age of onset, to distinguish this disease from locomotor ataxia. The marked inco-ordination and nystagmus render the diagnosis from spastic paraplegia easy. In disseminated sclerosis optic atrophy is generally present, but not in hereditary ataxia.

The disease is slowly progressive, in spite of treatment. Death is due to intercurrent affections.



## Differential Diagnosis of Chronic System Diseases of the Spinal Cord

TABLE I.

	LOCOMOTOR ATAXIA.	ATAXIC PARAPLEGIA.	HEREDITARY ATAXIA.
<i>Age</i> . . . .	Middle-aged men	Early middle life ; males.	Childhood or early youth.
<i>Causes</i> . . . .	The toxic effects of syphilis, rarely other toxins.	Exposure to cold, traumatisms, etc.	Occurs in many of the same generation. Neurotic predisposi- tion.
<i>Ocular symptoms</i> .	Various muscular paralyses or palsies.		
{ <i>Argyll-Robertson</i> <i>pupil</i>	Present.	Absent.	Absent.
{ <i>Nystagmus</i> . .	Absent.	Absent.	Present.
<i>Tendon reflexes</i> ( <i>knee-jerk</i> )	Lost.	Increased.	Lost.
<i>Disorders of Sensa- tion</i>	Lightning pains prominent ; girdle sensation ; numb- ness of feet.	Absent.	Absent usually ; occasional paræ- thesiæ.
<i>Inco-ordination</i> .	Characteristic gait ; lower limbs chiefly affected, upper limbs later.	Ataxia marked ; spasm and rigidity also present, and tend to increase.	Marked, but ir- regular and jerky ; may affect upper limbs.
<i>Speech</i> . . . .	Unaffected.	Seldom affected.	Often affected.

TABLE II.

	PROGRESSIVE MUSCULAR ATROPHY.	AMYOTROPHIC LATERAL SCLEROSIS.	PRIMARY SPASTIC PARAPLEGIA (LATERAL SCLEROSIS).
<i>Limbs most affected</i>	Upper — atrophy begins in thenar and hypothenar eminences. Uni- lateral at first.	Upper — atrophy may begin in muscles of forearm or deltoid. Uni- lateral.	Lower — <i>no atro-</i> <i>phy</i> ; but rigidity and spasm are pre- sent. Bilateral.
<i>Deformity</i> . . .	The "claw-like" hand.	Flexion of elbow, pronation of hand, flexion of wrists, and fingers into palms.	Adduction of legs. They may cross each other.
<i>Tendon reflexes</i> ( <i>knee</i> )	Unaffected.	Unaffected.	Exaggerated on both sides.
<i>Electrical changes</i> .	Reaction of de- generation some- times present.	Partial R. D. or diminished excit- ability.	Normal as a rule.



## IV. DISEASES OF THE MEDULLA OBLONGATA

Much that has been said about the spinal cord may be also said about the medulla oblongata, or bulb. It is a conductor of impulses to and from the brain, and it also contains the principal reflex centres. The more important points may be summed up as follows :—

1. It contains the deep origin of all the cranial nerves after the fourth. The nuclei of motor cranial nerves are homologous with the spinal motor nuclei in the anterior cornua, and may be affected simultaneously.

2. The *motor fibres* decussate to form the crossed and direct pyramidal tracts in the cord.

3. The upper *sensory* decussation (decussation of the fillet) also takes place in the medulla.

4. It contains the following centres : (a) *Centres essential to life* ; respiratory, cardiac, and vasomotor centres : (b) *Centres connected with the alimentary canal* ; those concerned with the acts of sucking, mastication, deglutition, and vomiting : (c) *Centres connected with the eye* ; those for winking and for the dilator pupillæ ; (d) *Centres for secretion* ; those for salivation, lachrymation, and perspiration.

It will be seen from the above table that even a small lesion may be attended by grave and diverse symptoms. A lesion such as an extensive hæmorrhage is incompatible with life. But the medulla is often the seat of degenerative changes, which may form a part of the phenomena of disseminated sclerosis, or may represent the terminal stages of progressive muscular atrophy or amyotrophic lateral sclerosis. When such changes affect the motor nuclei of the medulla before the pyramidal tracts or cells of the anterior cornua, they constitute *bulbar paralysis* ; but even in such cases the disease may ultimately implicate the spinal cord.

## PROGRESSIVE BULBAR PARALYSIS

(GLOSSO-LABIO-LARYNGEAL PARALYSIS)

An affection characterised by progressive paralysis and atrophy of the lips, tongue, palate, pharynx, larynx, and jaws, accompanied by difficult articulation and deglutition, and ending fatally.

**Etiology.**—It occurs most frequently in old people, and is rarely seen in persons under forty years of age. Men are more



frequently attacked than women. A nervous heredity can sometimes be traced, and prolonged exposure, or injuries such as a blow on the neck, have been assigned as causes. The disease is often associated with chronic diseases of the spinal cord (progressive muscular atrophy or amyotrophic lateral sclerosis).

**Morbid Anatomy.**—1. *Sclerotic* changes are found in the—

- (1) Motor nerve *nuclei* of the medulla.
  - (2) Motor nerve *roots* and fibres directly connected with the bulb.
2. Degenerative changes are found in the trunks and motor endings of the glosso-pharyngeal, spinal accessory, and hypoglossal nerves. There is atrophy of the tongue, lips, and muscles supplied by the above nerves. The muscular fibres are in a state of fatty degeneration, or may be replaced by fat.

The lesions originate in the motor nerve nuclei, the cells of which show shrivelled processes, shrunken cell bodies, absence of Nissl's bodies, and shrinkage or absence of the nuclei. The total number of cells is lessened. The changes extend throughout the neurone to the nerve-endings. The white substance of the medulla is often unaffected, but it may also undergo change; and degenerative lesions of the pyramidal tracts or anterior cornua may be found.

**Symptoms.**—Obviously, with lesions so widespread, the symptoms must vary with the nuclei involved; and if spinal or basal disease be present, the symptoms of these will be super-added. Yet, in spite of these facts, a fairly typical picture of glosso-labio-laryngeal paralysis may be presented. Tabulated, the principal symptoms are—

1. *Impairment of articulation* (*dysarthria*), due at first to defective movements of the tongue, but later to the atrophy of the lips; consequently, defective pronunciation of the letters involving the tip of the tongue is first most marked—*i.e.* the letters, T, K, D, or the exclamation SH!; later, there is difficulty in pronouncing the letters U, O, OO, W, P, B, M, owing to implication of the lips. Finally, *speech may become unintelligible*, but phonation is rarely entirely lost.

2. *Difficulty in swallowing.* The food may accumulate between the lips and gums.

3. *Symptoms indicative of paralysis of the palate—i.e.* nasal



twang of voice, regurgitation of fluids through the nose. The palatal and faucial reflexes are lost.

4. *Dribbling of saliva from the mouth.* This is a most marked symptom, and the patient is continually wiping the secretion away. Possibly more saliva is secreted than normally, but this symptom may be due to deficient deglutition, so that saliva is not *swallowed*.

5. *Symptoms indicative of paralysis of the larynx* are lowering of the pitch of the voice, aphonia, imperfect cough, etc. When the superior laryngeal nerve is paralysed, particles of food may get into the lung and excite a fatal pneumonia.

6. *Symptoms indicative of paralysis of the cardiac centre* are paroxysmal attacks of dyspnoea, a sensation of tightness across the chest, tachycardia and irregular action of the heart, etc.

The patient has usually a sad expression, which may contrast with his often buoyant spirits. There is impairment of emotional control. Tears or laughter are readily excited, though the intellect is unaffected. The condition of the tongue is very characteristic when the disease is well marked. Its muscular tissue is much atrophied, and the mucous membrane hangs in sack-like folds, wrinkled and covered with a dirty yellowish fur. It is tremulous, lies helpless in the mouth, and is only moved with great difficulty; hence the collecting of food between the teeth and cheeks. Sometimes, however, the tongue may be broad and flabby, from the accumulation of interstitial fat. The muscles of mastication may be also involved (showing implication of the fifth nerve).

¶ *The electrical reactions* are usually little altered, but R.D. is sometimes present in the affected muscles.

In some cases there is no wasting, and in these the degeneration is probably confined to the upper neurone. In such cases, the palatal and faucial reflexes are retained, and may be excessive.

**Prognosis.**—Invariably fatal. Death may occur from emaciation, but is more often due to the various complications that arise, especially pneumonia.

**Treatment.**—Careful attention to all the details of health, with the administration of tonics, such as arsenic or strychnine, are the usual measures. Electricity has been tried, without much benefit. The greatest care is essential in feeding, to avoid passage of food into the larynx. Feeding by the œsophageal tube may be required.



**Acute Bulbar Paralysis** of the apoplectic type is of vascular origin, and most frequently follows thrombosis. It occurs in old people with marked atheroma, but may follow syphilitic endarteritis in the young. Its onset is sudden, being accompanied by vomiting, vertigo, and sometimes loss of consciousness. There follow partial or complete loss of articulation (anarthria, *not* aphasia); dysphagia, with regurgitation of fluids through the nose; paralysis of the lower half of the face; and paralysis of the tongue, larynx, and pharynx. The paralysis may extend to the limbs, when the tendon reflexes are exaggerated, and Babinski's sign is found. Dyspnœa or Cheyne-Stokes breathing may occur; the pulse may be rapid, and the temperature raised. In fatal cases, death ensues in a few days, weeks, or months.

**Treatment** is mainly palliative—attention to feeding, etc. In cases where syphilis is even suspected, energetic specific treatment should be undertaken, and may bring about partial recovery.

## V. DISEASES OF THE BRAIN

### INTRODUCTORY

Before beginning the study of cerebral disease, we shall first consider a few of the elementary anatomical and physiological facts, without a knowledge of which brain diseases cannot be understood.

The brain proper consists of two large hemispheres, partly separated from each other by the great longitudinal fissure; but bound together below by various commissures.

**Externally**, each hemisphere is covered with grey matter, named the cortex, which is mapped out by furrows into a series of folds, termed convolutions. These convolutions become gradually more prominent as the animal scale is ascended, and reach their fullest development in the highly educated adult human brain.

Of the *Fissures* of the hemispheres the largest and most evident subdivide the surface of the cerebrum into lobes, and may be called *interlobar*; the smallest fissures—*intralobar*—divide the lobes into convolutions, which have received definite designations.

The *Interlobar Fissures* are three:—the *fissure of Sylvius*, or *lateral fissure*; the *fissure of Rolando*, or *central fissure*; and the *parieto-occipital fissure*.



The *Lobes* of the cerebrum are five in number; four are bounded by the interlobar fissures, and take their names from the bones of the skull in relation to which they lie. These are the *frontal*, the *parietal*, the *occipital*, and the *temporal* or *temporo-sphenoidal* lobes.

The fifth lobe—the *central lobe* (*insula* or *island of Reil*)—is not in contact with the bones of the skull, *but is hidden within the fissure of Sylvius*, the margins of which must be separated in order to see it.

The *external* convolutions of these lobes are:—(1) Of the frontal lobe—the *superior*, *middle*, and *inferior* frontal convolutions, and the *ascending frontal* or *anterior central* convolution; (2) Of the parietal lobe—the *superior* and *inferior* convolutions or lobules, the latter divided into the *supra-marginal* and *angular gyri*, and the *ascending parietal* or *posterior central* convolution; (3) Of the temporal lobe—the *superior*, *middle*, and *inferior* convolutions; and (4) Of the occipital lobe—the *superior*, *middle*, and *inferior* convolutions or lateral occipital gyri.

The convolutions on the mesial surface of the brain are separated from each other by the *calcarine*, *parieto-occipital*, and *calloso-marginal* fissures, the last being also known as the *sulcus cinguli*. These convolutions are:—the *gyrus fornicatus* or *gyrus cinguli*, the *marginal gyrus* or mesial aspect of the *superior frontal* convolution, the *paracentral lobule*, the *præcuneus*, the *cuneus*, the *dentate gyrus* or *fascia*, and the *hippocampal* and *uncinate gyri*.

**Interior of the Cerebrum.**—Internally the cerebrum consists of white matter, and ganglionic masses of grey matter. The solid mass of white matter *above* the corpus callosum is termed the *centrum ovale*.

Below the corpus callosum is an irregular and somewhat T-shaped cavity, divided by septa into smaller spaces, termed ventricles. The ventricles communicate with each other by variously named canals—thus, the lateral ventricles communicate with each other and the third ventricle by the foramen of Monro; the third and the fourth ventricles by the “*iter*” or aqueduct of Sylvius; the fourth ventricle and the sub-arachnoidal space by the foramen of Majendie.

The *Basal Ganglia* are masses of grey matter situated at the base of the brain, viz.—

1. The *Corpora Striata*, consisting of two portions—

(1) Caudate nucleus :—intra-ventricular portion.

(2) Lenticular nucleus :—extra-ventricular portion.



2. The *Optic Thalami*, containing sensory and optic fibres. The *upper* part of each thalamus appears in the lateral ventricles. The *under* surface rests upon the *crura cerebri*. Between the lenticular nucleus externally, and the caudate nucleus and optic thalamus internally, lies the mass of white matter termed the *Internal Capsule*. See p. 420.

3. The *Clastrum* is a narrow band of grey matter outside the lenticular nucleus. Its function is unknown.

4. The *Corpora Geniculata* are masses of grey matter forming rounded swellings on the lateral and median portions of the optic tracts.

5. The *Corpora Quadrigemina* contain sensory fibres, and are also implicated in the movements of the eye.

6. The *Pineal Gland*, apparently concerned with the regulation of growth in early life.

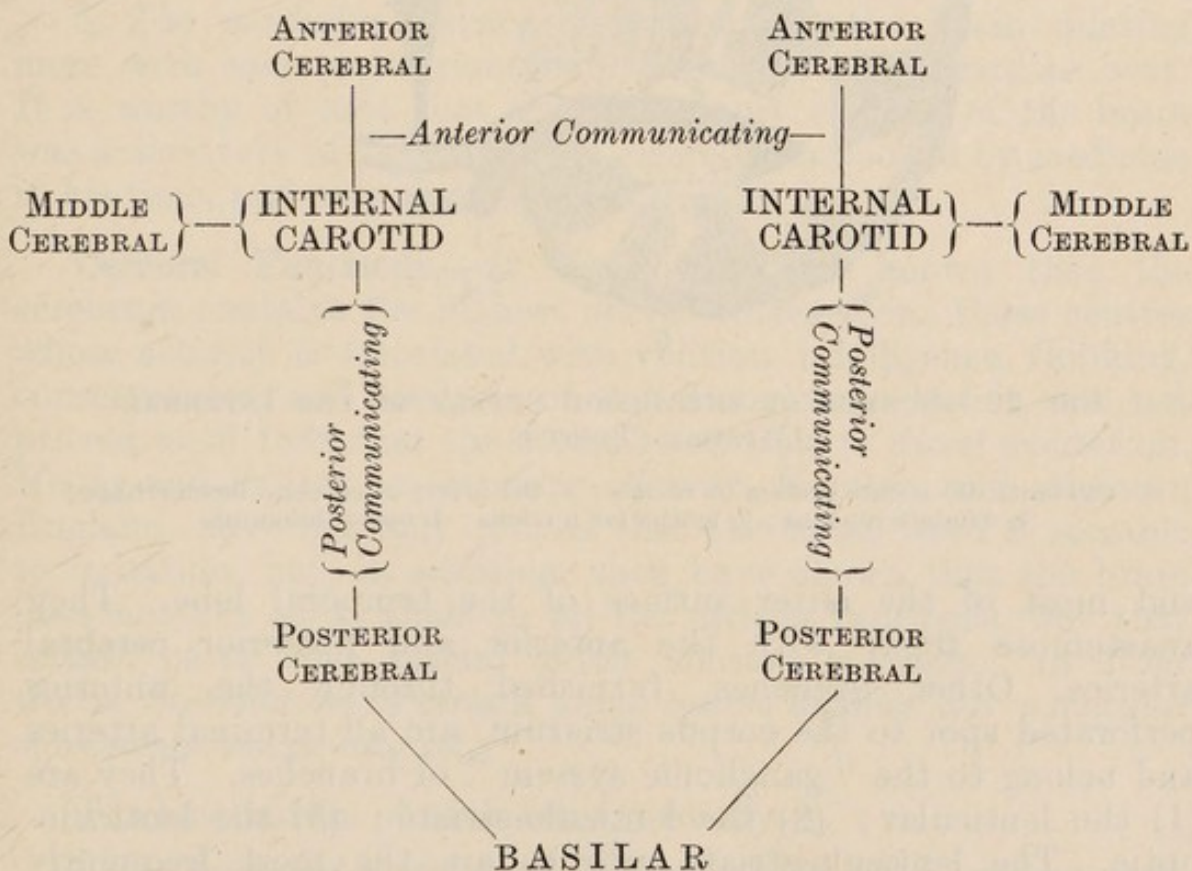
**Blood Supply.**—The arteries of the brain are derived from the two internal carotids, and from the two vertebral arteries, which unite to form the basilar artery.

The branches are practically arranged in two sets, viz. :—

*The Cortical group*—(anterior, middle, and posterior cerebral arteries).

*The Basal group*—comprising the circle of Willis, and the central arteries passing from it.

The circle of Willis may be represented by a diagram :—



So many pathological conditions result from cerebral hæmorrhage, that special attention should be directed to the course of



the middle cerebral artery, branches of which so often rupture, that it is really *the* artery of hæmorrhage.

*The Middle Cerebral or Sylvian Arteries* are the largest branches of the internal carotid, and pass upwards and outwards in the fissure of Sylvius till they reach the surface of the island of Reil, where they ramify in the pia mater, forming part of the cortical system of arteries. These branches supply the under and outer surface of the frontal lobe (in part), and the ascending frontal convolution, the outer surface of the parietal lobe,

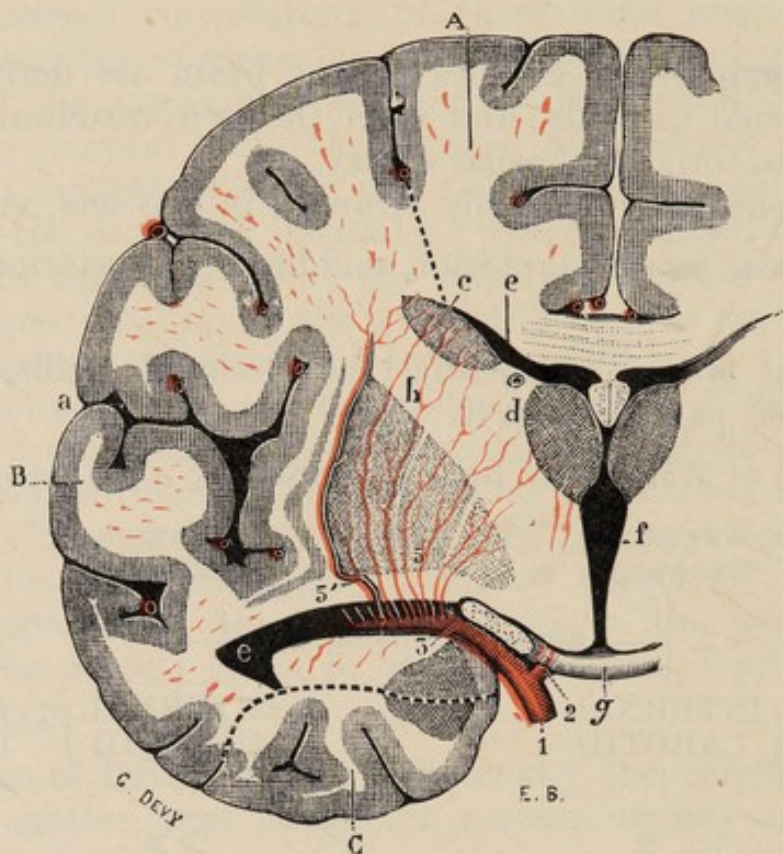


FIG. 22.—SCHEME OF THE BLOOD SUPPLY OF THE INTERNAL CAPSULE (TESTUT).

5. The lenticulo-striate system of vessels ; 5', the artery of cerebral hæmorrhage ; c, caudate nucleus ; b, lenticular nucleus ; d, optic thalamus.

and most of the outer surface of the temporal lobe. They anastomose freely with the anterior and posterior cerebral arteries. Other branches, furnished through the anterior perforated spot to the corpus striatum, are all terminal arteries and belong to the "ganglionic system" of branches. They are (1) the lenticular ; (2) the lenticulo-striate ; (3) the lenticulo-optic. The lenticulo-striate arteries are the most frequently ruptured in cerebral hæmorrhage (*see* Fig. 22).

*Cerebral Veins.*—The veins do not accompany the arteries,



but open into the various sinuses in the dura mater. They are arranged in two sets—

1. *The Superficial set*, which open into the superior longitudinal, the lateral, and cavernous sinuses.
2. *The Deep set*, which gather the blood from the *interior* of the brain, and empty into the straight sinus.

“ *The special characters of the cerebral circulation are—*

“ 1. The free anastomosis at the circle of Willis, which provides a ready supply of blood from other vessels in case of the sudden blocking of any of the more direct channels.

“ 2. The tortuous course through bony canals of the arteries as they enter the skull, thus mitigating the force of the heart's beat.

“ 3. Their ramifications in the pia mater before entering the substance of the brain.

“ 4. The thinness of the arterial walls, and the smallness of the capillaries.

“ 5. The existence of venous sinuses which are without valves, and which do not run with the arteries; the larger arteries, in fact, having no companion veins.” (Whitaker's *Anatomy of the Brain and Spinal Cord*.)

6. *The marked influence of respiration*, the brain dilating more with each expiration than it does with the cardiac beat. It is worthy of note that this respiratory rhythm of the brain was a discovery of Swedenborg's, entirely overlooked by medicine in his time, and only recently re-affirmed.

**Cerebral Functions.**—It has long been known that the cerebrum contains the highest nerve centres—viz., those centres whose activity is associated with volition, intelligence, thinking, consciousness, and analysis of sensation, etc.; but it was not proved until 1882 that the cortex is sensible to *direct* excitation. Hitzig and Fritsch in Germany, Ferrier, Horsley, and others in England, have not only proved that the *cortex* itself is sensible to irritation, but, in addition, they have shown that the brain does not act as a whole in all its various functions, but that certain parts have special duties allotted to them; in other words, *Stimulation of certain areas causes definite and particular movements, sensations, etc.*

The localisation of such areas is briefly—

1. *The Frontal lobes*, which are concerned in the higher psychical functions, and contain an anterior centre for *eye-movements*.

2. *The Rolandic area* (anterior central convolution), associated with motor functions. In the anterior central or ascending frontal convolution



are situated, in order from above downwards, the motor centres for the *legs, arms, face, lips, and tongue*. The motor centre for *speech* is in the *left inferior frontal convolution* (Broca's convolution). If the person be *left-handed*, the speech centre is in the *right convolution*. See Aphasia.

#### *Sensory Centres*

3. The cortical centres for *common sensation* are in the ascending parietal (posterior central) convolution.

4. The centres for *sight* are in the occipital lobe and angular gyrus (see Optic Nerve).

5. The centres for *smell* are in the hippocampal region (uncinate gyrus) in the temporal lobes.

6. The centres for *hearing* are in the superior temporal convolutions.

The results of stimulation of motor centres are seen on the *opposite* side of the body, though sometimes the resulting movements are bilateral (especially as regards movements of the eyes, trunk, and mouth).

In the case of speech, originally bilaterally innervated, the habitual training of the left side of the brain in right-handed people causes the government of the intricate movements of the mouth and throat to be assumed by the left centre only, and the right centre may actually lose its power of function. Cases have been frequently recorded where, through disease of the *left frontal lobe*, speech *for a time* has been entirely abolished, but has been regained in time *through the right centre re-acquiring its function*. In these cases the patients have had to learn language as a child does.

To render comprehensible the general features of brain diseases, it only remains to consider a few simple facts.

Note that *irritation* causes increased action; *paralysis* abolishes the functions of the centres. It must also be remembered that, though the *cerebral cortex alone* has to do with perception of sensation and with voluntary movement, these functions may be abolished by interrupting the afferent and efferent fibres to and from the cortex. The cortex, in fact, is the commander-in-chief; it receives its information by means of inferior officers—basal ganglia, afferent nerves, etc.; and then issues its *orders through a similar efferent mechanism*. The functions of the basal ganglia will now be readily understood. They are subordinate centres conveying and receiving orders from the cerebrum, but they are also able to act, as it were, on their own responsibility as regards certain complex reflexes and co-ordination of movements; thus, the optic thalami contain a large number of sensory fibres passing to the brain, and also a number of *filaments in connection with vision*. Lesions of the optic thalamus or of the cor-



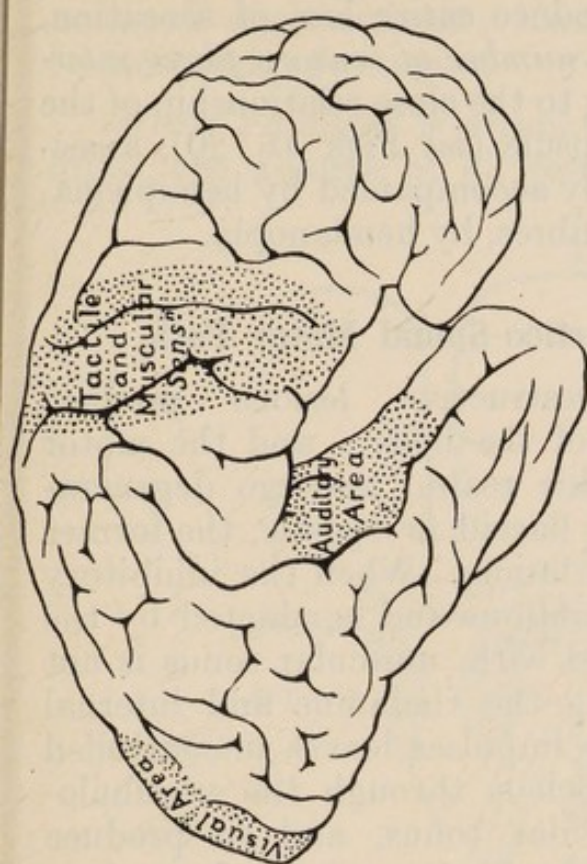


FIG. 24.—SENSORY AREAS ON THE OUTER ASPECT OF THE CEREBRUM (FLECHSIG).

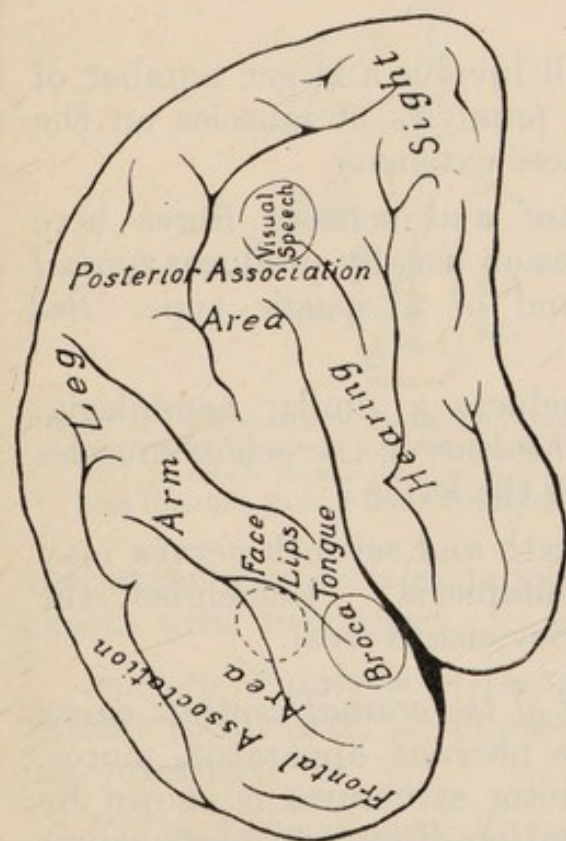


FIG. 23.—MOTOR AREAS IN THE ASCENDING FRONTAL CONVOLUTION (FARQUHARSON).

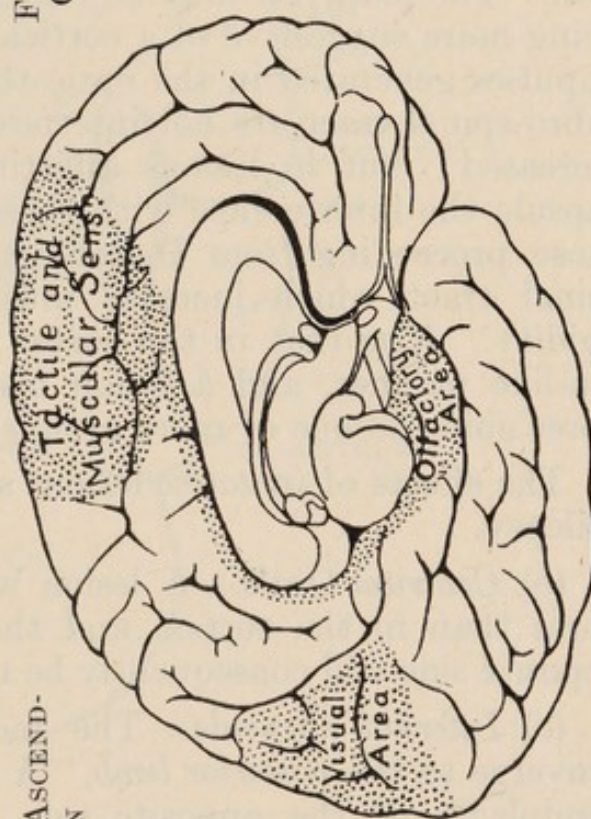


FIG. 25.—SENSORY AREAS ON THE INNER ASPECT OF THE CEREBRUM (FLECHSIG).



pora striata do not necessarily produce *entire* loss of sensation, but only a loss *proportionate to the number of sensory fibres interrupted en route to the brain*. Owing to the close relationship of the optic thalamus to the internal capsule (see Figs. 16, 20), anæsthesia due to such a lesion is usually accompanied by hemiplegia, and, from the implication of visual fibres, by hemianopia.

### Effects of Lesions of the Cortico-Spinal Motor Path.

(a) *The Cerebral Cortex*.—Destructive lesions produce paralysis of the opposite muscles of the body; and the motor fibres, cut off from the first trophic realm, undergo degeneration. The paralysis may be either flaccid or spastic, the former being more suggestive of a cortical lesion. When the inhibitory impulses generated in the optic thalamus and conducted by the rubro-spinal tract are not interfered with, muscular tonus is not increased; but in lesions affecting the thalamus and internal capsule the interference with these impulses leaves uncontrolled those proceeding from Deiter's nucleus through the vestibulo-spinal tract which increase muscular tonus, and so produce rigidity. *Note* that in the cortex the fibres are spread out in a fan-like manner, and a lesion may therefore affect only a few fibres going to one or more groups of muscles.

The effects of *irritative* lesions are described under Jacksonian epilepsy.

(b) *Centrum Ovale*.—A lesion will involve a larger number of fibres than in the cortex, and the paralysis of muscles on the opposite side will consequently be more extensive.

(c) *Internal Capsule*.—The motor and sensory fibres here converge to the *posterior limb*. A lesion usually produces *typical* hemiplegia of the opposite side and of a spastic type. See Hemiplegia.

(d) *Crura Cerebri*.—A lesion produces a similar hemiplegia, but owing to its relations with the *third nerve*, the *ocular* muscles may be paralysed on the *same* side as the lesion.

(e) *Pons*.—Note that here the sixth and seventh nerves may be paralysed in the resulting hemiplegia. Remember the crossed nature of the paralysis (see Seventh Nerve).

**Clinical Considerations.**—*Lesions of the cranial contents* cause either *irritation* or *paralysis* of the nervous apparatus, motor, sensory, or reflex. Irritation of motor structures is shown by *muscular twitchings* or *spasms*; irritation of sensory parts causes



*pain and hyperæsthesia*; irritation of reflex nerve centres leads to *increased reflex action*.

*Motor Paralysis* is estimated by noticing the position of the limbs, the absence of all resistance to passive movements, and

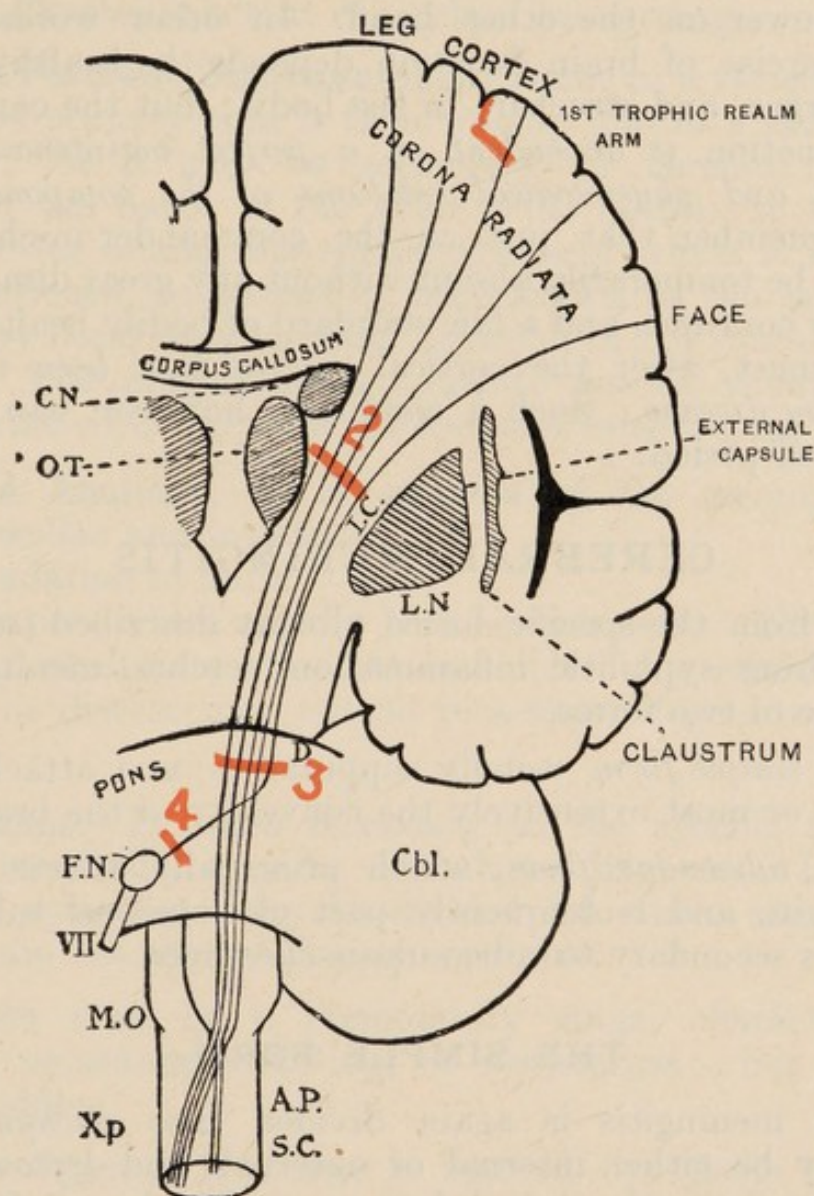


FIG. 26.—DIAGRAMMATIC DRAWING intended to show the effect of lesions interrupting the motor fibres at various levels. Observe a lesion at 1 involves but few fibres; at 2, a much larger number. Note the relations of the facial nerve at 3 and 4. (See Hemiplegia and Bell's Paralysis.)

the presence of stertorous breathing, or flapping of the lips and cheeks with respiration.

*Sensory Paralysis* is recognised by the insensibility of the patient to all external impressions, such as sound, light, pinching, pricking.

*Reflex Palsy* is specially indicated by a fixed condition of the



pupils, and the failure of contraction of the orbicularis palpebrarum when the conjunctiva is stimulated.

We have thus seen that the brain is the initiator or starter of impulses on the one hand, and exercises a controlling or inhibitive power on the other hand. In other words—on the normal exercise of brain function depends the healthy activity of every organ and structure in the body; but the carrying out of this function *is dependent on a perfect maintenance of the anatomical and physiological relations of the component parts*. Lastly, remember that just as the commander-in-chief of an army may be temporarily absent without any great disadvantage, so life may continue, and a fair standard of bodily health may for a time persist, after the cortical centres *have been injured or destroyed by disease*. Such a condition, however, can only last for a limited period.

### CEREBRAL MENINGITIS

Apart from the specific forms already described (*see pp. 50, 53*), and from syphilitic inflammation, cerebral meningitis may assume one of two forms:—

1. The *simple form*, usually suppurative, and attacking most frequently or most extensively the convexity of the brain.

2. The *tubercular form*, which principally affects the base of the brain, and is frequently part of a *general* tuberculosis, and always secondary to tuberculosis elsewhere.

### THE SIMPLE FORM

Simple meningitis is again divided into *pachymeningitis*, which may be either internal or external, and *leptomeningitis*. *External* pachymeningitis, being generally due to injury or disease of the bones of the skull, is chiefly of surgical interest.

**Pachymeningitis interna hæmorrhagica** (*hæmatoma of the dura mater*) is a rare affection, occurring in elderly people with degenerated arteries, especially in general paralytics and chronic alcoholics. On the inner surface of the dura is found, unilaterally or bilaterally, a thin vascular membrane, in one or several layers, into or between the layers of which small hæmorrhages may occur, or larger hæmorrhages producing a laminated clot. Whether inflammation or hæmorrhage is the primary factor is still a disputed point. Symptoms are variable. They commence



with convulsions deepening into coma. Recovery usually follows, although hemiparesis may persist for some time. There may be other symptoms of cerebral compression. *Fresh attacks occur with each fresh hæmorrhage*, the mental state deteriorates, and dementia develops. Death may occur in an apoplectic seizure. *Treatment* is that of cerebral hæmorrhage.

**Acute Purulent Leptomeningitis** is due to a variety of causes. It may be primary, and is then usually due to a pneumococcus infection; or it may be secondary to cerebral abscess or disease of the bones of the skull (*otitis media*), to the various specific fevers among the complications of which it has already been mentioned, to ulcerative endocarditis or the septic states, and it may occur as a terminal infection in the course of chronic diseases. The pneumococcus, various specific organisms, and the pyogenic cocci are the most frequent infecting agents.

**Morbid Anatomy.**—1. Hyperæmia of the meninges, which become swollen and injected.

2. Exudation of fibrin.

3. Effusion, which rapidly becomes purulent. The convexity is most affected, as a rule, but in children the effusion may be basal. The disease may extend in a lesser degree to the spinal meninges.

**Symptoms.**—In cases secondary to the specific fevers, the onset of meningitis may be difficult to diagnose. In primary cases, the symptoms may be divided into three stages, which, however, are not always sharply separable.

1. There may be a premonitory stage, characterised by headache, occasional vomiting, or restlessness; but often the onset is sudden.

2. The *Stage of Irritation* is marked by febrile symptoms, great intolerance of light, contracted pupils, exaggerated reflexes, rigidity of the limbs, and convulsions. Dulling of consciousness is early evident, and there are often periods of wild delirium. Kernig's sign may be present. The patient lies curled up in a characteristic manner, and the head is sometimes markedly retracted. The pulse, which is at first rapid, becomes slow and irregular. Headache is very violent, and *persists through the delirium*.

3. *Stage of Compression.*—The temperature tends to fall, irritability is replaced by stupor, and convulsions by paralysis. The pupils now become uneven or dilated, and ocular palsies



may occur. Optic neuritis is present in basal meningitis, but is less common when the convexity is attacked. The peculiar cephalic cry is a constant symptom. Cheyne-Stokes breathing, involuntary escape of fæces and urine, and stertorous breathing, usher in death, which often takes place within a week of the onset of grave symptoms.

Lumbar puncture is of great diagnostic value. The fluid is turbid, and there is a polymorphonuclear leucocytosis. The causative organism may also be identified.

### THE TUBERCULAR FORM

(“ ACUTE HYDROCEPHALUS ”)

Tubercular meningitis occurs most frequently in children, but is not uncommon in adults. While it is almost invariably either part of a miliary tuberculosis, or secondary to tuberculosis elsewhere, the primary focus is sometimes very difficult to find.

**Morbid Anatomy.**—After the invasion of the meninges (pia mater) by the bacilli, tubercles are formed, and their presence gives rise to inflammatory changes. The tubercles themselves may undergo caseation and liquefaction, or may in rare instances become sclerosed and calcified. The exudate is never frankly purulent, but is of a gelatinous fibrino-purulent character, and has a somewhat characteristic greenish hue.

The changes begin in, and principally affect, the perivascular tissue of the Sylvian and other arteries at the base of the brain, hence the *ventricles are often distended*, and the brain substance flattened up against the skull. Hydrocephalus is thus produced by obstruction of the veins of Galen.

**Symptoms.**—Here also the symptoms may be divided into three stages, and these are usually better marked than in simple meningitis.

1. *Prodromal.*—The child usually shows more or less definite symptoms of the tubercular diathesis, such as emaciation, want of appetite, or constipation alternating with diarrhoea. Irritability of temper and headache are perhaps the most common features previous to the onset of *definite* symptoms of the meningeal affection. Cerebral vomiting may be present. Such a condition may last a week or two, or sometimes a month.

2. *Irritative Stage.*—The symptoms are similar to those described under the simple variety, but the head is usually more



retracted and the neck more rigid; the abdomen is hollowed out or boat-shaped; the temperature oscillates; internal strabismus or other paralysis of cranial nerves may be present, and there is often marked vasomotor paralysis, manifested by the *tache cérébrale* (a red line upon the skin rapidly following a stroke of the finger nail). This, however, is not diagnostic. Vomiting is very constantly present, and may or may not be related to food. Headache and somnolence are marked. Children often start out of stupor with a shrill *hydrocephalic cry*, and in them grinding of the teeth is a frequent symptom. The pulse is irregular and slow. This stage continues for a week or so.

3. *Compression Stage*.—The pulse becomes more rapid with the exhaustion of the heart, the symptoms that accompany coma develop, and death may take place in from ten days to six weeks from the onset of acute symptoms.

In the adult, delirium may take the place of convulsions. The course of the disease is more rapid than in children, as the skull cannot expand, and hence intracranial pressure develops more quickly.

**Diagnosis.**—Lumbar puncture yields a cerebrospinal fluid which is less turbid than that of simple meningitis, and may be almost clear. There is lymphocytosis, and not an increase of the polynuclear cells. The following table may help to distinguish the two conditions:

SIMPLE MENINGITIS.	TUBERCULOUS MENINGITIS.
<i>Age</i> .—Any age.	<i>Age</i> .—Young children and young adults.
<i>Cause</i> .—Injury or local causes, fevers, etc.	<i>Cause</i> .—No local cause, but symptoms of tubercle elsewhere.
<i>Course</i> .—Short.	<i>Course</i> .—Longer than simple, especially the prodromal stage.
<i>Convulsions</i> .—May be present.	<i>Convulsions</i> .—Common, even during the compression stage; often precede death.
<i>Abdomen</i> .—Nothing marked.	<i>Abdomen</i> .—Markedly retracted.
<i>Morbid Changes</i> .—	<i>Morbid Changes</i> .—
1. Those of simple or suppurative inflammation.	1. Those associated with the presence of tubercle, and formation of peculiar greenish pus.
2. Attacks convexity of brain.	2. Attacks the base of brain.
3. Ventricles not distended.	3. Ventricles are distended, and hydrocephalus may follow.
<i>Lumbar Puncture</i> .—Fluid turbid; polymorphonuclear leucocytosis.	<i>Lumbar Puncture</i> .—Fluid much less turbid or almost clear; lymphocytosis.



**Treatment of Meningitis.—**

1. *Simple Meningitis*.—Whenever possible, remove any obvious cause by surgical measures. The room must be darkened and absolutely quiet; ice should be applied to the head; and leeching or venesection may be practised in the full-blooded. A saline or mercurial purge should be given, and for the headache, convulsions, and other nervous symptoms, bromides, chloral, or phenacetin (acetphenetidin). The diet must be fluid; in the later stages preventive treatment must be used for bedsores, and the distended bladder must be relieved. Lumbar puncture may be used therapeutically, and urotropin may be tried. If the organism can be discovered, a serum may be injected.

2. *Tubercular Meningitis*.—This form demands similar treatment after symptoms are developed, and in addition mercurial inunction may be tried, as it has sometimes produced at least temporary improvement. During the prodromal stage, everything should be done to combat the tubercular condition by careful hygiene and diet, by the administration of cod-liver oil, and by fresh air.

**HYDROCEPHALUS**

Acute hydrocephalus, as we have seen, is a synonym for tubercular meningitis. *Chronic* hydrocephalus, to which the term is now usually restricted, may be—

1. Congenital. The causes are not known, but there is sometimes a family proclivity to the affection.
2. Mechanical; due to obstruction of the veins of Galen, as by the pressure of a tumour, or to blocking of the foramen of Magendie, as by meningitis, or of the iter.
3. "Idiopathic." This form is due to serous effusion into the ventricles, and may occur at any age. A serous meningitis and an angioneurotic condition have both been invoked to explain it.

**Morbid Anatomy of Congenital Hydrocephalus—**

*Skull*.—The sutures fail to unite, and the skull as a whole does not ossify as in health. A characteristic deformity thus develops; the brow overhangs, the circumference of the cranium is greatly increased, and is *disproportionate to the size of the face*, and the fontanelles remain open. The circumference of the head may reach thirty inches or even more.

*The Ventricles* are distended, and one, two, or more of them



may be affected. Their lining membrane is thickened and granular. The fluid is clear and contains albumin and salts. Its quantity varies, sometimes reaching as much as fifteen pints.

*The Brain Substance* is much compressed, the convolutions flattened out, and the cortex much thinned.

*Certain Cranial Nerves* may be pressed upon, and subsequently atrophy, especially the *optic* nerves.

**Symptoms.**—The size and shape of the head sufficiently indicate the nature of the disease, even at an early period; later, the symptoms that develop are—

1. General arrest of development.
2. Impaired digestive functions, and distension of the abdomen.
3. Walking power is slowly gained, or not at all.
4. Mental deficiency, sometimes complete idiocy.
5. Convulsions, and sometimes blindness.
6. Condition of apathy, coma, and death, usually in from five to seven years. Cases have been known to attain the age of thirty years.

**Treatment** is very unsatisfactory, if not hopeless.—

1. Pressure by strips of plaster or elastic bands.
2. Puncturing at various intervals, and drawing off the fluid. The ventricles may be tapped, and direct drainage established, or the fluid may be more slowly removed by puncture of the subarachnoid sac in the lumbar region.

## SINUS THROMBOSIS

This is the formation of blood-clot in one or other of the intracranial sinuses. It may be simple or infective. The *simple form* occurs in ill-nourished children, and in the debilitating diseases of adults (phthisis, cancer, fevers, anæmia). The superior longitudinal sinus is most often affected. Its walls are not inflamed, but the lumen is filled with adherent clot, which tends to become organised and absorbed. *Infective thrombosis* follows middle ear disease, caries of other parts of the skull or of the teeth, retropharyngeal abscess, erysipelas of the scalp, etc. The lateral sinus is oftenest affected. Its walls are inflamed; the clot breaks down and becomes purulent, and meningitis or cerebral abscess may occur.



**Symptoms** may be absent in simple thrombosis. There are generally headache, vomiting, convulsions or delirium, and drowsiness deepening into coma. There may be epistaxis from engorgement of the nasal veins. In children the fontanelle may be prominent. In thrombosis of the cavernous sinus, the orbital veins are engorged, and the eyelids œdematous; the fundus shows retinal hæmorrhages and engorged veins; and there may be paralysis of ocular muscles. In thrombosis of the lateral sinus, the mastoid region is œdematous and tender; the thrombosis extends to the jugular vein, which can be felt in the neck as a hard, tender cord; there is local meningitis, and possibly optic neuritis. There is a purulent discharge from the ear.

In infective thrombosis, symptoms of general sepsis are superadded,—vomiting, rigors, profuse sweating, remittent fever, and rapid, irregular pulse. Septic pneumonia, due to pulmonary infarction, may follow, or an abdominal type of infection,—dry tongue, vomiting, diarrhœa, and meteorism, ending in a typhoid state. Meningeal symptoms are also present, and death is preceded by coma.

**Treatment.**—In the simple form treat the constitutional state, and favour free circulation through the brain, particularly avoiding constriction of the neck by clothing. In the infective form the treatment is surgical.

## ENCEPHALITIS

Encephalitis is an inflammation of the substance of the brain, which may be either hæmorrhagic or suppurative, acute or chronic. The acute form may be focal or diffuse; in the chronic form there are scattered inflammatory patches. The hæmorrhagic form is associated with infective diseases, such as enteric, influenza, diphtheria, syphilis, and with the exanthemata. It may follow injury to the head. The suppurative form follows suppuration of the accessory cranial cavities—ear, orbit, nose.

**Acute Hæmorrhagic Encephalitis** (focal) attacks the grey and white, but especially the grey matter of the mid and hind brain and of the cortex. The nuclei of cranial nerves are thus involved. The condition is analogous to acute anterior poliomyelitis (*q.v.*), and is often called *polioencephalitis*. The affected parts are found to be softened, and show engorgement and thrombosis of vessels, hæmorrhages, and proliferation of leucocytes, the nervous elements being destroyed.



*Symptoms.*—Sudden onset ; headache, vomiting, convulsions, fever, drowsiness, leading to coma and death, or partial recovery. Optic neuritis is sometimes seen. Local symptoms are sudden ophthalmoplegia (*polioencephalitis superior*), or sudden bulbar paralysis (*polioencephalitis inferior*). The nuclei of the fifth and seventh nerves may also suffer.

**Acute Diffuse Encephalitis** is rare. It is oftenest seen in children, when it may follow infections or be caused by traumatisms. In the adult it is associated with syphilis. Large areas of the brain on one or both sides are involved in the inflammatory process, which leads to softening (*red softening*). Later, the affected portions may become atrophied or cystic (*porencephaly*). The disease may be fatal, but more often ends in recovery, leaving behind it, however, paralysis of a cerebral type—infantile hemiplegia or diplegia—and in some cases epilepsy.

### SUPPURATIVE ENCEPHALITIS— ABSCESS OF THE BRAIN

The causes of cerebral abscess are to be found chiefly in disease of the accessory cavities, especially the *middle ear* ; but disease of the cranial bones, fracture, or gun-shot wound of the skull, may give rise to it, and also infection from a distance (metastatic abscesses), as in septic pneumonia or peritonitis, osteomyelitis, or pyæmia. The temporal lobe is most often affected, and next the cerebellum. Any part of the brain may suffer, according to the seat of the causative disease.

**Morbid Anatomy.**—The abscess is usually situated in the white matter, and may be separated from the surface by healthy brain tissue, the infection having been carried by the venous sinuses and perivascular lymphatics. Usually there is a localised meningitis, the dura adhering to the brain. If the abscess is acute, the pus is not encapsulated, but surrounded by broken-down nervous tissue ; if chronic, there is a definite capsule.

**Symptoms of Acute Abscess.**—Pain in the ear, radiating over the side of the head ; cessation of aural discharge ; rigors, and slight fever ; vomiting. After a few days, lassitude, drowsiness, inability to fix the attention or to answer questions ; temperature normal or subnormal ; pulse slow and full ; respirations slow, tending to Cheyne-Stokes type ; optic neuritis in the later stages ; sometimes cerebral palsies ; convulsions *uncommon*, unless the Rolandic area is involved. Ultimately the lethargy



deepens into coma and death, which may be sudden from rupture into the lateral ventricles. *Localising symptoms* :—

1. *Temporal lobe*. Paralysis of the third nerve on the side of the ear disease. Hemiplegia on the opposite side, most marked in the face, least in the leg. Word-deafness when the left first temporal convolution is affected.
2. *Cerebellum*. Giddiness and staggering gait, tendency to fall to the side ; retraction of the head and stiffness of the muscles of the neck ; nystagmus, deviation of the eyes *away from* the side of the abscess ; sometimes hemiparesis *on the same side* as the abscess with variable intensity of the knee-jerk, which is sometimes exaggerated.
3. *Frontal lobe*. Localising signs may be absent, or there may be paralysis of the limbs (arms especially) and the face on the opposite side, with aphasia if the abscess is on the left side.

Remember that abscess may be entirely latent, or lead only to a more or less transitory irritability, inertia, and headache. After these have passed off, injury to the head or relapse of a previous ear trouble may, even years afterwards, light up acute symptoms and lead to death.

**Treatment.**—Attend at once to all chronic suppurative conditions of the ear, nose, or frontal sinuses (prophylaxis), and employ strict antiseptic precautions in dealing with erysipelas or scalp wounds. When abscess is actually present, immediate operation is the only treatment, even in the absence of localising symptoms. A clue to the site of the abscess will be found in the seat of the primary disease.

## CEREBRAL HÆMORRHAGE

**Etiology.**—Hæmorrhage into the brain occurs most frequently in men between the ages of forty and sixty, but may occur at any age. Amongst the more important predisposing causes are—

1. A certain type of build. It is frequent in stout, plethoric men, with short, thick necks.
2. Certain occupations, among which are those of (1) butchers and publicans, owing to arterial changes produced by excessive consumption of nitrogenous food and alcohol ; (2) carters, hammermen, etc., as the result of vascular strain.



3. Certain blood diseases—leukæmia, pernicious anæmia, scurvy.

4. Vascular degeneration, due to *chronic renal disease*, the rupture being aided by the forcible pulsations resulting from cardiac hypertrophy. *Syphilitic* degeneration may lead to hæmorrhage, but more frequently causes thrombosis.

5. Removal of the natural *support* of the vessels, as in cerebral softening.

6. Injuries from without the cranium, etc.

The most important exciting causes are—

1. Disease of the vascular walls.

(1) Miliary and other aneurysmal dilatations.

(2) Arterio-sclerosis and atheroma, whether due to gout, renal disease, alcoholism, or other causes. Independently of renal disease, the blood-pressure tends to rise with advancing years.

2. Vascular strain, as from exercise, or causes such as straining at stool, or violent coughing. It will be remembered that the brain expands during expiration, and expiration *with a closed glottis* very greatly increases the intracranial pressure.

**Site of the Hæmorrhage.**—The most common form of cerebral hæmorrhage is *into the substance* of the brain, and is usually due to rupture of the lenticulo-striate and lenticulo-optic branches of the middle cerebral artery, which supply (as we have seen) the basal ganglia. These arteries are the most frequent seats of miliary aneurysm, and the reason why they are so frequently ruptured is probably that they are the most direct branches of the middle cerebral, which is in turn the most direct branch of the internal carotid. Meningeal hæmorrhage may also take place, as in depressed fracture of the skull or pachymeningitis hæmorrhagica.

**Anatomical Changes.**—If the hæmorrhage be severe, it tears up the brain tissue, or it may destroy the basal ganglia, and bursting into the lateral ventricles distend them, and flow through the aqueduct of Sylvius into the fourth ventricle. The effects of a limited hæmorrhage are naturally less extensive, but some degree of laceration of the brain substance always follows. The effused blood subsequently undergoes the following changes :—

(1) Retraction of the clot, and separation of the serum, which is partially absorbed.



- (2) Discolouration of the clot, and formation of hæmatoidin crystals.
- (3) Formation of a *serous cyst*. Occasionally there may be complete absorption, when only a fibrous cicatrix is left.

The surrounding tissues also undergo change :—

- (1) The irritation of the clot produces an inflammatory reaction, leading to—
- (2) Connective tissue proliferation, forming a capsule around the clot.

*The fibres of the motor path*, interrupted by the hæmorrhage, are thus cut off from the first trophic realm (cortex). Secondary descending degeneration therefore occurs, and involves the motor tracts on the affected side, and after the decussation in the medulla, on the opposite side (crossed pyramidal tract).

*Summary.*—It will be apparent that if the patient survives the primary shock (the apoplectic fit), he will suffer from symptoms due to one of three causes :—

1. Motor paralysis (hemiplegia) from interruption of the motor fibres.
2. Inflammatory reaction.
3. Secondary descending degeneration.

**Symptoms.**—There may be premonitory warnings of the attack, such as giddiness, headache, etc. The attack may occur when the patient is resting or asleep, but much more frequently it is directly attributable to an overloaded stomach, a severe strain, coughing, or the exertion in running to catch a train. Apoplexy is not synonymous with cerebral hæmorrhage ; still, the condition which supervenes on such a lesion is generally summed up in the description of an—

*Apoplectic Fit.*—The patient is suddenly seized with severe pain in the head, feels faint or giddy, and quickly falls into a comatose condition. Sometimes the loss of consciousness is more gradual, and may not be complete for some hours (*ingravescent apoplexy*). The face is flushed and bloated, the pulse full and tense, the breathing stertorous, and the cheeks puffed out. The snoring noise is due to the paralysed tongue and palate falling back, and impeding the entrance of air. The pupils are dilated or irregular. The limbs at first are flaccid, but may become rigid with the onset of reaction (so-called *early rigidity*). During the coma both the superficial and the deep reflexes are abolished ;



later they return, first on the sound side, and afterwards on the affected side, where they are exaggerated. There are inability to swallow, retention or involuntary dribbling of urine, and involuntary evacuations. The head and eyes are frequently turned to one side—*i.e.* the patient looks *towards* the lesion and *away* from the paralysed side (*conjugate deviation*), owing to the balancing action of the associated muscles being lost from paralysis. When the case is going to end fatally, the coma increases, temperature falls, and Cheyne-Stokes respirations usher in the fatal end. In pontine hæmorrhage, however, which is usually rapidly fatal, the temperature *rises* even to hyperpyrexia, and the pupils are contracted. In favourable cases the primary disturbance is not so grave, and the patient may regain consciousness in a few hours, or even after two or three days. "Reaction" then sets in, the temperature rises, as does the pulse, perspiration occurs, and there is restlessness, with excitement amounting sometimes to delirium. In this stage, which may last from a few days to several weeks, trophic disturbances may occur, such as bedsores on the paralysed side, and the case may end fatally; but recovery is more common. It is rarely complete, but usually attended by more or less paralysis or typical hemiplegia.

It is possible even in the comatose condition to make out the existence of paralysis. The cheek is more puffed out on the affected side, the naso-labial groove is obliterated, and the angle of the mouth is lower, while saliva trickles from it. If the limbs upon the two sides be raised alternately and then let fall, those on the paralysed side fall in an absolutely flaccid fashion, while upon the other side some tone is maintained. The cremasteric and abdominal reflexes are absent on the paralysed side, on which Babinski's sign is often present.

In *cortical* hæmorrhage—

1. Consciousness is seldom completely lost at the onset of paralysis.
2. The paralysis is of a monoplegic type.
3. The onset is attended with convulsions.
4. The prognosis is more favourable.

For localising symptoms, see p. 477.

**Treatment** of Cerebral Hæmorrhage.—An apoplectic patient should be moved as little as possible. An improvised couch should be made for him in the room where he fell, and if he must be moved to hospital it should be in an ambulance. The head should be slightly raised, and ice applied to the scalp on the side



of the lesion, and a hot-water bag may be applied to the feet. The bowels should be moved by an enema, calomel grains v (grm. 0.3) on the tongue, or a minim of croton oil. Venesection may be performed if the pulse is full and the blood-pressure high; if, on the other hand, the pulse is weak, or if the symptoms are due to thrombosis or embolism, it is best not to resort to bleeding. Compression of the carotid may be useful in cases of ingravescent apoplexy. Relieve stertor by turning the patient on his side; draw off the urine by catheter; and feed by the rectum if the coma is prolonged. In the stage of reaction fever diet must be given, preventive treatment must be adopted for bedsores, and headache and cerebral excitement must be treated by sedatives and cold applications.

## EMBOLISM AND THROMBOSIS

### (SOFTENING OF THE BRAIN)

Instead of a free extravasation of blood, we may get blocking of blood-vessels through detached clots (embolism), or a local clotting of blood (thrombosis).

**Etiology.**—See Table, p. 477.

**Morbid Anatomy.**—The changes following thrombosis or embolism will vary according to the site and size of blood-vessels affected. Briefly, the results are degeneration and softening of the parts supplied by the affected artery. Blocking of a terminal artery causes the affected area to look slightly paler at first; then extravasation from the engorged veins and distension of the capillaries may take place and cause an infiltration of blood. The resulting softening may be of the red, yellow, or white types, according to the amount of blood effused into the affected area, which undergoes changes similar to those described under hæmorrhage, with the ultimate formation of a cyst or a cicatrix. Two things the student must remember—

1. If the clot be of a *septic* nature, an abscess is likely to form.
2. If *not* septic, it is surprising how slowly the softened area undergoes further change.

Thrombosis may follow upon embolism, a small branch being first blocked, and the thrombus extending backwards till it blocks a large branch or the main trunk of the middle cerebral artery.



The following table shows the main etiological factors and the symptomatic differences between the two conditions.

DIAGNOSTIC TABLE

CEREBRAL EMBOLISM.	CEREBRAL THROMBOSIS.
<p><i>Causes.</i>—Most commonly associated with valvular lesions of the heart, aneurysms, or a suppurating thrombus.</p> <p><i>Age.</i>—Chiefly in young adults.</p> <p><i>Onset.</i>—Sudden—no prodromal symptoms.</p> <p><i>Convulsions.</i>—Rare.</p> <p><i>Paralysis.</i>—A sudden hemiplegia of the right side, <i>with aphasia</i>. The left middle cerebral artery is most frequently plugged.</p> <p><i>Consciousness.</i>—Not usually entirely abolished.</p>	<p><i>Causes.</i>—1. Arterio-sclerosis, atheroma and syphilitic disease of the arteries. Bright's disease and alcoholism therefore predispose.</p> <p>2. Pressure upon the veins or arteries (tumour or abscess).</p> <p><i>Age.</i>—In the old from vascular degeneration; in the young from syphilis.</p> <p><i>Onset.</i>—Gradual—with prodromal symptoms.</p> <p><i>Convulsions.</i>—Convulsive attacks are not uncommon, as the cortex is frequently affected.</p> <p><i>Paralysis.</i>—Paralysis is more gradual. Aphasia is not so common, as either the right or left side may suffer.</p> <p><i>Consciousness.</i>—Often not lost. Coma may come on gradually, after the hemiplegia.</p>

*Blocking of particular arteries.*

1. *Vertebral.*—Rare, left more common than right. The symptoms are those of acute bulbar paralysis.

2. *Basilar.*—Bilateral paralysis with *rise of temperature*.

3. *Posterior Cerebral.*—Affections of vision (hemianopia) and hemianæsthesia (posterior portion of internal capsule).

4. *Middle Cerebral.*—Hemiplegia, with aphasia if the lesion is on the left side.

5. *Anterior Cerebral.*—Grave interference with the higher intellectual faculties.

Lastly, when small cortical vessels are affected, there may be, as in hæmorrhage, convulsions at the onset, the paralysis is monoplegic, and it is often transitory, for a collateral circulation may be at least partially established.

**Treatment.**—In the apoplectic seizure, avoid venesection or free purgation, but see that the bowels are opened. If the heart be weak, ammonia or digitalis may be given. Where



sypilis is the cause, iodide of potassium or mercurial inunction should be at once begun. The stage of reaction must be treated as in hæmorrhage.

### HEMIPLEGIA

By hemiplegia is meant paralysis of the face, arm, and leg on one side of the body, resulting from a lesion of the opposite cerebral hemisphere. The movements of the muscles of the trunk (especially the respiratory muscles), and those of the vocal cords, are not usually affected, inasmuch as they are bilaterally innervated, and the destruction of one centre means that the opposite centre assumes its functions.

**Causes.**—Hemiplegia is most commonly the result of embolism, thrombosis, or a free hæmorrhage. It may be caused by depressed fractures involving the Rolandic area. It may be due to a tumour involving the motor fibres anywhere between the cortex and the medulla, in which case it develops gradually. In hæmorrhage the motor fibres are torn; in embolism or thrombosis there is softening of the cerebral tissues. Functional hemiplegia occurs in some cases of hysteria.

**Symptoms.**—When the hemiplegia is not preceded by an apoplectic fit, the patient often first becomes aware of anything wrong by finding on awakening from sleep—

1. Loss of power on one side, or
2. Difficulty in speaking, or even loss of speech (dysarthria or anarthria). This is to be distinguished from *aphasia*, which may be present in right-sided hemiplegia, if Broca's convolution on the left side is implicated in the lesion, but is not present in left-sided hemiplegia.

In "ingravescent hemiplegia" (tumour), unconsciousness may develop slowly and pass into complete coma. In the ordinary forms coma may be present at the outset, and pass away, or there may be only a feeling of cloudiness or bewilderment, which may pass off or deepen into coma.

The arm is usually paralysed to a greater degree than the leg, and the face least. The face usually recovers before the leg, and the leg before the arm, while coarse movements of large joints are re-established before the highly specialised movements of small joints. Improvement is therefore least in the hand and fingers. Sensation is rarely affected to a marked extent. The paralysis of the face is most notable in its lower segment, thus differing from Bell's paralysis (*see Diseases of*



Cranial Nerves). The tongue is not put out readily, and deviates towards the *paralysed* side. Whistling is impossible, and food accumulates between the gums and cheek on the paralysed side. The muscles of mastication are unaffected. In the limbs the muscles do not waste, and they respond normally to electricity. They are at first flaccid, but may become rigid a few hours or days after the initial lesion has occurred ("early rigidity"). This may pass off, or persist till "late rigidity" appears some weeks afterwards. As time wears on, certain important symptoms appear, as a result of the secondary degeneration of the motor fibres cut off from their trophic centres. The limbs, before flaccid, now become rigid, and this late rigidity is always most marked in the arm, which is adducted, flexed at the elbow, and resists extension. Frequently the wrist and fingers are also flexed. Such *contractures* are often accompanied by pain. The tendon reflexes are exaggerated, and ankle clonus is often obtained. The plantar reflex is of the extensor type (Babinski's sign). As the patient walks, the body is inclined to the normal side, and, since the foot is dropped, he either sweeps the affected leg forward in a circular arc, or flexes the knee unduly, to clear the toes from the ground. That any movement is possible after severance of the fibres from the cortical centre is due to the preservation of the subsidiary motor tracts, particularly the rubro-spinal, which controls stock movements such as those of sitting, standing, and walking. Inhibitory influences, transmitted through it from the thalamus, are, however, lost by damage to that ganglion. Hence the exaggerated reflexes, and also the spasm and rigidity, due, as has already been explained, to the increased muscular tonus produced by impulses from the vestibulo-spinal and other subsidiary tracts. After recovery from the initial coma, the sphincters are unaffected, and control over the urine and fæces is regained.

Later changes that may occur are—tremors of the affected limbs, inability to maintain one position of the fingers and toes (*athetosis*), and post-hemiplegic *chorea*. The two last are more common after infantile than adult hemiplegia. There may be vasomotor disturbances, arthritic pain and swelling, and sometimes muscular atrophy (chiefly in the hand), from nutritional disturbances of the cells of the anterior horn.

*Alternate or crossed paralysis* means paralysis of the limbs on one side, and of a cranial nerve or nerves on the other. It occurs when the lesion is in the crus, the third nerve of the same side being implicated, or in the pons, when the fifth,



sixth, or seventh nerves may be affected (*see* Diseases of Cranial Nerves, and diagram, p. 463).

**Treatment.**—Endeavour to prevent the onset of rigidity and contracture by the prolonged employment of massage, passive movements and galvanism. Massage may be begun within a fortnight, and the other measures as early as four weeks after the onset, at first while the patient is in bed. Later, “Swedish movements” may be employed. Faradism is to be avoided if there is any tendency to rigidity, and so also is strychnine, and for the same reason. Treat any general condition, such as Bright’s disease.

### CEREBRAL PALSIES OF INFANCY AND CHILDHOOD

These affections are caused by pathological changes occurring before or during birth, or in the early years of childhood. Maternal disease or injury during pregnancy, injury during birth, and after birth the infectious diseases of childhood, are among the chief causes. Recent work has shown that in many instances in which no obvious cause had hitherto been found, there was a positive Wassermann reaction either in the mother or the child. The satisfactory results of specific treatment in such cases confirm the view that they are of syphilitic origin.

**Morbid Anatomy.**—Only the late changes are well known, as death rarely occurs in the early stages. The probable antecedent conditions are mal-development, meningeal or cerebral hæmorrhage, arterial thrombosis, encephalitis, and changes due to maternal or congenital syphilis. It is always the cerebrum that is affected, and lesions may be found on one or both sides. These conditions lead in after years to (1) sclerosis of the convolutions (*microgyria*), in which the convolutions are small and the cortical cells atrophied; (2) cavity formation (*porencephaly*), which follows upon vascular thrombosis or encephalitis.

**Symptoms.**—*Hemiplegia*. In this type are found paresis, rigidity, contracture, and often tremors of one side of the body. There may be convulsions at the onset. The affected side is ill developed, and shows marked atrophy. There may be athetosis, or choreiform movements. This form arises within the first two or three years after birth, and follows infectious diseases.



*Diplegia and paraplegia (Little's disease)* constitute the more usual "birth palsies." There is often a history of difficult labour, which has given rise to *meningeal hæmorrhage*. There are paresis, rigidity, and contracture of the legs, with adductor spasm; there may or may not be paresis of the arms, which show athetoid or choreiform movements. Sometimes the lower segment of the face is implicated. The tendon reflexes are exaggerated. The gait is spastic, and there may be cross-legged progression from the adductor spasm. Deformities, such as club-foot, are frequent.

In all these forms some degree of mental impairment, which may even amount to idiocy, is commonly present. Epilepsy may also follow, particularly in the hemiplegic form, the paretic side being specially affected. This may not occur till many years have passed.

These palsies are not dangerous to life, but seriously affect growth and mental development. They are to be *treated* on general principles. The child should be educated separately, and much attention should be given to physical training. Deformities may require surgical treatment, and epilepsy prolonged treatment by bromides. If the case is seen soon after birth, the clot may be removed by operation. When there is a positive Wassermann reaction in either mother or child, energetic specific treatment should be instituted, as it often leads to remarkable improvement.

## CEREBRAL SYPHILIS

Syphilis affecting the brain may give rise to vascular lesions (obliterative endarteritis), and gummata, or to degenerative diseases. The vascular lesions may occur early—and sometimes quite early—the degenerative conditions (*see* General Paralysis) much later. The severity of the attack of syphilis has little influence on the development of nervous lesions, and they may follow even prolonged and careful treatment.

**Morbid Anatomy.**—Obliterative endarteritis causes a uniform narrowing of the lumen of the cerebral arteries and arterioles, the basilar and vertebrals, and the middle cerebral and its branches being chiefly affected. The arteries of the basal ganglia do not suffer so often. The lumen of the affected artery is ultimately obliterated by thrombosis. There is usually periarteritis, with formation of gummatous nodules. Complete occlusion of course leads to cerebral softening.



Isolated gummata—single or multiple—may be found over the cerebral convexity, at the base, on the cranial nerves, or in the substance of the brain. A diffuse gummatus meningo-encephalitis may also occur, especially at the base.

**Symptoms** are similar to those produced by other cerebral lesions, but they may be combined in a suggestive way. Signs of double or multiple lesions, a tendency to improvement and relapse, and a tendency to improvement under treatment, are all significant. Headache, usually worse at night, vertigo, insomnia, apathy, epileptiform convulsions or fits, amnesia, mental confusion, are frequent prodromata. From *vascular occlusion*, hemiplegia and aphasia may arise. The onset is gradual, generally without unconsciousness, the paralysis is often, but not always, temporary, and it may be followed by another attack on the same or the opposite side. From *cortical gummata* arise localised convulsions (Jacksonian epilepsy), with only incomplete unconsciousness, and often monoplegia. The paralysis comes on slowly, and is persistent. Optic neuritis sets in early, and is very intense. From *gummatous meningitis of the base* arises paralysis of the cranial nerves. All of these may be affected, but most frequently the third or sixth, or both of them. Optic neuritis is exceptional, but bi-temporal hemianopia from gummata in the interpeduncular space may occur. There are usually other signs of intracranial mischief (headache, vertigo, etc.). From *diffuse arterial and meningeal lesions* arises syphilitic dementia, which may be accompanied by local or general convulsions, aphasia, affections of cranial nerves, and more constantly by amnesia, mental confusion, somnolence, and melancholia or sometimes mania.

**Prognosis** depends upon the stage of the cerebral affection. If vascular occlusion has led to softening, or if fibrous transformation has occurred, there will not be much improvement; while early cases may be immensely improved, or even cured, by energetic treatment.

**Treatment.**—In cerebral syphilis the more vigorous forms of treatment are chiefly employed. Mercury is of the first importance. It may be given by inunction either of the ordinary Ung. Hydrargyri, or of Ung. Hydrargyri Oleat., which is cleaner and less irritating; or intra-muscular injection of a soluble salt, of calomel, or of mercurial cream, may be practised. Salvarsan, if given, must be given cautiously, the initial dose being small. Iodides may be given alone or in combination with mercury. The doses should be large—15 to 30 grains



(grm. 1·0-2·0) of the iodide of potassium thrice daily. Such doses are less likely than smaller doses to produce iodism. Sulphur baths aid the elimination of mercury from the system, and thus permit the use of larger doses.

## TUMOURS OF THE BRAIN

These are of three kinds—(1) Infective granulomata—tubercle, gummata, actinomyces; (2) neoplasms—sarcoma, glioma, carcinoma, and benign tumours; (3) cysts.

The most important are—

1. *Tubercle*.—The tumours may vary from the size of a millet seed to that of a small orange. Usually they are multiple, and most common in the cerebellum and about the base of the brain. The histological characters have been already described under tuberculosis.

2. *Gummata* are very common (see Cerebral Syphilis).

3. *Gliomata* or *neurogliomata* may be either dense, firm tumours, or *soft and vascular*. They consist of small cells with round or oval nuclei and branching processes in a network of delicate fibrils. Enormous, single-nucleated, spindle-shaped cells are often present. Gliomata infiltrate the substance of the brain, and cannot be sharply differentiated from the normal tissues. They are usually innocent tumours, and may remain for years without creating very marked symptoms.

4. *Sarcomata* attack the membranes and pons, but may be found in other parts of the brain. They may be large tumours, hard, fibrous, and encapsulated, or soft, infiltrating, and sometimes cystic.

5. *Carcinoma* is rare, and secondary to cancer elsewhere.

6. *Cysts* may be the result of hæmorrhage or softening, congenital defects, or hydatids.

**Symptoms** vary according to the site, size, and nature of the tumour. The more constant are—

1. *Headache*.—This may be diffuse and of a dull aching character, but is much more often acute, stabbing, persistent, and localised. There is sometimes tenderness over the seat of the tumour.

2. *Vertigo* is a marked symptom, especially when the tumour affects the cerebellar region.



3. *Vomiting* is very persistent, often painless, and occurs whether food be taken or not. It is most common when the cerebellum is affected.

4. *Double Optic Neuritis*.—"Choked disc" is almost pathognomonic. It may be slight in cortical tumours, but is present in eighty per cent. of all cases. It is followed by optic atrophy. Blindness is not necessarily present during the neuritic stage, but when atrophy appears visual acuity diminishes, and may be lost. The neuritis is often most marked on the side of the tumour.

5. *Mental Disturbance* is sometimes absent, but the patient is often, in the early stages, highly emotional, and later, dull and apathetic.

6. *Localising Symptoms* may be absent, but usually there are phenomena either of irritation or destruction of the affected part of the brain, the former leading to convulsions, paræsthesiæ, and subjective sense impressions, the latter to paralysis, anæsthesia, and defect of special sensation.

**A.** Prefrontal area. Lethargy, somnolence, stupor, dementia; often anosmia on the affected side.

**B.** Rolandic area. Jacksonian epilepsy followed by monoplegia; increased tendon reflexes on the paralysed side; motor aphasia if Broca's convolution is affected.

**C.** Angular region. Word blindness with or without hemianopia and hemianæsthesia.

**D.** Basal ganglia. Hemiplegia, hemianæsthesia, and hemianopia.

**E.** Cerebellum. General symptoms severe. Ataxia, with tendency to fall to one side; unilateral affections of cranial nerves (fifth and eighth usually); knee-jerks absent or exaggerated.

**Diagnosis.**—Headache, vomiting, and double optic neuritis are the classical symptoms of cerebral tumour, in the presence of which the diagnosis can be made with considerable certainty. The differential diagnosis between tumour, abscess, and tuberculous meningitis is given in the adjoined table, page 485.

**Treatment.**—*Medical.*—If syphilis is the cause, specific treatment (*see* p. 482) must be employed; other forms are practically unamenable to any known medicinal treatment. The iodide should nevertheless be given, as it often produces temporary benefit. Tuberculous tumours may be treated by the usual remedies.



*Surgical.*—Trephine when necessary for the relief of pressure, and remove the tumour if possible, though extirpation can be carried out in only a small percentage of cases.

Though in most cases permanent benefit is not to be hoped for, everything should be done to make the patient's life bearable, by relieving pain, etc., with opiates or by other means.

### DIFFERENTIAL DIAGNOSIS

TUMOUR.	ABSCESS.	MENINGITIS.
History indefinite.	Otorrhœa or other suppurative condition.	Tuberculous history or diathesis.
Onset gradual.	Onset usually abrupt.	Onset rapid.
Optic neuritis usually well marked.	Optic neuritis usually absent or late.	Optic neuritis rare.
Monoplegia, hemiplegia or localised convulsions, in definite order.	Focal symptoms indicative of cerebellum or temporal lobe.	Irregular palsies and convulsions.
Febrile symptoms absent.	Temperature sometimes subnormal.	Temperature irregular.
Duration, months to years; regular course.	Duration, variable with latent periods.	Duration of weeks, at times irregular.

[ALDREN TURNER.]

### APHASIA

Aphasia is a morbid condition consequent on a cortical or subcortical lesion, whereby speech, writing, or reading may become impaired, either from (1) an inability to co-ordinate the necessary movements involved in speech, or (2) from a defective interpretation of sounds or visual impressions.

To understand the various forms of aphasia it is necessary first to consider the factors employed in expressing our thoughts. Language is *gradually* learned. A child has first to become, through the auditory word centre in the first left temporal convolution, familiar with the memories of verbal sounds, and to associate them with particular objects. Attempts to reproduce the words open up a path through commissural fibres to the motor speech centre in Broca's convolution, and speech in response to speech now becomes possible. As the child is taught to read, a new centre for word memories—the visual word centre in the angular gyrus—comes into action, and the reproduction by writing of characters or words recognised by this route is



effected through commissural fibres going to the writing centre in the posterior part of the second left frontal or ascending frontal convolution. Still later co-ordination of auditory and visual centres occurs in the process of reading aloud, where the printed word is recognised by the visual centre, from which impulses pass to the auditory centre and to Broca's convolution, and in that of writing to dictation, where the spoken sound is recognised by the auditory centre, from which impulses pass to the visual centre and to the centre for writing. Thus, intelligent and rational speech, though apparently simple, involves many complex processes. It requires—

1. The aid of memory to *co-ordinate the necessary ideas* formed by incoming impressions derived through hearing, seeing, and in the case of the blind, through the sense of touch.

2. A mechanism by which these ideas can be spoken and written.

In short, speech involves a healthy continuity between the chief motor centre in the left inferior frontal lobe and the visual and auditory centres on the one hand, and between the chief motor centre and the muscles employed in speech or reading aloud on the other.

Until quite recently the scheme above indicated was generally accepted as a probably accurate representation of the facts in regard to localisation of the speech centres, and it is still the prevalent conception. The cortical centres referred to are motor and sensory, and are thus distributed:—(1) a motor (*glosso-kinæsthetic*) speech centre, in the third left frontal (Broca's convolution), or in left-handed persons the third right frontal convolution; (2) a motor (*cheiro-kinæsthetic*) centre for writing, in the ascending frontal or posterior part of the second left frontal convolution; (3) sensory centres: (a) the *auditory word centre*, in the posterior part of the first temporal convolution; (b) the *visual word centre*, in the angular gyrus. The motor centres connect with each other and the sensory centres by commissural fibres. The similar but subordinate centres in the right hemisphere have a limited capacity for taking over the functions of the left centres when these are destroyed. On the current assumption, the different forms of aphasia are to be explained by lesions of particular centres.

1. Lesions of the glosso-kinæsthetic centre cause *motor aphasia*—the patient is unable to express himself in words, although there is no paralysis of articulation, and he shows that he understands what is said to him.



2. Lesions of the cheiro-kinæsthetic centre cause *agraphia*—the patient cannot express himself in writing.

3. Lesions of the visual word centre in the angular gyrus cause *word blindness* or *alexia*—i.e. the patient can see print, but cannot read it.

4. Lesions of the auditory word centre in the temporo-sphenoidal lobe cause *word deafness*—the patient hears, but does not understand the words.

(3) and (4) are both forms of sensory aphasia, (1) and (2) of motor aphasia. But the terms are loose, for word deafness is usually followed by motor aphasia, even if the lesion be limited to the auditory word centre, and word blindness is similarly followed by agraphia.

Of late, however, much doubt has been thrown on the existence of a separate visual word centre, and the tendency of Marie and his followers is to assume one large sensory speech centre (zone of Wernicke), including the left supramarginal convolution, angular gyrus, and base of the first and second temporal convolutions. Marie even denies, on post-mortem evidence, that a separate motor speech centre exists in Broca's convolution. In his view damage to the speech centre leads to varying degrees of failure of speech as a whole, and in partial aphasia the most recently acquired and most complex elements of speech are the first to be affected. Motor aphasia is due, he holds, to a lesion of the cortical speech centre combined with interruption of the motor fibres below the cortex. The subject is highly controversial, but few go so far with Marie as to deny the part played by Broca's convolution in the production of speech.

A further distinction is drawn between *cortical* and *subcortical* aphasia. It is impossible here fully to describe the various forms, but the difference between the conditions can be made clear by an example. In *subcortical* auditory aphasia the communicating tract between the centre for hearing and the auditory word centre is interrupted, but the auditory word centre itself is intact. The patient therefore does not understand what is said, and cannot repeat words, but his auditory word memory is unaffected, and he can therefore speak spontaneously and read aloud. When the *cortical* centre is destroyed, his auditory word memory disappears, and he no longer understands his own speech, which therefore becomes an unintelligible jargon. Analogous differences exist in connection with the other varieties of aphasia. The following table indicates the chief distinctions between its motor and sensory forms.



MOTOR APHASIA.	SENSORY APHASIA (VERBAL AMNESIA).	
	WORD DEAFNESS.	WORD BLINDNESS.
<p>Patient almost completely loses power of speech. Words like oaths, "yes," or "no," may be retained.</p> <p>Understands what is said to him.</p> <p>Cannot repeat words.</p> <p>Recognises written words but cannot write them. Cannot copy print into writing, though he may copy letters (aphasia and agraphia). Rarely can write (aphasia without agraphia).</p> <p>Is aware of his errors—he can recall words but not utter them.</p> <p>Mental impairment is but slight.</p>	<p>Can still speak, sometimes with little aphasia, but sometimes merely gibberish.</p> <p>Does not understand what is said.</p> <p>Cannot repeat words.</p> <p>May be some word blindness and agraphia, or patient may recognise and write words freely.</p> <p>Is unaware of his errors of speech—auditory word memory is destroyed.</p> <p>Mental impairment is marked.</p>	<p>Speech little affected.</p> <p>Understands what is said.</p> <p>Can repeat words.</p> <p>Cannot recognise written or printed words, or write them (agraphia). May recognise letters or his own name. If the damage is partial, may write wrong words or in wrong order (<i>paragramia</i>).</p> <p>Is unaware of his errors in writing—visual word memory is destroyed.</p> <p>Mental impairment is slight.</p>

It should not be forgotten that although aphasia is most commonly due to organic lesions of the brain, and therefore permanent, *temporary* aphasia is not altogether uncommon. This occurs mainly in old people, and is held to be caused by disturbance of the cerebral circulation arising from anæmia, arterio-sclerosis, or possibly temporary spasm of the vessels.

### DISSEMINATED SCLEROSIS

Disseminated, multiple, or insular sclerosis is a disease characterised by scattered patches of sclerosis throughout the central nervous system. It may be limited to the brain, or to the spinal cord; but the *cerebro-spinal* variety is much more common.

**Etiology.**—The disease is most common in early adult life, and affects both sexes. Exposure to cold, injuries, and mental worry are possible predisposing causes. The real cause is not known. It may be a toxic agent, and the disease has frequently been noted to follow upon one or other of the specific fevers; or it may depend upon congenital abnormality of the nervous tissues. It has no connection with syphilis.

**Morbid Anatomy.**—Patches of sclerosis, sharply defined, and varying in colour from pink to ashy grey, some hard and



leathery, others soft, are found scattered throughout the white matter of the cord and the substance of the medulla and cerebrum, and sometimes the cranial nerves. Their presence causes pressure on the nerve-cells and fibres. Nerve-cells, when they are affected, undergo atrophy, but the degeneration of the nerve fibres is partial, the medullary sheath disappearing while the axis-cylinder persists. Hence there are no extensive ascending or descending degenerations.

**Symptoms.**—In the presence of widespread morbid changes of which the site is variable, it is evident that the symptoms must also vary, but in a typical case some or all of the following symptoms are to be found :—

1. *Impairment of Speech.*—The patient has a slow monotonous way of pronouncing each syllable separately (scanning or staccato speech), or in other cases there is a slurring of the ends of words.

2. *Nystagmus* or oscillation of the eyeballs, best seen on voluntary movement of the eyes. Other ocular symptoms, such as impairment of vision and partial optic atrophy, may be present. Transitory squinting or diplopia may occur.

3. *Muscular tremor*, absent when the muscles are at rest, and excited by voluntary movement (“*intention tremor*”). The tremor is coarse and jerky in character, increases during the continuance of a movement, and affects the hands first and chiefly; but the whole body, including the head, may suffer.

4. *Paresis* at first of one or both lower limbs, with *spasm* and *exaggerated reflexes*. This symptom may improve or disappear in one limb while developing in another. Rigidity of the lower limbs in the position of extension may follow later. The abdominal reflex is usually absent, and this may be an early sign.

5. *The gait* may be that of spastic paraplegia, or this may be combined with ataxy. When walking, the patient may shoot suddenly forward or to one side, and may fall, or bruise himself by bumping against various obstacles.

Disturbances of sensibility are often absent, but there may be paræsthesiæ and occasionally girdle sensation. The mental faculties may be impaired and memory may suffer. In some cases there are intercurrent apoplectiform or epileptiform attacks, from which the patient recovers in one or two days. The sphincters are not seriously affected until late, though there may be some delay in micturition.

The onset of the disease is insidious, and its course is protracted, the *average* duration being from three to six years.



It begins most commonly with spinal symptoms—those, namely, of spastic or ataxic paraplegia, and cerebral symptoms may be long deferred. But even comparatively early there is usually some indication—nystagmus, optic atrophy, absent abdominal reflex, for example—that the malady has a wider spread than a system disease of the cord. In some cases cerebral symptoms appear first, and the spinal cord is affected later. Temporary improvement is not unusual, but the general direction is downwards. In the last stages dementia is common. Death may be due to an apoplectiform attack, to extension of the changes to the grey matter, leading to paralysis of the bladder, bed-sores, etc., or to intercurrent disease.

**Diagnosis.**—The combination of nystagmus, intention tremor, and staccato speech is characteristic. The diagnosis between disseminated sclerosis and general paralysis sometimes offers difficulties, and depends on the recognition of the peculiar mental symptoms in the early stages of the latter disease, and on the presence of the Wassermann reaction, which is absent in disseminated sclerosis. In young women hysteria may cause confusion, but a diagnosis of hysteria should never be made unless organic disease can be definitely excluded.

**Treatment.**—The main principles of treatment are to secure rest and avoid over-fatigue, and to maintain the general nutrition. Tonics are of some value, but the drug which has most influence is iodide of potassium. Gentle massage and warmth to the limbs are of use in spastic conditions, and the galvanic current applied along the spine is also beneficial. Electricity to the limbs is contra-indicated, as tending to aggravate rigidity.

## GENERAL PARALYSIS OF THE INSANE

### (PARALYTIC DEMENTIA)

A chronic degenerative disease of the cortex and meninges, accompanied by progressive derangement of the mind and by progressive paresis.

**Etiology.**—The disease seldom occurs before twenty years of age, although a *juvenile form* is occasionally found in the subjects of congenital syphilis. It begins most frequently in the thirties, and the average age at death is forty. It affects all classes, but chiefly those living in large towns. Men are much oftener attacked than women. *Syphilis* is the essential cause of general paralysis. Supposed until recently, like locomotor ataxia,



to be a parasymphilitic affection, it is now shown to be due to the presence of spirochætes in the tissues of the brain. The Wassermann reaction is positive in the cerebro-spinal fluid in about 95 per cent. of the cases. Mental worry or overwork, alcoholism, and injury to the head are predisposing causes.

**Morbid Anatomy.**—The following are the most important changes :—

1. Thickening of the membranes, with hæmorrhages between the brain and dura mater.
2. Atrophy of the frontal and central lobes.
3. Degeneration of cerebral blood vessels.
4. Distension of the ventricles by fluid.

*Microscopically*, the following changes are found :—Thickening of the pia arachnoid, overgrowth of the neuroglia, partial or complete destruction of the nerve-cells. The association fibres are absent or greatly diminished. These changes, in Mott's view, are the late result of cortical irritation and meningo-encephalitis with perivascular infiltration, conditions which lead to chronic necrosis of nerve-cells and proliferation of the neuroglia. Bolton holds that syphilis is not the sole essential cause of general paralysis, but that it occurs in such syphilitics as possess "cortical neurones of subnormal durability," and would, if uninfected, develop in any case a moderate degree of non-symphilitic dementia.

**Symptoms.**—The disease begins insidiously, sometimes a few years, sometimes as long as twenty years, after infection, the average interval being about ten years. Its characteristic symptoms are progressive loss of intelligence, defect of speech, paralytic attacks, and pupillary changes. In the early stage headache, irritability, and alternating excitement and depression are frequent. The finer qualities of the intelligence are the first to go—a man of previously correct behaviour becoming untidy, careless of his person, neglectful of ordinary politeness, or heedless of decency. *Memory*, especially of the immediate past, is always markedly impaired, and the power of mental calculation is much affected. The facial expression in repose is often vacant. The mental state varies; in many instances there are *hallucinations of grandeur*, in which the patient imagines himself enormously powerful or enormously rich, disposing, under the latter impression, of much of his capital if the disease is not early recognised; in other instances there is from the first a state of



apathetic dementia, or there may be hypochondriacal delusions. Excitability, in the early stages, may approach mania.

The *Argyll-Robertson* pupil is present. This sign appears early, and may precede mental change. The pupils are often unequal.

The *speech* is slurring or blurred, and there is a tendency to drop syllables out of long words. *Tremor of the lips and tongue* is frequent, and there is often tremor of the hands. Writing is at first tremulous, afterwards syllables are omitted, and finally the script becomes meaningless.

Symptoms of implication of the posterior columns are often present (absent knee-jerks, lightning pains, optic atrophy, etc.); but even more commonly affection of the lateral columns with exaggerated tendon jerks and slight paresis. This may even precede the mental change. Fatigue is readily induced.

At a later stage, as a rule, *paralytic attacks* appear. They may be either apoplectiform or epileptiform in character, and leave behind them transitory monoplegia, hemiplegia, or aphasia, and a deteriorated mental condition. The last stage is one of complete imbecility and paralysis, with loss of control of the sphincters. Death may be due to bedsores, inanition, cystitis, or other inflammatory complications, or it may occur in one of the paralytic seizures. The average duration is two or three years, but some cases last for ten. Remissions are frequent and deceptive.

There are many departures from the typical course, and, as we have seen, motor symptoms may precede the mental defect, or indications of ataxia may be very prominent. The tremor of the lips and tongue is an important element in the diagnosis, and, along with the *Argyll-Robertson* pupil and the altered mental condition, leaves little room for doubt. Where the mental symptoms are slightly marked, the diagnosis may be aided by lumbar puncture, the cerebro-spinal fluid showing lymphocytosis, an increase of globulin, and a positive Wassermann reaction.

**Treatment.**—The patient must in some instances be confined to an asylum; in other cases careful supervision, hygiene, and diet may retard the progress of the disease. If the case is recent, specific treatment should be employed, and salvarsan has been credited with good results; but it must be remembered that nerve-cells once lost are never replaced, and that remissions are apt to simulate improvement. Cold to the head, bromides, or hyoscine may be used for the maniacal excitement.



## VI. DISEASES OF PERIPHERAL NERVES

## CRANIAL NERVES

All the cranial nerves have at least two origins, deep and superficial; all behind the fourth nerve may be said to have their deep origin in the fourth ventricle. The nerves of the special senses—*i.e.* first (smell), second (sight), eighth (hearing), have in addition a special connection with the so-called “centres,” situated in the cortex of the brain. The sense of taste is omitted from this list, because, although the glosso-pharyngeal carries the fibres for the posterior third of the tongue and the palate, there is no proof that it carries any taste fibres at its exit from the medulla, and there is much evidence in favour of the fifth being the nerve most implicated. Division of the fifth above the Gasserian ganglion results in paralysis of taste on the affected side, even when the glosso-pharyngeal remains intact. For the normal perception of impressions derived from the special senses, there must evidently be a healthy continuity between *centre*, *nerve trunk*, and *special ending*; destruction of any of these will interfere with the carrying out of the function.

## OLFACTORY OR FIRST PAIR

**Anatomy.**—The true olfactory nerves issue from the olfactory cells of the nasal mucous membrane, and ascend through the cribriform plate of the ethmoid bone to the olfactory bulbs. There they arborise round the dendrons of the so-called “mitral cells,” the axons of which pass along the olfactory track to the cortical centre in the uncinate gyrus.

**Physiology.**—The nerves of smell. The appreciation of pungency should be carefully distinguished from the sense of smell, or the detection of odours. The perception of pungency is due to painful stimulation of the fifth nerve in the *lower* part of the nose.

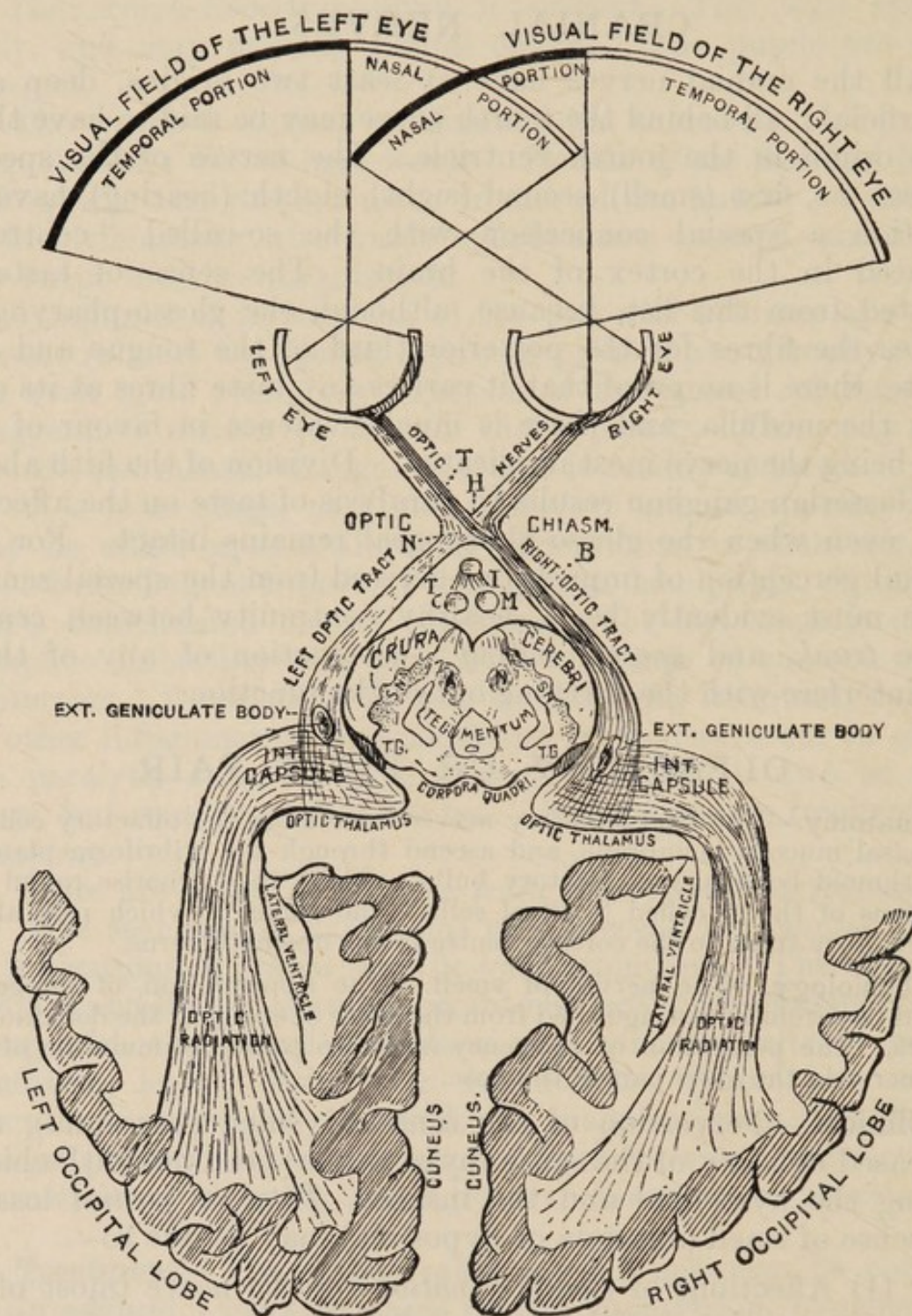
**Clinical.**—Perversion of the sense of smell (*parosmia*), and increased delicacy of the sense (*hyperosmia*), are met with chiefly among the hysterical and the insane. *Total or partial* loss of the sense of smell (*anosmia* or *hyposmia*) may be due to—

- (1) Affections of the *terminations* of the nerve (most often associated with nasal catarrh or polypi).
- (2) Lesions of the *nerve trunk*.
- (3) Lesions of the *centre*, in the uncinate gyrus.

## OPTIC NERVES

**Anatomy.**—Commencing in the retina, the fibres pass backwards in the optic nerve through the choroid and sclerotic to





THE OPTIC AND VISUAL TRACTS (Starr).

N, Lesion causing nasal hemianopia. T, Lesions causing temporal hemianopia.  
 H, Lesion causing bilateral heteronymous hemianopia. B, Lesion of tract causing homonymous hemianopia.



the optic foramen, and thence to the optic commissure, where the nerve proper terminates. Thus we get, on tracing backwards—

1. Optic nerves.
2. Optic chiasma or commissure, where *partial* decussation takes place.
3. Optic tracts.
4. Ganglionic centres { Optic thalamus.  
External corpora geniculata.  
Anterior corpora quadrigemina.
5. Cortical centres in the occipital lobes, connected with the ganglionic centres by the optic radiation.

The fibres from the inner side of each retina cross in the commissure to the optic tract of the opposite side; those from the outer part turn backwards into the tract of the same side (*see diagram*).

#### Clinical—

1. Affections of the *terminations* in the retina include—

(a) Functional disturbances due to toxic effects, viz. :—

- (1) Uræmia.
- (2) Jaundice.
- (3) Drugs—quinine, santonin, etc.

(b) Hysterical amaurosis.

(c) Night blindness—(nyctalopia). Objects are clearly seen by day, but not in the dusk; or (hemeralopia), objects are *easily* seen in the shade, but in the sunshine with difficulty.

(d) *Retinitis*, which may occur in a number of general diseases, chiefly such as affect the condition of the blood. The most common are chronic Bright's disease, leukæmia, pernicious anæmia, and syphilis. *Retinitis* is sometimes present in diabetes and in purpura.

2. Lesions of the *Nerve*.

(a) Optic Neuritis.—The disc becomes blurred, swollen and red from congestion; there may be slight extravasations of blood; finally, there is an increase in the fibrous tissue, and atrophy of the nerve elements, causing often total blindness. Optic neuritis is common in tumours of the brain and cerebellum, cerebral abscess, and cerebral meningitis, and may be present in chronic Bright's disease, diabetes lead-poisoning, and severe anæmias.



The blindness is preceded by changes in three directions :—

- (1) Diminished acuity of vision.
- (2) Contraction of the visual *field*.
- (3) Contraction of the colour field.

There may be a considerable degree of optic neuritis before vision is materially interfered with.

Tobacco amblyopia is due to a *retrobulbar* neuritis. The loss of sight is gradual, and at first there is no change in the fundi, but if the condition persists, there may be *permanent* changes in the optic discs.

- (b) *Primary Optic Atrophy* occurs in locomotor ataxy, and sometimes in disseminated sclerosis, general paralysis, and other nervous affections. A partial atrophy may occur in those who smoke excessively.

3. Lesions of the *Chiasma*.—If the lesion is confined to the central portion, *i.e.* the decussation, as in tumours of the pituitary body (acromegaly), the fibres passing to the *inner* or nasal portion of each retina will be involved, causing blindness of the *outer* half of each field—*temporal* hemianopia. If it is at the lateral part on one or other side, it will cause a unilateral *nasal* hemianopia, and if there is such a lesion on both sides, a bilateral nasal hemianopia results. If the lesion is more extensive there may be total blindness of the eye on the same side, with temporal hemianopia of the opposite side ; or a more extensive lesion still may cause total blindness.

4. A lesion *behind the Chiasma*, whether in the optic tracts, optic thalamus, corpora quadrigemina, or geniculate bodies, causes *homonymous hemianopia*, that is, blindness of the *temporal* half on the same side, and the *nasal* on the opposite side.

5. A lesion in the occipital *Cortex* also causes homonymous hemianopia ; but there is, in addition, word blindness, *i.e.* the patient *can see*, but fails to *read letters*. (See Aphasia.)

### THIRD, FOURTH, AND SIXTH NERVES

**Anatomy.**—The third nerve arises from the floor of the aqueduct of Sylvius, passes through the tegmentum of the crus on the inner side, and forwards through the outer wall of the cavernous sinus to be distributed to—

1. The sphincter pupillæ.
2. The ciliary muscle.
3. All the muscles of the eyeball except the superior oblique and external rectus. *It also supplies the levator palpebræ superioris.*



**Physiology.**—The nerve is concerned in the accommodation of vision and in certain movements of the eyeballs.

## PARALYSIS.

1. Ptosis or drooping of the upper lid.
2. Divergent squint, with inability to move the eye inwards, downwards, or upwards.
3. Loss of accommodation.
4. Paralysis of sphincter pupillæ = dilatation of pupil (*mydriasis*).
5. Double vision.

## IRRITATION.

1. Convulsions or spasm of the muscles.
2. Convergent squint.
3. Contraction of pupil (*myosis*).
4. Great interference with field of vision.

**Clinical.**—Symptoms due to paralysis or irritation of the third nerve are common in many intracranial affections; for instance, the former condition is usually well marked in fractures of the base of the skull, the “*compression*” stage of meningitis, etc.; whilst *irritation* of the nerve may be brought about by tumours, and occurs in the *first stage* of meningitis, and sometimes in hysteria.

It should be noted that only a *few* of the great number of fibres running in the third nerve may be affected, and the clinical phenomena will obviously vary with the number of fibres involved. We shall first consider the fibres to the iris—

I. *Paralysis of the Iris* may be of three types—

1. Accommodative iridoplegia—failure to accommodate for near vision; commonly seen in diphtheritic paralysis.

2. Reflex iridoplegia—failure of reaction to light, while the response to accommodation is preserved. This constitutes the Argyll-Robertson pupil, a most important symptom in locomotor ataxy and in general paralysis of the insane. In this case, the lesion probably interrupts the fibres between the corpora quadrigemina and the centre for the sphincter iridis in the third nucleus.

3. Loss of the skin reflex—failure of the pupil to dilate when the skin of the neck is pinched—is due, not to involvement of the third nerve, but to paralysis of the sympathetic in the neck.

II. *Paralyses of the Ocular Muscles.*

1. Ptosis or paralysis of the levator palpebræ.
2. Strabismus or squinting.
3. Paralysis of the whole nerve.

The description of strabismus is deferred until the anatomy of the fourth and sixth nerves has been discussed. The *symptoms* of paralysis of the *whole nerve* have already been



mentioned. There is a rare form of it, *recurrent* paralysis, which is unilateral, associated with unilateral attacks of migraine, and periodic. It is most frequent in women. With this possible exception, paralysis of the whole nerve is of organic origin, and

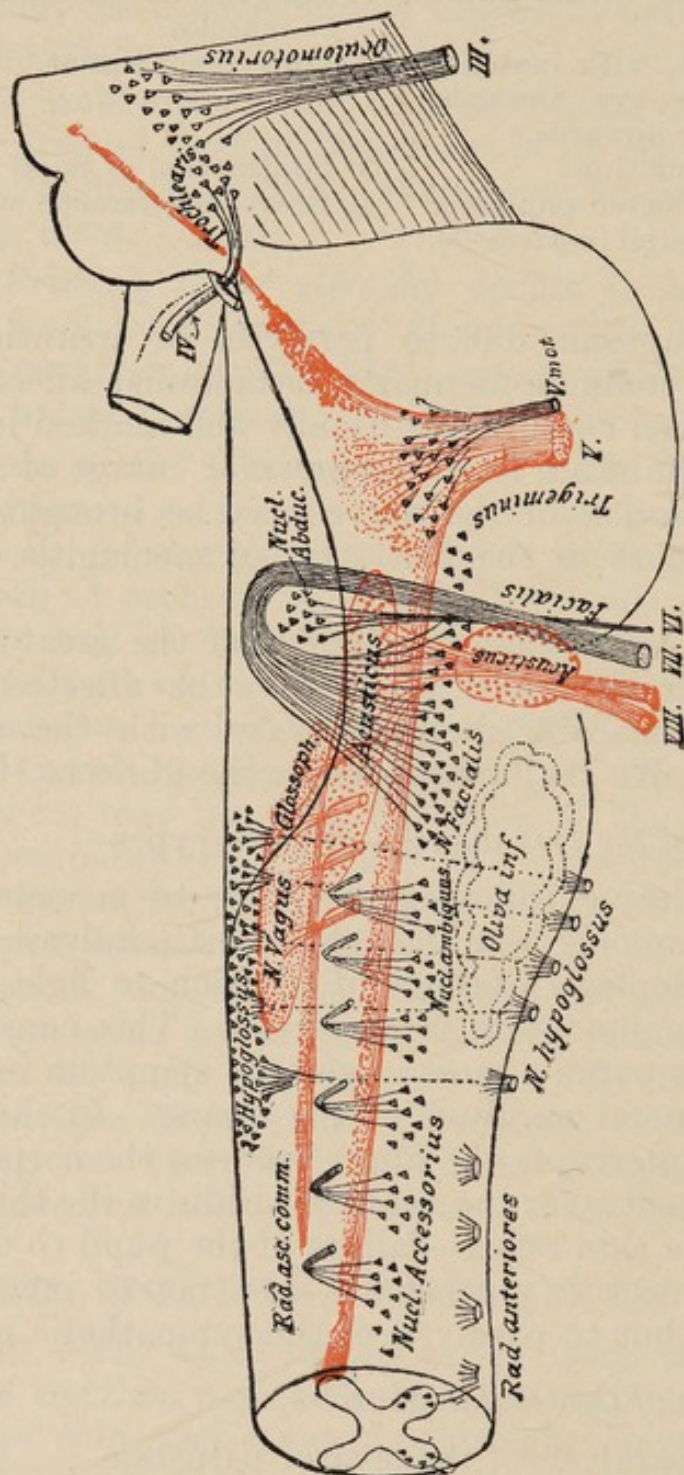


FIG. 27.—NUCLEI OF ORIGIN OF THE CRANIAL NERVES.

may be due to disease of the trunk (meningitis, gumma, neuritis) or of the nucleus. When it is due to a lesion in the crus, the nerve is paralysed on the side of the lesion, and the limbs on the opposite side (crossed paralysis).

*Ptosis* may occur independently of paralysis of the other



parts of the nerve. It may be congenital or due to hysteria, it sometimes occurs in locomotor ataxy, and it may be a symptom of cerebral lesions.

**Fourth Nerve.**—The nucleus of the fourth nerve is almost continuous with the lower end of that of the third. The nerve runs thence to the upper part of the roof of the fourth ventricle, where it decussates with the opposite fourth nerve. It then runs round the outer side of the crus to the base of the brain, and through the wall of the cavernous sinus to the orbit, ending in the superior oblique, which turns the eyeball downwards and outwards.

**Sixth Nerve.**—Arising from a nucleus in the floor of the fourth ventricle, it runs through the pons, and appears in the groove between the pons and medulla near the middle line. In the cavernous sinus it lies close to the internal carotid. It ends in the external rectus, which turns the eyeball outwards. Fibres pass in the posterior longitudinal bundle from its nucleus to the opposite third nerve and internal rectus.

*Squinting or Strabismus.*—In order to have perfect sight, there must be perfect harmony between all the ocular muscles; for instance, when we look at an object to the right, we turn *both* eyes in that direction, and thus we call into action the *external* rectus (sixth nerve) of the *right* eye, and the *internal* rectus (third nerve) of the *left* eye. In other words, on looking to the right side, we employ the services of two nerves, the third and sixth. Other movements will involve other combinations, such as the third and fourth nerves, third, fourth, and sixth, etc. Obviously, then, there must be an arrangement for the harmonious or co-ordinated actions of the third, fourth, and sixth nerves; and their nuclei are in fact connected by means of fibres of the posterior longitudinal bundle beneath the aqueduct of Sylvius. A failure of this harmonious action produces, on certain movements of the eyes, an alteration in vision, termed *strabismus*, characterised by—

1. Limitation of movement in certain directions.
2. Diplopia or double vision—*i.e.* the appearance of a *true* and *false* image of the same object. It is useful to remember that in all cases of paralytic squint, the *false image* is displaced *in the direction of traction* of the paralysed muscle, and the eye itself in the opposite direction.
3. Erroneous projection—*i.e.* errors in judgment of the position of objects, which to be seen require the use of the weakened muscle. The greater energy exerted for its contraction causes an over-estimation of the distance.
4. Secondary deviation. If an object be fixed by the affected eye while the sound eye is covered, the latter makes a still wider movement in the same direction. The increased effort required



to move the weakened muscle of the affected eye is responded to by an excessive movement of the muscles of the sound eye, which act in combination with it.

### RESULTS OF PARALYSIS OF SPECIAL OCULAR MUSCLES WHEN THE OTHERS ARE SOUND

Paralysis of Rectus Superior: inability to raise the eyeball properly above the horizontal level: the pupil may diverge somewhat downwards, and a little outwards (from action of the rectus inferior and the obliqui).

Paralysis of Rectus Inferior: inability to lower the eyelid properly below the horizontal level; the pupil may diverge somewhat upwards, and a little outwards (from action of the rectus superior and the obliqui).

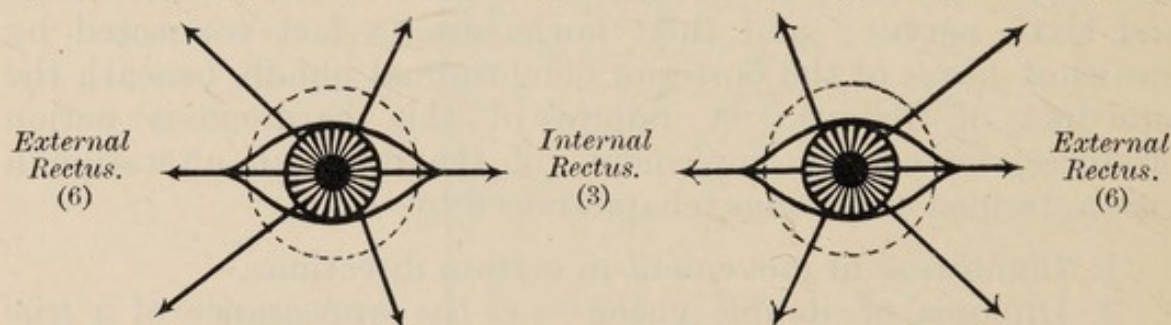
Paralysis of Rectus Externus (sixth nerve): inability to turn the eyeball properly outwards; the pupil diverges inwards (from action of the rectus internus).

Paralysis of Rectus Internus: inability to turn the eyeball properly inwards; the pupil diverges outwards (from action of the rectus externus).

Paralysis of Obliquus Superior (fourth nerve): but little alteration in movements of the eyeball; slight deviation of the cornea upwards and inwards, or simply upwards.

Paralysis of Obliquus Inferior: but little alteration in movements of the eyeball; slight deviation of the cornea downwards and inwards. (Paralysis of the sphincter of the iris, giving rise to a moderate dilatation of the pupil, and paralysis of the accommodation, often accompany this form of paralysis; this depends on the branch to the lenticular ganglion being given off from that branch of the third nerve which goes to the inferior oblique muscle. Occasionally, however, the lenticular branch arises from the sixth nerve.)—Finlayson's *Manual*.

*Inferior Oblique (3). Superior Rectus (3). Superior Rectus (3). Inferior Oblique (3).*



*Superior Oblique (4). Rectus Inferior (3). Rectus Inferior (3). Superior Oblique (4).*

FIG. 28.—Diagram to illustrate the directions towards which the pupil is moved by the separate action of the six muscles of the eyeball. The eyes are turned inwards and outwards by the external and internal recti; the internal rectus of one side is the yoke-fellow of the external rectus of the opposite side in these conjugate movements. The eyes are turned upwards by the superior rectus and the inferior oblique, downwards by the inferior rectus and superior oblique.

The eyes are represented facing the observer; if it be imagined that the eyes are looked at from the back, so that the diagram right-left becomes left-right, it will serve to illustrate the displacements in the field of vision caused by paralysis of individual muscles and nerves.—Waller's *Physiology*.



Strabismus is a frequent cause of headache, and many of the failures in the treatment of headache are due to the neglect of testing the visual apparatus, and thereby failing to rectify "eye-strain."

*Nuclear ophthalmoplegia* is a paralysis affecting the ocular muscles from causes implicating the nuclei of the nerves, and is therefore of central origin. It may be *internal*, when the ciliary muscle and the sphincter pupillæ are alone affected, *external* when the ordinary movements of the eyeball are involved, or *total*, when there is absolute immobility of the globe, and the pupillary movements are lost. It is usually chronic, and very frequently due to syphilis, but it may set in acutely as the result of hæmorrhage or inflammation.

*Nystagmus* is a clonic rhythmical contraction of the muscles of the eyeballs, giving rise to a rapid oscillatory movement, usually from side to side, but often also in a vertical direction. It is best seen on asking the patient, while keeping his head fixed, to follow with his eyes the movement of the examiner's finger. It occurs in childhood as the result of diseases of the eyeball interfering with vision; it is common in albinos; it is found in miners working with insufficient light in a cramped position, as the result of exhaustion of the ocular muscles; and it is a symptom of disseminated sclerosis, hereditary ataxy, and many varieties of cerebral disease, including tumours of the pons and cerebellum. The movement is usually bilateral, and it ceases during sleep.

The symptom has evidently no localising significance, as it occurs in lesions in many different situations, but it is of value in distinguishing between organic and functional disease. Nystagmus does not occur in hysteria.

*Irregular spasm* of ocular muscles may occur in lesions at the base of the brain (meningitis), and sometimes in hysteria. *Conjugate deviation* has been referred to under Hemiplegia. When the lesion is destructive, the deviation is away from the paralysed side and towards the lesion; when it is *irritative*, on the other hand, as in epilepsy, the deviation is towards the side on which the convulsion is greatest.

## FIFTH CRANIAL NERVE

**Anatomy.**—The fifth cranial nerve has a most extensive origin. The motor root arises from the floor of the fourth ventricle, and also from the outer wall of the aqueduct of



Sylvius; the sensory arises from the Gasserian ganglion and ends in cells in the lower part of the pons and medulla. The nerve in front of the Gasserian ganglion divides into three sensory branches—

1. Ophthalmic.
2. Superior Maxillary.
3. Inferior Maxillary, with which the motor root becomes fused in the pterygo-maxillary region. The two first divisions, therefore, are entirely sensory; only the third is a mixed nerve.

By means of these branches, the fifth nerve has connections with four important ganglia—viz., the ophthalmic branch with the ophthalmic or lenticular ganglion, the superior maxillary with Meckel's ganglion, and the inferior maxillary with the otic and submaxillary or lingual ganglia.

The *ophthalmic branch* passes to the orbit through the sphenoidal fissure. It carries with it fibres from the sympathetic, which pass through the lenticular ganglion to innervate the dilator pupillæ. It is the sensory nerve of the skin of the upper eyelid and upper palpebral conjunctiva, forehead, front of the scalp to the vertex, mesial part of the skin of the nose; of the eyeball and lachrymal gland; and of the anterior and upper part of the nasal mucosa.

The *superior maxillary branch* passes through the foramen rotundum to the infra-orbital canal, being connected in the spheno-maxillary fossa with Meckel's ganglion. From the ganglion the great superficial petrosal runs backwards to join the facial. The nerve supplies the skin of the cheek, upper lip, side of nose, lower eyelid, and lower palpebral conjunctiva; the mucosa of the upper lip, upper cheek, upper jaw, hard and soft palate, uvula, tonsil, upper part of pharynx; the upper teeth, part of the nasal mucosa, and that of the middle ear. It contains some fibres of taste.

The *inferior maxillary branch* passes through the foramen ovale with the motor root. The motor and sensory parts become fused together, and the motor fibres of the trunk thus formed supply the temporal muscle, the masseter, both pterygoids, the anterior belly of the digastric, the mylohyoid (muscles of mastication), and the tensor tympani. The sensory fibres supply the skin of the posterior part of the temple, part of the outer ear, the lower lip, lower cheek, chin, lower teeth and gums, tongue, floor of the mouth, and salivary glands. The lingual branch of the nerve is joined by the chorda tympani from the seventh. The auriculo-temporal is joined by a branch from the ninth (glosso-pharyngeal), which it sends to the parotid gland.

*Taste Fibres.*—The lingual branch of the inferior maxillary supplies the anterior two-thirds of the tongue. The taste fibres which it contains, however, pass from the lingual to the chorda tympani, which runs with the facial to the geniculate ganglion, and thence by the great superficial petrosal to Meckel's ganglion, where they enter the second division of the fifth. The glosso-pharyngeal supplies the posterior third of the tongue, but its taste fibres pass from it (through the petrous ganglion) by way of its tympanic branch (Jacobson's nerve) to the small superficial petrosal, and thence through the otic ganglion to the third division of the fifth. Section of



the whole fifth nerve above the Gasserian ganglion destroys taste on the whole of the affected side.

Paralysis of the *whole* nerve causes—

1. Loss of sensation to the parts supplied.
2. Paralysis and atrophy of all the muscles supplied, with R.D.
3. Diminished secretion of tears, absence of the corneal reflex, and sometimes inflammatory changes in the cornea, due to anæsthesia (irritating particles are not felt, therefore not removed).
4. Destruction of taste in the affected side of the tongue. The *fifth* is the nerve of taste, and the other nerves—viz., seventh and ninth—administer to taste *only inasmuch as they contain fibres from the fifth*.
5. Inability to detect pungent odours.

*Paralysis may be due to—*

1. Disease of the pons.
2. Injury or disease at the *base* of the brain, medulla, etc.
3. Pressure from tumours, etc., on the branches as they pass through their foramina.
4. Peripheral neuritis. Paralysis due to neuritis (inflammation of the ganglion) is attended by *trophic changes*, such as herpes zoster and greyness of the hair. It is also associated with intense *pain*, which may persist for years.

A reference to the distribution of the nerve will give the symptoms attendant on paralysis of the whole nerve or of its different branches.

*Neuralgia* may occur in all the branches of the fifth nerve (*see Neuralgia*).

*Progressive unilateral atrophy* of the face (facial hemiatrophy), a rare disease of childhood and youth, may be due to disease of the fifth, or to a sympathetic vasomotor affection.

## SEVENTH NERVE

The seventh or facial nerve arises from a nucleus in the lower part of the pons, and emerges in the groove between the olive and restiform body. It enters the internal auditory meatus with the auditory nerve, passes through the Fallopian aqueduct, and leaves the skull by the stylo-mastoid foramen. At the geniculate ganglion it is joined by the great and small superficial petrosal nerves. Within the aqueduct it gives off a branch to



the stapedius, and the chorda tympani (*see* Fifth Nerve). The nucleus is connected by motor fibres with the cortex of the fissure of Rolando. Briefly, its distribution is as follows :—

1. *Motor* to—

- (1) All the muscles of the face except the levator palpebræ.
- (2) The stylo-hyoid, the posterior belly of the digastric, and the platysma myoides.

2. *Secretory* to the submaxillary and sublingual glands, and the glands of the mouth and tongue.

3. *Sensory*.—In the Fallopian aqueduct it is associated with the chorda tympani, and a lesion there will consequently destroy the sense of taste in the anterior two-thirds of the tongue.

*Paralysis* of the seventh nerve causes—

1. *Motor* changes—

The face is drawn to the opposite side.

The eye is wide open and unwinking.

The cheek puffs out with expiration.

Food collects between cheek and teeth.

Absence of wrinkles.

2. *Sensory* changes—

Loss of taste in the anterior two-thirds of the tongue, if the nerve is affected in the aqueduct.

**Clinical.**—Paralysis may be due to lesions affecting—

1. The cortical fibres—supranuclear paralysis.
2. The nucleus itself.
3. The nerve trunk in its tortuous course through the pons and bony canals, or after its exit from the stylo-mastoid foramen (Bell's paralysis).

The causes of the *cortical* (supranuclear) form are those of hemiplegia ; of the *nuclear*, hæmorrhage, tumour, or extension of chronic spinal diseases ; and of the *peripheral*, extension of middle-ear disease, injury to the bones of the skull, exposure to draughts and cold, and “rheumatic neuritis.”

The features of the paralysis will vary with the site of the lesion.

When the lesion is in the cortical fibres, the facial paralysis is associated with hemiplegia, and is on the *opposite side from the lesion*, that is, on the *same side* as the paralysed limbs ; but either because the upper branches of the nerve escape, or because movements habitually bilateral are but slightly affected in upper neuron paralysees, the orbicularis palpebrarum is *not* involved, and thus *movements of the eyelids are not affected*—i.e., winking is possible, and the eye can be closed.



If the lesion be in the *lower* section of the pons, it will involve the facial fibres *between* the nucleus and their emergence in the pons. Thus we get *crossed paralysis*—i.e. the face is paralysed on the *same* side as the lesion, the arms and legs (as before) on the opposite side.

**Symptoms.**—When the nerve is affected after it leaves the stylomastoid foramen (Bell's paralysis), the affected side is immobile, smooth, and expressionless. The eye cannot be shut, and usually waters freely. The brow cannot be wrinkled. The attempt to smile or to whistle brings out the contrast between the two sides. Articulation of labial consonants is defective. The food collects between the cheek and teeth, owing to buccinator paralysis. The skin of the neck cannot be voluntarily moved (paralysis of the platysma).

If the affection is *within the aqueduct*, there are in addition loss of taste in the anterior two-thirds of the tongue (chorda tympani), and hypersensitiveness to sounds (*hyperacousis*) especially of low pitch, from paralysis of the stapedius.

If the nerve is affected between *the pons and the geniculate ganglion* there is no loss of taste, but deafness on the same side is almost always present from paralysis of the auditory nerve. If it is affected *in the pons*, there is no deafness or loss of taste, but there is usually paralysis of the sixth, as the facial fibres form a loop around its nucleus.

The electrical reactions are highly important. When there are *no polar changes, the case recovers rapidly*. If the reaction of degeneration be present, the prognosis is much more unfavourable, and recovery, which is incomplete, may be followed by contracture of the weakened muscles. To sum up—

In the *nuclear or subnuclear* form of paralysis the whole of the one side of the face is involved, and typical "Bell's paralysis" results.

When the paralysis is of *cortical* origin, it is associated with hemiplegia, and—

1. *The upper part of the face escapes*, or is but slightly implicated.
2. *Voluntary* movements are more affected than *emotional* movements.
3. The paralysed muscles do not atrophy, nor are their electrical reactions altered.

When the lesion is in the lower portion of the *pons*, we get *crossed paralysis*, but of the nuclear type, as regards the facial muscles affected.



**Treatment.**—Remove if possible any pressure on the nerve by causing absorption of inflammatory products. Where the cause is syphilis, use specific remedies. Treat any ear disease, surgically if need be. In rheumatic cases, apply a blister over the point of exit of the nerve, and give diaphoretics and a smart purge. Maintain the tone of the paralysed muscles by massage and by a moderately strong faradic or continuous current applied for about a quarter of an hour several times daily, the positive pole being on the nape of the neck. Iodide of potassium may be given internally. The peripheral type is usually very amenable to treatment. In obstinate cases an anastomosis may be made between the spinal accessory or hypoglossal nerve and the distal portion of the facial; but the operation is a severe one, and the improvement produced is often only partial.

*Facial spasm* may be due to organic disease, but is more commonly idiopathic. It affects women oftener than men, and occurs in adult life. It is frequently associated with other signs of a nervous predisposition, such, for example, as hysteria or migraine. The spasm is generally clonic, but sometimes twitching may alternate with a tonic contraction. The orbicularis palpebrarum alone may suffer, but usually there are repeated rapid twitchings of the muscles about the mouth, associated with a swift blinking movement of the eyelids. The affection is generally unilateral, and the prospect of cure is not good, though temporary improvement may take place. Treatment consists in removing any cause of reflex irritation, and in the employment of nerve tonics, sedatives, or electricity.

## EIGHTH OR AUDITORY NERVE

**Anatomy.**—The eighth nerve has two divisions, associated with nuclei in the floor of the fourth ventricle. Its vestibular root, connected with the semicircular canals, enters the medulla internal to the restiform body, and passes backwards to the vestibular nucleus. The cochlear root passes, external to the restiform body, from the cochlea to the medullary striæ. It is the true auditory nerve; while the vestibular root is the nerve of equilibrium.

**Physiology.**—The nerve fulfils two functions—

Its cochlear part is the nerve of hearing, or that by which sound undulations are conducted from the labyrinth to the cortex, and there analysed. The nucleus of the cochlear nerve is connected, through the central auditory tract, with the posterior quadrigeminal and internal geniculate bodies. Thence fibres pass to the cortical auditory centre.



Its vestibular part, by means of its fibres in connection with the ampulla and semicircular canals, transmits impulses from the oscillations of the endolymph in those regions, to the *cerebellum*, with which through the nucleus of Deiters it is indirectly connected, and thus assists the co-ordinating function in the maintenance of the equipoise of the body.

Paralysis of the whole nerve causes total deafness, often attended by certain unpleasant subjective and objective symptoms.

Excitation causes unpleasant noises often accompanied by intense giddiness, nausea, and oscillations of the eyeballs (nystagmus).

It is important to remember the close sympathy and relation that exist between the cerebellum and the optic, third, and auditory nerves.

**Symptoms** connected with the eighth nerve may be due to—

1. Functional disturbances. Such symptoms as noises, buzzing, or ringing of the ears (*tinnitus*), are often due to local causes—accumulation of wax, altered tympanic pressure, etc.

2. Lesions affecting the cortical centre, causing word deafness—*i.e.* spoken words are heard as noises, and not understood. (This condition may be associated with “word blindness,” *which see.*)

3. Lesions of the nerve at the base of the brain, causing permanent deafness. These are generally associated with lesions of the seventh nerve.

4. Disease of the labyrinth of the ear. The symptoms will depend on the extent of the lesion: there may be—

- (1) Hypersensitiveness.
- (2) Diminished hearing.
- (3) Total deafness.

Affections of the nerve cannot always be distinguished from those of the labyrinth, as both may cause “nerve-deafness.” This is tested by the absence of bone conduction of sound.

The subjective symptoms are typically brought out in—

5. *Ménière's Disease*.—An affection characterised by sudden and intense nausea, vomiting, vertigo, noises in the ear, and partial nerve-deafness of one ear. As we have already mentioned, nystagmus is frequently present. The attacks occur paroxysmally. The symptoms are due to disturbance of intralabyrinthine pressure, caused by—

1. Hæmorrhage into the labyrinth or semicircular canals.



2. Inflammation of the labyrinth.
3. Pressure of basal tumours or gummata upon the nerve.

**Treatment.**—In any affection of this nerve first try to remove any discoverable cause. If the condition is associated with gout or syphilis, treat these diseases primarily. Keep the external meatus clean, as deafness, giddiness, etc., are often caused by dried wax pressing on the drum. During an attack of Ménière's disease, the patient should be kept quiet in a darkened room; a brisk purge may be administered; and antipyrin, with an effervescing mixture containing caffeine, may be prescribed. Blisters over the mastoid process, repeated once a week, should be applied. Bromide of potassium in full doses is often useful. Salicylates may also be tried, or quinine pushed to the stage of cinchonism. When the nerve is affected through disease of the bony canal, surgical measures may be necessary.

## NINTH, TENTH, ELEVENTH, AND TWELFTH NERVES

These nerves arise from nuclei in the lower part of the floor of the fourth ventricle, the twelfth near the middle line, the rest laterally. The eleventh is connected with the anterior cornu of the grey matter of the cord as low as the fifth cervical nerve. Its "bulbar part" really belongs to the vagus or tenth nerve, as it arises from a continuation of the same nucleus, whilst the "spinal part" (the true spinal accessory) arises from cells in the cervical cord. The ninth, tenth, and "bulbar part" of the eleventh (really part of the tenth) leave the side of the medulla behind the olivary body. The spinal accessory enters the skull by the foramen magnum. The twelfth leaves the front of the medulla in front of the olivary body. They all lie in the lower part of the posterior fossa of the skull. The twelfth enters the neck by passing through the anterior condyloid foramen, the rest through the jugular foramen. As they enter the neck they all lie between the internal carotid and the internal jugular vein, and are connected by communicating branches. The ninth and tenth are mixed nerves, the others motor.

The *ninth or glosso-pharyngeal* is a sensory nerve for the posterior third of the tongue, soft palate, fauces, and pharynx; motor for the middle constrictor of the pharynx and stylo-pharyngeus; inhibitory for movements of deglutition. The *tenth, vagus, or pneumogastric*, supplies the pharynx, larynx, heart, lungs, œsophagus, stomach, and in part the intestines and spleen. It sends an auricular branch to the skin of the outer ear. Its lowest fibres of origin ("bulbar part" of the spinal accessory) are motor fibres for larynx and pharynx, and inhibitory fibres for the heart. Its pulmonary fibres are motor for the bronchial muscles, sensory for the respiratory passages. It is motor and sensory for the œsophagus, sensory for the stomach, partly motor for the stomach and intestines. The *eleventh or spinal acces-*



*sory*, exclusive of its "bulbar part," supplies the sterno-mastoid and upper fibres of the trapezius. The *twelfth or hypoglossal* supplies the muscles of the tongue and depressors of the hyoid bone.

The **glosso-pharyngeal nerve** may suffer in disease at the base of the brain, thrombosis of the jugular vein, tumours and injuries external to the skull. Isolated paralysis of this nerve has not been observed in man. If it is affected above the petrous ganglion, anæsthesia of the pharynx and back of the tongue, and dysphagia, are observed; if at or below the ganglion, also loss of taste in the posterior third of the tongue, from implication of the associated fibres of the fifth nerve (*see* p. 502).

The **Vagus Nerve** may be implicated in diseases of the cranial bones or of the meninges, and in vertebral aneurysm. In the neck injuries, tumours, or aneurysms; in the thorax mediastinal tumours, or aneurysms, may compress the nerve. Its recurrent laryngeal branch may be compressed on the right side by pleuritic apical adhesions, on the left by aortic aneurysms. The nerve may be paralysed in alcoholic paralysis, poisoning by lead or arsenic, after diphtheria or influenza, in diseases of the medulla, and in disseminated sclerosis.

*Symptoms* vary with the seat of the lesion. Intracranial lesions may affect all the roots, or only the upper or lower. The hypoglossal is often also affected. If the whole nerve is implicated, there is unilateral paralysis of the soft palate, fauces, and larynx, with laryngeal anæsthesia. If both nerves are implicated, the paralysis is bilateral, and there are in addition frequency of the cardiac action, with irregularity; slow, irregular respiration; gastric pain and vomiting, and sometimes loss of the sensations of hunger and thirst. For details of the laryngeal paralysis, *see* p. 337.

The **spinal accessory nerve** is affected by diseases at the base of the brain, it may suffer in disseminated sclerosis or progressive muscular atrophy, and in the neck it may be pressed upon by tumours or abscesses. It is sometimes attacked by "rheumatic" neuritis. Weakness and wasting of the sterno-mastoid and upper part of the trapezius, with R.D., are the result. The chin is rotated towards the opposite shoulder, and the shoulder on the affected side can be only imperfectly raised.

The **hypoglossal** suffers more frequently within than outside of the cranium. Weakness and wasting of one half of the tongue, with R.D., are the chief symptoms. The mucous



membrane of the tongue is wrinkled, and thrown into longitudinal folds. In facial hemiatrophy (*see* Fifth Nerve) one half of the tongue may atrophy, but there is no R.D.; in hemiplegia there is lingual paralysis, but neither atrophy nor R.D.

## SPINAL NERVES

The **Phrenic Nerve**, arising chiefly from the fourth cervical root, with fibres from the third and fifth, supplies the diaphragm, and sends fibres to the pleura, pericardium, inferior cava, and right auricle. It may be affected in fracture of the cervical vertebræ, spinal hæmorrhage, and tumours of the cord; in the neck by wounds, tumours, or aneurysm; in the thorax by tumours, aneurysm, or pleural affections. It may be involved in peripheral neuritis.

**Symptoms.**—When both nerves are affected, there is bilateral paralysis of the diaphragm. The epigastrium sinks in during inspiration, and the movements of the upper part of the thorax are excessive. Dyspnoea occurs on the slightest exertion. Respiratory diseases are rendered more grave by the existence of this condition.

Unilateral paralysis can only be recognised by careful comparison of the movements of the two sides.

*Prognosis* is good in rheumatic and post-diphtheritic paralysis, bad in central lesions.

**Treatment.**—In paralysis due to central lesions, treat the lesion; in peripheral paralysis stimulate the nerve by electricity, placing one pole deeply to the outer side of the clavicular head of the sterno-mastoid, the other over the epigastrium.

Paralysis of the other spinal nerves need not be referred to in detail. A knowledge of their anatomical distribution and function should enable us to reason what to expect.

## NEURITIS

Inflammation of a nerve or of its fibrous sheath.

**Etiology.**—Neuritis may be due to exposure to cold, traumatism, prolonged pressure, or spreading of contiguous inflammation. Predisposing causes are gout, syphilis, *diabetes mellitus*, and also poisoning by lead or arsenic.



**Morbid Anatomy.**—In simple neuritis the sheath of the nerve and the interstitial substance are more affected than the fibres, which suffer only secondarily. The ordinary inflammatory changes are present.

**Symptoms** vary with the special nerve affected. There may be disturbed sensibility, pain in the peripheral distribution of the nerve, or tenderness in its course. Where a motor nerve is involved, twitchings of muscles and rapid atrophy follow, along with paralysis of function. If trophic fibres are involved there may be "glossy skin," oedema of the extremities, herpes, etc.

**Treatment.**—Treat any specific cause by appropriate remedies; locally fomentations or fly blisters. If there be paralysis, galvanism or massage.

## MULTIPLE (PERIPHERAL) NEURITIS

Inflammation affecting simultaneously or in rapid succession nerves in different parts of the body.

**Etiology.**—The disease is due principally to the action of poisons circulating in the blood on the terminations of peripheral nerves. These poisons are of various classes:—

1. Extrinsic: alcohol, arsenic, bisulphide of carbon, lead.
2. Microbic: as in the specific fevers (particularly *diphtheria*), septic states, beri-beri, malaria, influenza, etc.
3. Due to metabolic changes; as in diabetes, gout, syphilis, etc.

General malnutrition is responsible for cachectic and senile cases.

**Morbid Anatomy.**—The changes are most extensive at the peripheral terminations of the nerves, and are usually symmetrical. They are both inflammatory and degenerative, but often only visible under the microscope. The degenerative changes affect the nerve fibres, in which the axis cylinders are atrophied and the medullary sheath broken up. The inflammatory changes are seen in the interstitial tissue, and are much less marked.

Alcoholic paralysis is one of the best examples of multiple neuritis, and will therefore be described as the typical form.

**Etiology.**—It usually occurs amongst *secret* drinkers, and for that reason women are more often attacked. Most cases occur after thirty-five years of age. Club-men and brewers furnish a number of cases.



**Symptoms.**—We must remember that the manifestations of chronic alcoholism differ much in individuals; there are at least four well-marked types.

1. That in which tremor predominates.
2. The anæsthetic type.
3. The paralytic type.
4. The ataxic form.

The onset is insidious, and is accompanied by tingling, like "pins and needles," in the feet and hands, along with numbness of the fingers and toes. Vasomotor spasm may cause "dead fingers," and muscular cramps are frequent. Progressive paralysis compels the patient to take to bed. The extensors are more implicated than the flexors, and consequently the feet and wrists are dropped (the feet generally first). Other muscles are gradually involved, and phonation may be lost or much weakened. Atrophy of the affected muscles rapidly supervenes, and is especially well marked in the extensors below the knee and elbow. Partial or complete reaction of degeneration is present, and there is loss or diminution of the tendon reflexes. The atrophied muscles and the nerve trunks are often exquisitely tender. There are therefore anæsthesia to touch and hyperæsthesia to pain. The mental state frequently suffers: memory, especially of recent events, may be almost lost, ideas of space and time are confused, and delusions are often marked (*Korsakow's syndrome*).

The ataxic form resembles tabetic ataxy in the numbness of the feet, absence of knee-jerk, and to some extent in the gait, which, however, differs from that of true ataxy, in that the patient not only throws out his feet brusquely, *but steps high in order to clear the ground with his dropped toes*. "Alcoholic pseudo-tabes" is, however, rare.

**Treatment.**—The supply of alcohol must be completely cut off, and the patient kept perfectly at rest in bed. To ease the pain, iodide of sodium, antipyrin, or morphia may be given, and hot applications used locally. The paralysis should be treated by strychnine hypodermically, and a mixture of strychnine and capsicum may help to relieve the alcoholic craving. Insomnia may be treated by bromides or trional. The gastric symptoms of alcoholics require careful dieting. General tonic treatment is also necessary. Afterwards good feeding, massage, and electrical stimulation are necessary.



In all cases of multiple neuritis, the special cause must be carefully sought out, and eliminated by appropriate treatment as speedily as possible.

## VII. FUNCTIONAL DISEASES OF THE NERVOUS SYSTEM

### NEURALGIA

This term means paroxysmal pain in the area of distribution of a sensory nerve, usually without discoverable structural change, although in other cases such changes have been noted. It is commonest in adult life, and affects males more than females, except during pregnancy and at the climacteric. It follows upon debilitating diseases, exposure to cold and damp, toxic influences, peripheral irritation (carious teeth, errors of refraction, etc.). The chief symptom is paroxysmal pain, slight or intense, arising spontaneously or from local stimuli, lasting a few seconds or several minutes, and sometimes recurring many times a day. Reflex motor spasms are frequent. Secretory disturbances (lachrymation, etc.) and vasomotor or trophic changes may occur. There are usually "tender points" over the points of exit of nerves through bony foramina, the points where they cross bones or fasciæ, and the corresponding vertebral spines.

**Trigeminal Neuralgia** or "tic douloureux" is very common and very severe. It may affect all the divisions of the fifth nerve, but much more usually one or two, most frequently the ophthalmic. It is almost never bilateral. Exciting causes may be found in the mouth, nose, or eye. The symptoms are sudden onset of almost unbearable pain in the area of the affected branch, from which it may spread to other branches, involuntary spasm of the facial muscles, lachrymation, excessive secretion of nasal mucus, or salivation. During the attack the hand is pressed upon the affected part; in the intervals even a touch may set up the pain, or it may follow movement of the facial muscles, or washing the face. In long-standing cases the hair may fall out or become grey. "Tender points" are to be found at the exit of the branches from their bony foramina.

Other forms of neuralgia — cervico-occipital, intercostal, mammary, sciatic, etc.—require no special description here.

**Treatment.**—Remove any causes of peripheral irritation.



Improve the general health by tonics and massage. In recent cases diaphoretic treatment—hot air, hot baths, etc.—is often useful. Quinine or gelsemium may be pushed till toxic symptoms appear. Relieve pain by one or other of the antipyrin group, salicylates, or iodides. If necessary, give morphia hypodermically, but do not allow the patient to treat himself thereby. Galvanism, the positive pole being over the tender point, is sometimes of use. The most successful drug in trigeminal neuralgia is gelsemium, but it must be pushed. If these measures fail, injection of absolute alcohol into the affected branch of the nerve or into the Gasserian ganglion is often successful. Surgical treatment, in the last resort, comprises nerve stretching and nerve excision, from which relief is usually temporary, and excision of the corresponding ganglion—in the case of the fifth nerve the Gasserian ganglion.

## MIGRAINE

(HEMICRANIA. SICK HEADACHE)

An affection characterised by paroxysmal headache, nausea, and vomiting, and sometimes preceded by disorders of vision. It usually begins about puberty, rarely after thirty, and is somewhat more common in women. Hereditary tendency is very marked, and allied neuroses are often seen in other members of the family. An exciting cause for the first attack is often undiscoverable, but worry, overwork, disturbances of the general health, and especially eye strain and, in the young, adenoids, predispose to repetitions. Migraine is probably due to the action of alimentary or metabolic toxins either directly upon the cortical cells or upon the cerebral circulation.

**Symptoms.**—An attack usually begins with headache, but may be preceded by depression or visual disturbance. The pain, which begins over the forehead, very often in the early morning, is at first slight. It soon becomes severe and boring, is localised just above one eyebrow, and spreads thence till it involves the whole side of the head, and sometimes the neck and arm. The nerves of the scalp are tender on pressure. The attack lasts twelve to twenty-four hours or more; it is accompanied by increasing nausea, and ends in vomiting, which brings relief. In about half the cases there are visual disturbances either before or during the attack. Sometimes merely flashes of light may be noted, in other cases there may be partial or complete homonymous hemianopia, with “spectral”



bright spots appearing in the blind side of the field of vision. The disposition to migraine tends to pass off about the end of the first half of life.

**Treatment.**—In the attack, give a smart saline aperient at once. Absolute rest and quiet in a darkened room, warmth to the feet, evaporating lotions to the head, antipyrin or phenacetin, are the other elements of treatment. In the intervals, improve the general health by attention to the stomach and bowels, correct eye strain, remove adenoids, etc. Tonics, such as iron or strychnine, may be given. A prolonged course of bromides with tincture of gelsemium often does much good; and Gowers recommends in the intervals a course of nitroglycerin.

## EPILEPSY

A chronic paroxysmal affection characterised by sudden attacks of unconsciousness, with or without convulsions, and often by a tendency to mental deterioration. Attacks without convulsions are known as minor fits (*petit mal*), those with convulsions as major fits (*grand mal*).

Jacksonian epilepsy differs so much in etiology, treatment, and pathology, that it will be separately described.

**Etiology.**—The disease frequently begins in early childhood; probably one-fourth of the cases begins before the child is ten years of age, and *three-fourths* before twenty. *When it begins in adults* suspect in the first instance *cerebral syphilis*. Epilepsy in adults may also be due to chronic alcoholism. Uræmia due to chronic nephritis and hystero-epilepsy may cause similar convulsions, but these are not idiopathic epilepsy. Fits due to *local* causes are usually of the Jacksonian type.

*Heredity* is a most important factor, in so far as children of families in which there is a history of insanity, hysteria, alcoholism, neuralgic affections, etc., are prone to this affection.

The exciting causes usually assigned by relatives probably merely determine an attack, but do not cause the disease. Frights and falls or injury to the head are often mentioned. Reflex irritations, as from a tight prepuce, masturbation, worms, dentition, are also blamed, and any undue cause of nervous irritation, whether corporal or emotional, may lead to an attack. In women, attacks are often related to the menstrual period.

**Pathology.**—The structural changes which have been found are purely secondary. The symptoms are due to a functional



disturbance of the cortical centres. Should an epileptic subsequently become hemiplegic from a lesion in the internal capsule, the convulsions do not affect the paralysed limbs, showing that they originate in the cortex, impulses from which are now cut off. Convulsions may be produced by excitation of any part of the cortex, and the nature of the aura may help to localise the seat. Thus a visual aura probably indicates the occipital lobe, an auditory aura the temporo-sphenoidal, and a motor aura the Rolandic area. The actual muscular spasm is due to the sudden violent action or "discharge" of the cortical grey matter.

### Symptoms.—

*Grand Mal*—divided into three stages.—

1. *Aura*, or warning. Vertigo is a very common form. The aura may be referred to the stomach (sinking or nausea), or to the heart (palpitation); it may be visual or auditory; it may be sensory—numbness, "pins and needles," etc., beginning distally and mounting up the limb; motor—twitchings or contractions; or psychical—a dreamy state, or a sense of terror. The aura of idiopathic epilepsy, in contrast to that of the Jacksonian form, is usually very brief.

2. *Fit*.—

(a) The patient drops down suddenly after uttering a piercing shriek (epileptic cry). The cry is sometimes absent.

(b) *Tonic Stage*.—The head and eyes are *turned to the side* on which the convulsions are stronger, the hands clenched, the elbows flexed, the legs rigid and extended, and the muscles of the chest by their contraction interfere with the respirations, so that the preliminary pallor is succeeded by lividity. The pupils are dilated, the conjunctival reflex lost, the eyes fixed, and sensibility is abolished. This stage lasts from thirty to forty seconds, and then passes into the—

(c) *Clonic Stage*.—The convulsions of the face quickly extend to all the muscles of the body; the tongue may be pushed between the teeth and bitten severely; and the foam at the mouth is then coloured with blood. The face assumes a purplish hue, and the eyes seem to protrude from their sockets. Urine, semen, or fæces may escape involuntarily, and the pulse is often much embarrassed by the muscular contractions. This stage lasts about two or three minutes, and then the convulsions cease. The breathing gradually becomes easier, the features are less



cyanosed, and consciousness may be quickly recovered; but more commonly the clonic convulsions are succeeded by—

3. *The Stage of Coma*.—The comatose condition is accompanied by congestion of the face and stertor, but gradually passes into a natural sleep, from which the patient wakes with a feeling of exhaustion.

During the attack the superficial and deep reflexes are abolished, while for a short time after it they are exaggerated. There is commonly a large excretion of urine after the fit. In the attack the temperature is slightly raised. One attack may be rapidly followed by another, with no recovery of consciousness in the interval, and this condition may persist for some hours ("status epilepticus"). In such cases the temperature rises even to hyperpyrexia, and death may result from exhaustion.

*Petit Mal*, or minor epilepsy :—

In this form the patient is suddenly seized with unconsciousness, the eyes become fixed, or the speech momentarily incoherent, but there are *no convulsions*. The attacks vary much in severity, and are sometimes so slight as not to be noticed by others than the patient. This form of epilepsy must be remembered, for though the fits are often trifling, yet they may be succeeded by serious post-epileptic phenomena.

*Post-epileptic conditions* are of two varieties :—

1. *Automatism* : a condition in which the patient performs actions, of which he is then and *afterwards entirely unconscious*. He may begin to undress, or may pocket objects in front of him, or offer violence to a bystander, etc.

During this stage the memory of *past* events remains, and thus people whom the sufferer dislikes may be attacked or killed, apparently deliberately. The condition is most common after *petit mal*.

2. *Post-epileptic mania*, in which homicidal impulses are often manifested.

**Prognosis**.—The frequency of epileptic attacks varies considerably, and the prognosis is more favourable the longer the interval between them, and the older the patient at the onset of the disease. Epileptics may be perfectly healthy between the attacks, and may finally recover completely; but too often some mental weakness shows itself in a permanent form. Imbecility has developed after a few fits in children. The dangers during



the fit must not be forgotten—fatal falls, burns, or other injuries are common.

**Diagnosis.**—The distinctions between epilepsy and hysterical convulsions, uræmia, and malingering are shown in the following table :—

	EPILEPSY.	HYSTERIA.	MALINGERERS.	URÆMIC CONVULSIONS.
<i>Consciousness</i>	Lost.	May be partially lost.	Normal.	Lost in later stages; coma is prolonged and deep.
<i>Pupils</i> . .	Dilated during fit.	Normal.	Normal.	First contraction, followed by dilatation.
<i>Tongue</i> . .	Often bitten.	Normal.	May be bitten to simulate real fit.	Normal.
<i>Restraint</i> .	Necessary to prevent accident.	Necessary to control violence.	Not necessary.	Not necessary.
<i>Onset</i> . . .	Rapid and sudden, patient falls unconscious.	Usually after some mental excitement, patient may fall into a "dazed" condition.	Always under conditions where the fraud may hope to gain sympathy. He falls in a business-like manner, taking care not to sustain painful injuries.	Preceded by alterations in health, urine, etc. Patient is usually in bed before convulsions come on.
<i>Duration</i> .	A few minutes.	Much longer.	Variable.	Prolonged.
<i>Recovery</i> . .	Moderately rapid.	Very variable.	Very rapid after object has been gained.	Slow, if not fatal.

**Treatment.**—Any cause of reflex irritation should be searched for and removed, and the general health should be kept as perfect as possible. Dangerous occupations must be avoided, and bathing forbidden. Stimulants are not allowed. Of drugs, bromides stand first; no permanent ill effects have been definitely traced to a very prolonged use of these drugs. The daily dose for an adult should be from gr. xlv to ziss (grm. 3·0-6·0), and this should be continued for two years after the fits have ceased, and then only gradually withdrawn. If the fits are nocturnal, the whole daily dose may be taken before bedtime. The smallest dose should be given that will control the fits; and if symptoms of bromism (lethargy, drowsiness, cold extremities, feeble pulse) appear, it may be necessary temporarily to stop the drug. The



bromides may be combined with arsenic to prevent the bromide rash. Should they fail, other drugs that may be used are borax gr. v-xv (grm. 0·3-1·0) thrice daily, zinc oxide in pill, and belladonna. Careful attention must be paid to the state of the bowels, menses, etc.

*During the Fit.*—Loosen the collar, corsets, etc., and put something between the teeth to prevent the tongue being bitten; give an inhalation of nitrite of amyl; and protect the patient from injury through his convulsive movements. If there is a well-defined aura, as numbness or twitching in one hand or foot, the attack can often be prevented by prompt ligature of the limb above. If the attacks are nocturnal, the patient should sleep on a hard bed, and use a hard pillow. During the seizure he tends to turn on his face, and may be asphyxiated in the post-epileptic coma. Gowers states that when a person is found dead in bed, and lying on his face, there is a very strong presumption of death after convulsion.

In the status epilepticus a full dose of bromide gr. 40-60 (grm. 2·5-4·0) or chloral gr. 45 (grm. 3·0) should be given by the rectum, or hyoscine hypodermically. Chloroform is useful to check the convulsions until other drugs have time to act.

## JACKSONIAN EPILEPSY

An affection characterised by epileptiform convulsions, often unattended with loss of consciousness, and dependent on a coarse lesion of the cerebral cortex. Although of organic origin, the affection is described here on account of its close relationship to other convulsive disorders.

**Etiology.**—Jacksonian epilepsy is very frequently associated with syphilis, and is therefore most frequently seen amongst adult men; but cortical fits may be due to other tumours, to abscesses, meningitis (especially pachymeningitis hæmorrhagica), and injuries.

**Symptoms.**—The attack begins with twitchings of a muscle or group of muscles in the arm, leg, or face. The spasms may be distinctly localised, and the patient, being conscious, often watches the progress of their march, and thus affords most important information, which often enables the physician to localise the lesion. The convulsions very rarely become general, but may implicate the whole of one side. They are seldom painful. Consciousness may be lost late in the fit, which is



usually followed by a transitory paralysis of the affected muscles or side.

Sensory disturbances precisely analogous to the motor fits may also occur, or may precede them as an aura.

**Diagnosis.**—The slow onset, absence or late appearance of unconsciousness, and the localised type of the convulsions, sufficiently distinguish them from ordinary epilepsy. *The part which first shows rigidity during the convulsion* points toward the motor centre for *that part* as the *seat of the greatest irritation*. The sensory disturbances are of equal localising value.

**Treatment.**—

1. If syphilis is the cause, give iodide of potassium, mercury, or salvarsan (*see Cerebral Syphilis*).

2. If the case is not syphilitic, then trephine, explore, and remove if possible any discoverable source of irritation. Bromides may be used as in epilepsy.

## INFANTILE CONVULSIONS

**Etiology.**—The main factor is the instability, and especially the reflex excitability, of the incompletely developed nervous system of the child, aided by such predisposing causes as—

1. *Rickets*. The chief exciting cause in this condition is reflex irritation, especially from gastro-intestinal irritation, and teething; round-worms, otitis media, and phimosis are also occasional reflex causes.

2. Infectious fevers. Convulsions take the place of the rigors of the adult.

3. Cerebral congestion or asphyxia, as is well seen in severe cases of whooping-cough, or after undue exposure to the sun in a perambulator.

4. Less often cerebral diseases, as at the onset of infantile hemiplegia or diplegia, and meningitis.

5. Congenital syphilis.

6. Hereditary predisposition.

**Symptoms.**—Infantile convulsions are most common before the eighteenth month. They may frequently recur, and may end in the establishment of epilepsy. The symptoms are less severe than those of true epilepsy, though the fit may last longer. The convulsions are bilateral, but often not universal.

**Treatment.**—During the fit:—A few whiffs of chloroform,



a warm but not hot bath, ice to the head, or small doses of chloral by the rectum. After the fit:—A dose of grey powder or calomel, saline laxatives, and bromide in appropriate doses for a week or more. Remove any source of peripheral irritation; attend to the bowels, diet, and fresh air, and treat the general condition.

## TETANY

A condition characterised by tonic muscular spasm chiefly in the distal parts of the limbs, with increased excitability of muscles and nerves.

**Etiology.**—Probably due to a toxin or toxins acting on the peripheral motor neurones. In childhood males, in adult life females, are more often affected. In children, rickets is almost always present; in adults, the disease occurs in connection with pregnancy and lactation, *gastric dilatation* and gastro-enteritis, extirpation of the thyroid, and after specific fevers.

**Pathology.**—The occurrence of tetany after thyroidectomy is due to simultaneous removal of the parathyroids, and it is therefore supposed that these glands neutralise a metabolic toxin, which, in their absence, may produce the symptoms. When tetany occurs in presence of the parathyroids, the toxin must, from other causes, be produced in such quantity that they are unable to neutralise it.

**Symptoms.**—Spasm commences in the hands, then attacks the feet, and may extend to the arms and legs, or even the trunk and face. The thumb is flexed and drawn into the palm, the fingers are flexed at the metacarpo-phalangeal joint, extended at the others, and adducted, so that the hand becomes cone-shaped ("accoucheur's hand"). The wrist and elbow are flexed, and the shoulder adducted. The wrist is drawn to the ulnar side, and the forearm is carried across the chest. In the feet the toes are flexed, especially the great toe; the foot is inverted and the ankle extended. If the trunk is affected there is usually *emprostotonos*, less often *opisthotonos*. The spasm may last from a few minutes to a few days. It passes off slowly, and recurs after irregular but brief intervals. It is usually painful, and passive movement increases the pain. Between the spasms tapping on a muscle or its motor nerve sets up a brisk contraction (*Chvostek's sign*). Pressure on a nerve trunk, or compression of the limb impeding the circulation, will set up a spasm (*Trousseau's sign*). The electrical reactions, galvanic and faradic, are increased in intensity.



In *tetanus* the spasm is more continuous, the muscles of the jaw are early implicated (trismus), and the fingers and hands escape.

**Treatment.**—Remove if possible any evident cause. Improve the general health, and keep the patient at rest. Subcutaneous or intravenous injection of a soluble salt of calcium often arrests the symptoms in tetany after thyroidectomy, and may be tried in other cases, but the treatment must be kept up. Of other drugs, bromides and chloral are the most serviceable. In cases due to gastric dilatation, gastro-enterostomy should be performed.

## HYSTERIA

A form of psychical disorder, characterised by alterations of character and disposition without failure of intellect, and by motor and sensory disturbances without organic disease of the brain or cord, and due to a lessened power of inhibition, and an excessive response to internal or external stimuli.

**Etiology.**—Hysteria is seen principally, but not exclusively, amongst women, and was for long regarded as symptomatic of disorders of the ovaries or uterine functions. This view is still much too prevalent, especially among the laity, but such conditions may lead indirectly to hysteria by the production of a depressed mental state. It may be the result of nerve exhaustion from any cause. Hereditary instability of the neurones is one of the most important factors. Injudicious treatment by relatives is also important. The patients are chiefly young adults. The exciting causes, in those predisposed to the affection, are chiefly psychical—fright, shock, grief, etc. ; but physical causes, such as sexual excess, are also operative, and the disease is not uncommon among prostitutes. It must not be forgotten that hysteria and organic disease of the nervous system may co-exist, and that the symptoms of the former may for a time mask those of the graver affection.

**Symptoms** present an extraordinary variety, and in some instances may closely simulate organic disease. The more common phenomena may be tabulated as follows :—

1. Disturbances of the sensory apparatus :—

- (1) Anæsthesia of localised areas, or complete hemianæsthesia and hemianalgesia. The distribution never corresponds accurately to that of spinal segments, nerve



roots, or nerve trunks, but rather to the *mental concept* of a hand or foot, a limb, or a side of the body. Thus in an anæsthetic hand the upper limit of anæsthesia may encircle the wrist as the top of a glove does, in a leg the anæsthetic area may correspond to that of a stocking, and so on. Complete hemianalgesia is excessively rare in *organic* hemianæsthesia. There may be unilateral loss of one or other special sense, *on the same side*. Anæsthesia may disappear during sleep, and in hypnosis.

- (2) Hyperæsthesia of the skin or special senses.
- (3) Loss of the muscular sense.
- (4) *Globus hystericus*, a sensation as of a "ball of wind" rising from the epigastrium to the throat.
- (5) Neuralgias. "Tender points" over the vertebral spines, the ovarian region, etc. Pressure on these may excite a convulsion ("hysterogenetic zones").

2. Special senses. The ocular disturbances are the most important:—

- (1) Narrowing of the visual field.
- (2) Narrowing of the field for colours, the perception of red disappearing last (dyschromatopsia).
- (3) Amaurosis.
- (4) Conjunctival anæsthesia.

These affections may be unilateral or bilateral, but are most marked on the same side as the anæsthesia or paralysis.

3. Disturbances of the motor apparatus:—

- (1) *Paralytic*. Usually paraplegia of rapid onset, without wasting or electrical change. It may be partial or complete. Knee-jerks exaggerated, *ankle-clonus absent*. Sphincters unaffected. Hemiplegia or monoplegia may occur, also aphonia.
- (2) *Spastic*. Contractures of a limb or segment of a limb, entirely flexor in the arms; in the legs, toes flexed, foot dropped and inverted, knees and hips extended. Contracture of abdominal muscles may cause "phantom tumour," which disappears under chloroform.
- (3) *Convulsive* (hystero-epilepsy). After an aura, tonic stage (opisthotonos), followed by leaping or struggling with *purposive* movements. Screaming, talking, or singing. Consciousness impaired, not lost. Tongue never bitten. Clonic spasms may also occur.
- (4) Tremors or inco-ordinate movements.



## 4. Visceral and vasomotor disturbances :—

- (1) Palpitation or tachycardia.
- (2) Vomiting, belching, etc. Anorexia nervosa.
- (3) Retention of urine or anuria.
- (4) Vicarious menstruation.

## 5. Psychological disturbances :—

Increased suggestibility.

“Fixed ideas,” referable to some previous shock or fright, and colouring the symptoms. These ideas are often latent, and only to be discovered by careful investigation, or in hypnosis.

Hypnoid states,—somnambulism, trance, or catalepsy. Sudden transitions of character, from joyousness to sadness, or from apathy to alertness.

Morbid desire for sympathy, sometimes leading to wilful additions to the real symptoms,—heating the thermometer, factitious skin eruptions, etc.

Nymphomania may occur.

It is a notable feature of hysteria that the intelligence is not impaired. Melancholia or mania may exist as associated psychoses, but are no part of hysteria itself, and a true hysterical delirium is rare. The false statements so often made are due to a wrong interpretation of surroundings altered by disordered bodily sensations, and are not usually deliberately deceitful, although in some instances there is perversion of the moral sense and actual lying.

It will be seen from the above table that almost endless symptoms may occur in various forms of hysteria ; and it may so closely simulate organic disease that the best diagnosticians have been deceived. It is nevertheless possible to make the diagnosis in the majority of instances. Points in favour of hysteria are (1) the abrupt appearance and disappearance of symptoms after trifling causes ; (2) a collocation of symptoms not explicable by an organic cause ; (3) absence of symptoms to be expected in organic disease (optic neuritis, incontinence of urine, ankle-clonus, nystagmus, etc.). In cases of contracture or phantom tumour, an examination under chloroform may be necessary.

**Treatment.**—The successful treatment of hysteria depends upon the recognition of its psychological nature, and upon the discovery and removal of its cause. The search for the emotional or terrifying ideas which, though they have become



subconscious, still dominate the clinical manifestations, is often laborious, and may require the aid of hypnosis or psycho-analysis in expert hands. Waking suggestion is of the utmost value in treatment. That it should be effective, the physician must first gain the patient's full confidence, and a combination of firmness and kindness with discretion is an essential part of his equipment. Change of air and scene, a quiet, well-regulated life, Weir-Mitchell treatment, massage, faradism to paralysed limbs, anti-spasmodic remedies in convulsions, are all of them adjuvants to the reintegration of the psychical condition. It should be remembered that in such cases the worst attendants are sympathising relatives.

## NEURASTHENIA

A condition of nervous exhaustion, leading to altered bodily nutrition.

**Etiology.**—Neurasthenia may be due to a congenital weakness of the neurones, and then appears early. It is more commonly acquired, and is therefore a disease of adult life. Prolonged overwork, especially if associated with worry, or lasting grief, is the chief cause, but sudden shock or injury to the nervous system may also give rise to the disease, which is then spoken of as *traumatic neurasthenia*. Drug habits, especially the morphia habit, may produce the symptoms, or aggravate them if present. They may also follow *influenza* or enteric fever. Alcoholism is an indirect predisposing cause.

**Symptoms.**—The patient is of spare build, or distinctly wasted. His appetite is variable, his bowels irregularly costive or loose, and he suffers from insomnia or broken sleep. He is much depressed and broods continuously on his symptoms, which afford him a melancholy joy. He lacks concentration, and is unfit for mental work. He is restless, and unduly irritated by loud sounds or bright lights. He dwells upon his visceral sensations. Headache, tinnitus aurium, dimness of vision, and spinal irritation are frequent symptoms. Palpitation, præcordial pain, tachycardia, and vasomotor disturbances are common. The muscles are flabby, and may be wasted. Fine tremors in the hands are often present. The knee-jerks may be exaggerated. Dyspepsia is frequent, and flatulence very marked.

**Diagnosis** is mainly dependent on the presence of subjective



symptoms, and the absence of signs of organic disease. The distinction between neurasthenia and hysteria is not always easy to draw, but in the former there are no paralyses, anæsthesiæ, or disturbances of the special senses; the symptoms do not appear and disappear abruptly; the onset is usually gradual, except in cases due to shock or injury; and the majority of patients are males.

**Treatment.**—The causes and definition of the disorder make the line of treatment evident, the main indications being complete change, rest, and tonics. The habit of introspection must be overcome by the substitution of light and varied occupations, or by the development of a hobby. As in hysteria, isolation and Weir-Mitchell treatment may be necessary.

### CHOREA (ST. VITUS'S DANCE)

A disease most common in childhood, characterised by spontaneous irregular movements of the limbs or the whole body, with a tendency to endocarditis and other rheumatic phenomena.

**Etiology.**—It is most frequently seen amongst female children between five and ten years of age, and also during the adolescent period. Girls are attacked three times more frequently than boys. It hardly ever occurs before the age of three. Emotional or excitable children are prone to this affection, and a neuropathic tendency is often traceable in the family history. Clinical records show a more or less constant relationship between chorea, endocarditis, and rheumatism. Although there are cases in which neither a history of rheumatism nor endocarditis can be traced, the *family* history very constantly reveals evidence of rheumatism. In the adult, chorea may occur during pregnancy, but chiefly in the first pregnancies of unmarried girls, where the influence of emotion counts for much.

The chief *exciting cause* is fright, shock, or emotion. Other alleged causes, such as excessive study or overstrain at school, are more apt to aggravate an existing chorea than to cause one. Eye strain is of only secondary importance, and imitation of choreic movements in another, formerly considered important, is of little or no significance. "No cases have arisen in this way at the Great Ormond Street Hospital during thirty years" (Colman and Collier).

**Pathology.**—No constant lesion is found in chorea. Many



observers have noticed punctiform hæmorrhages in the cortex, and also embolic plugging of the small capillaries of the basal ganglia, with small foci of softening; but such conditions are infrequently found. They led to the theory that chorea is caused by detached bits of endocardial vegetations which become arrested in the cortical vessels, and there set up irritative changes. Endocarditis, however, is by no means invariably present, and this view is now generally abandoned. Various micro-organisms, chiefly streptococci and staphylococci, have been found in the cerebral cortex and cerebro-spinal fluid, and Poynton and Payne have isolated a diplococcus which produces chorea in animals, and can be recovered from their cortical arterioles. The modern view is therefore that chorea is of microbic origin, and is to be attributed essentially to "inherent instability in the sensorimotor sphere, together with a toxæmia or a shock sufficient to disarrange the customary association- or contact-areas in the cortex, basal ganglia, and cord." Most probably both the endocarditis and the chorea are the result of the circulation in the blood of a toxin, which in the majority of cases is rheumatic, but in others is of unknown origin.

**Symptoms.**—1. Irregular and purposeless convulsive movements of the limbs, trunk, face, and tongue, rendered worse on attempting to execute voluntary acts and in fine movements, such as picking up pins, conveying a cup to the lips, writing, or touching a particular point. The movements of the trunk, as the child lies in bed, may cause the body to be first twisted to one side, and then suddenly to the other. Sturges sums up the symptoms as "an extremely exaggerated fidgetiness." The movements cease as a rule during sleep, but may be so violent as to prevent it. The condition may affect one side only (hemi-chorea).

2. Combined with the irregular movements there is inco-ordination, manifested in the movements of writing, in unsteady gait, and in irregular movements of respiration, the diaphragm and intercostal muscles failing to act together. Muscular weakness is a later symptom as a rule.

3. Irregular and rapid action of the heart, sometimes amounting to "delirium cordis."

4. Soft systolic murmurs at the apex of the heart, due to endocarditis, or hæmic murmurs at the base. There may be other rheumatic phenomena.

5. Altered mental conditions are sometimes marked, the



patient laughing or crying in turns on being spoken to. Increased irritability is common and, in some cases, chiefly in adults, there may be maniacal excitement (*chorea insaniens*).

An ordinary case lasts from one to three months. Recovery is always gradual, and relapses are common.

**Treatment.**—1. *Rest in bed* is the first and essential point of treatment.

2. Arsenic in increasing doses, cod-liver oil, abundant food, and careful attention to the bowels. Antipyrin or phenacetin in increasing doses is also useful. Salicylates are of value only in rheumatic complications. When the movements are very severe, chloral may be freely given.

As soon as convalescence seems established, allow the patient plenty of fresh air and gymnastic exercises.

## SPASMODIC TORTICOLLIS

(SPASMODIC WRY-NECK)

A painful tonic or clonic contraction of the muscles of the neck, twisting the head to one side and upwards, or causing marked retraction.

**Etiology.**—The patient is of a neurotic type, and often of neurotic extraction. The disease occurs in middle life, and more frequently in women than males. Shock, anxiety, prolonged ill-health, and local injury may precede the symptoms.

The *muscles* affected are (1) the sterno-mastoid, inclining the head forwards and towards the shoulder of the same side; (2) the splenius of the opposite side, drawing the head backwards and turning the face to its own side; (3) later, the upper part of the trapezii, the trachelo-mastoids, and the other deep neck muscles, on both sides. The *nerves* supplying the sterno-mastoid and trapezii are the spinal accessory and the anterior primary branches of the second, third, and fourth cervical nerves; those for the splenii and other deep muscles the posterior primary branches of the first five cervical nerves.

No morbid changes, central or peripheral, have been found; but torticollis is probably due to functional disorder of the cortical centres governing the affected muscles.

**Symptoms.**—The spasm is either tonic or clonic. Sometimes both forms are combined. In the tonic form the head is retracted and the face turned to one side, the shoulder of



that side being also raised. In severe cases the spasm is bilateral. There is then no unilateral rotation, but the head is strongly retracted, and there is wrinkling of the forehead from over-action of the frontalis. In the clonic form there are jerking movements of the muscles, rotating the head from time to time into the position described. The spasm may spread to the facial and brachial muscles. It is aggravated by excitement, and disappears or diminishes in sleep. The affected muscles in time hypertrophy.

**Treatment.**—Drugs are too often of little use. Bromides and chloral may be of temporary benefit; morphia is very apt to induce morphinism. Bastian has produced permanent benefit in some cases by keeping the patient asleep for three or four weeks under the influence of chloral and bromide of sodium, 10 gr. (gram. 0·6) of each every four hours, waking him only for food and medicine, which should be given at the same time. Strict isolation must be observed during this treatment. Surgical measures are often necessary. The operation consists in resection of a portion of the spinal accessory nerves, and excision of the posterior branches of the first four or five cervical nerves.

## PARALYSIS AGITANS

(SHAKING PALSY. PARKINSON'S DISEASE)

An affection characterised by rhythmical tremors of certain muscles, progressive weakness, and later a peculiar gait and attitude.

**Etiology.**—It affects men more than women, and is rarely found in patients under forty years of age. It is commonest after fifty. The influence of heredity is not marked; anxiety, shock, traumatism, are the usual causes assigned, while specific infections appear to have little influence.

**Morbid Anatomy.**—No constant changes are known. The disease is probably due to premature senile changes in the motor cortex, giving rise to irritation of certain motor points, and at the same time diminishing the inhibitive power of the brain.

**Symptoms** are insidious in their onset. Insomnia, unnatural irritability, and general weakness of the limbs may precede the more characteristic symptoms, which are—

1. *Rhythmical contraction* of certain muscles of the fingers and arms; the fingers are flexed with the thumb resting against



the forefinger; the alternating flexion and extension causes a movement like that of rolling pills. The tremors usually spread to the arm and then to the leg of the same side, and afterwards in the same order to the opposite side. The trunk and head are not often affected except by the transmitted tremor of the limbs. The movements at first are checked by voluntary effort or support, but later, as in chorea, they are actually increased on attempting any voluntary restraint. They cease during sleep. The point of distinction between these and other tremors is that in paralysis agitans the tremor is continued during repose, and in whatever position the patient may be, all through the waking hours. It may be confined to one side for several years.

2. *Weakness of the affected muscles.*

3. *Rigidity and contraction*, causing a characteristic attitude and gait, with a mask-like immobility of the face.

4. *Attitude.* The body is bent forwards, the head bowed and held stiffly, and the "vertebra prominens" stands out in bold relief. The arms are flexed at right angles at the elbows, and adducted, while the elbows stand out from the sides. The fingers are flexed at the metacarpo-phalangeal joints, extended at the others. The legs are slightly flexed at the hip and knee, and the thighs are slightly adducted.

5. The *gait* is very characteristic. The first steps are hesitating and slow, but become quick, and the patient appears to trot rather than walk. If he be gently pushed forwards or backwards he continues walking in the direction of the push and may be unable to stop. The forward movement is known as *festination*, the backward as *retropulsion*. Although there is marked weakness of the muscular actions, the muscles do not waste, nor are the electrical reactions altered. Complete paralysis occurs only late in the disease. The tendon reflexes may be increased, but there is no ankle-clonus. Rigidity of the facial muscles leads to an expressionless appearance, the face becoming fixed and mask-like.

6. *Defect of speech.* The speech may be slow and monotonous, or slow at first and afterwards hurried, but articulation is not interfered with.

7. *Subjective symptoms*, such as formication, flushes of heat, and perspiration.

The disease runs a very chronic course, and is fatal usually through some intercurrent complication.

**Treatment** is unsatisfactory. Arsenic and sedatives, attendance to hygiene and diet and avoidance of alcohol, are the main



indications. Gowers finds cannabis indica, combined with arsenic, the best drug treatment. Galvanism and passive movements of the limbs may sometimes be of use.

## RAYNAUD'S DISEASE

A disorder dependent on vasomotor disturbances causing constriction of the peripheral arterioles, and inducing changes of various degrees, known as—(1) Local syncope; (2) Local asphyxia; (3) Local gangrene. The changes are paroxysmal, and usually symmetrical.

**Etiology.**—The disease is most frequent between the ages of twenty and forty, and more frequent in females than in males. Hereditary nervous influence is sometimes apparent, and the patients are usually neurotic. Occasionally some of the specific fevers, malaria, syphilis, functional nervous affections, may be followed by Raynaud's disease. Exposure to cold is the most usual exciting cause.

**Pathology.**—The local condition is due to vascular spasm, with subsequent dilatation of the capillaries and venules, conditions which are supposed to be due to functional disturbance of the vasomotor centres. Hyaline changes have been found in the arteries of the cord, and endarteritis obliterans in the arteries of the nerves to the affected limbs. In these nerves a parenchymatous neuritis has also been found. The symmetrical distribution suggests a toxic cause.

### Symptoms.—

1. *Local Syncope.*—This condition resembles the dead fingers or toes produced by intense cold—one or more fingers, *or the whole hand*, may be affected. Often a severe reaction sets in, and the parts numbed before now become intensely red, hot, and painful. The syncope may last from a few minutes to several hours.

2. *Local Asphyxia.*—This condition is characterised by the fingers, toes, ears, and sometimes patches of skin on the arms and legs becoming intensely congested and livid, the capillary circulation being almost arrested. The congestion gives rise to swelling, stiffness, and pain; the latter is often succeeded by marked anæsthesia. The duration is similar to that of syncope.

These attacks may occur in winter time for many years as “chilblains.”

3. *Local or Symmetrical Gangrene.*—The parts asphyxiated



become cold, insensible, and black in colour—*i.e.* changes identical with necrosis elsewhere. Usually a line of demarcation forms and limits the necrotic condition.

In any given case, these three conditions may occur successively, but more often syncope or asphyxia occurs alone, and either may end in gangrene. Recurrences are frequent, and the paroxysms are markedly periodic.

**Complications.**—*Paroxysmal hæmoglobinuria* is frequently present (about six per cent. of the cases). Other complications are rare, but there may be hæmorrhages, skin eruptions, effusion into serous sacs (joints), delusions, partial coma, or convulsions.

**Treatment.**—Improve any obvious conditions of ill-health. Locally, make the circulation more brisk by gentle friction, warmth, when it does not increase the pain as it may sometimes do, and galvanism, in the form of the galvanic bath. Soothe pain by sedatives, such as belladonna, morphia, conium, etc.



# DISEASES OF THE MUSCLES

## THE MUSCULAR DYSTROPHIES

These affections, however various in form, are all of them essentially due to a congenital defect in the development of the muscular system, which manifests itself sooner or later in atrophy on the one hand, and hypertrophy or pseudo-hypertrophy on the other. In all there is either a marked *hereditary influence*, or a *family predisposition*, the disease affecting several members of the same generation; and all of them begin either in childhood or comparatively early in adult life.

**Morbid Anatomy.**—In all forms the muscular system is primarily affected, and changes in the nervous system are either absent or inconstant. The muscular fibres are some of them atrophied, some hypertrophied, their nuclei are increased, and the perimysium proliferates, leading to increase of fibrous tissue. An extensive deposit of fat-cells takes place within the perimysium, and may entirely replace the muscle fibres, which become fissured and vacuolated. Ultimately certain muscles may completely disappear.

The changes begin in the muscles *of the trunk*, and in the proximal section of the limbs adjacent to them. Not all the muscles undergo atrophy; some increase in size either from the development of fat or connective tissue or from real hypertrophy, and this increase is most common in the upper limbs in the infraspinatus, deltoid, and triceps, in the lower limbs in the glutei, sartorius, and muscles of the calf.

**Pseudo-hypertrophic Paralysis** occurs principally amongst boys, and has a tendency to affect more than one male member of the same family. It may be hereditary, and in such cases the tendency is transmitted through the female line, while only the male children suffer. It commonly begins in childhood.

The muscles most conspicuously *atrophied* are the latissimus dorsi and lower part of the pectoralis major, but the biceps, serratus magnus, and flexors of the hip may also suffer. The *pseudo-hypertrophy* is seen in the *calves*, the glutei, and the infraspinatus and deltoid.



*Symptoms* are very characteristic when the disease is fully developed, but in the earlier periods they are somewhat obscure.

1. Impaired locomotion from muscular weakness; the little fellow lags behind his playfellows.
2. Hypertrophy of muscles with atrophy of others.
3. Certain deformities, through contraction of muscles un-antagonised by the paralysed ones. *Lordosis* is conspicuous.
4. The characteristic gait and movements.

During walking the abdomen is thrown out, with a corresponding *hollowing of the back*; the legs are widely spread out, the whole effect being a *waddling gait*.

The getting up from a recumbent position is very characteristic, especially if there are no objects near, by which the child can aid himself. He first gets on his hands and knees; then extends his knees to the utmost; and lastly, quickly grasping first one knee and then the other, he moves his hands alternately higher and higher up the thighs till the trunk is raised ("climbing up the thighs").

It is remarkable how easily some patients can manage these movements. Later, the muscular weakness is such that they require the aid of near objects to grasp at, and finally failure is complete, the patient being helpless.

There is no reaction of degeneration. The response to both galvanism and faradism becomes progressively less, but as long as any true muscular tissue is left, there is some response to stimulation. The knee jerk is lost when the tonicity of the extensors fails.

The *hereditary* muscular dystrophy of Leyden-Möbius affects the same muscles as pseudo-hypertrophic paralysis, but all the muscles are atrophic.

**The Juvenile Type** of muscular dystrophy described by Erb arises about puberty or in early adult life, and may occur in either sex. The muscles of the shoulder girdle and upper arm are first and principally affected, and the lower limbs suffer later. The latissimus dorsi, serratus, and pectoralis major suffer, and also the trapezius, rhomboids, biceps, triceps, and supinator longus. The deltoid and the hand escape.

In the **Infantile or Facio-Scapulo-Humeral Type**, which may begin either in infancy or at puberty, the facial muscles are primarily involved, and the lips may be thickened from pseudo-hypertrophy. The orbicularis palpebrarum also suffers, and the



disease spreads to the shoulder girdle and arms. The tongue and ocular muscles are not affected.

**Prognosis** is least favourable in the pseudo-hypertrophic and infantile forms, in which few patients survive to the age of twenty, and the majority die of intercurrent pulmonary disease, due to weakness of the respiratory muscles. In the other forms life may be prolonged for many years.

**Diagnosis.**—These affections are to be distinguished from spinal muscular atrophy by the following points. They begin in early life, and show a hereditary or family tendency; they involve most markedly the muscles of the trunk and proximal sections of the limbs, while the distal sections, and particularly *the hands*, are little affected; *fibrillary tremors are absent*; electrical reactions are only quantitatively diminished; and the atrophy is often combined with hypertrophy or pseudo-hypertrophy.

**Treatment.**—Most benefit is to be expected from a thorough system of well-planned gymnastic exercises, massage, careful hygiene, and galvanism. The diet should be highly nourishing. Cod-liver oil or malt may be given.

Tonics such as strychnine and phosphorus may be employed, and of these strychnine hypodermically is most frequently used. Arsenic also is often prescribed, and may be given hypodermically or by the mouth. The thinness of the chest walls renders the patient very prone to chest affections, which are often fatal, and should be particularly guarded against.

## THOMSEN'S DISEASE

### (MYOTONIA CONGENITA)

A rare condition, characterised by a tendency of the muscles to tonic spasm during attempts at voluntary movements.

The disease is probably always associated with some congenital defect, but auto-intoxication has been suggested as the cause. Heredity is an important factor, and more than one member of a family may be affected.

**Morbid Anatomy.**—The muscular fibres are hypertrophied, and may be more than twice the ordinary size. The nuclei of the sarcolemma are increased, and there is a slight increase of interstitial tissue. Among the hypertrophied fibres atrophic fibres have been found.



**Symptoms.**—1. After *rest*, the patient experiences tension and stiffness of the muscles on attempting to rise, etc.

2. Difficulty in *relaxing* the muscles.

3. In severe cases the spasm may be at first so great as to cause the patient to remain locked in the position in which he is ; but it yields gradually to repeated attempts at movement.

4. The muscles of the tongue, face, and eyes may be similarly affected. The acts of micturition, defæcation, and respiration are not usually involved.

5. The muscles are usually enlarged, and respond excessively to stimuli, swelling locally after a slight blow.

6. The electrical reactions are markedly disturbed. There is increased irritability, and the contraction following faradic, and especially galvanic, stimuli is unduly prolonged. A.C.C. is as easily elicited as K.C.C. A continuous galvanic current sets up a series of wave-like contractions, passing from cathode to anode (*myotonic reaction*).

Note that continued movements and warmth decrease the spasm ; mental worry and cold usually aggravate it.

**Treatment.**—Massage and regular gymnastic exercises, with warm bathing, may alleviate the spasm. The disease is incurable, but not fatal.



# INDEX

- Abdominal aorta, aneurysm of, 294
- Abscess of brain, 471
- — differential diagnosis, 485
- of liver, 217
- — dysenteric, 77
- — pyæmic and tropical, 218
- of lungs, 368
- subphrenic, 240
- Acetone, test for, 387
- Achilles-tendon jerk, 424
- Achylia gastrica, 177
- Acidity, gastric, estimation of, 159
- Acidosis, 137
- Acromegaly, 323
- Actinomycosis, 87
- etiology, 87
- morbid anatomy, 88
- symptoms and treatment, 89
- Addison's disease, 319
- — symptoms, 320
- — treatment, 321
- Adherent pericardium, 253
- Ægophony, 330
- Æstivo-autumnal fever, 84
- Agglutinins, 14, 15
- Agraphia, 487
- "Ague-cake," 83
- Ague, symptoms, 83
- Air-hunger in diabetes, 136
- Albumin, tests for, 384
- Albuminuria, 389
- Albuminuric retinitis, 398
- Albumoses, tests for, 385
- Alcohol in disease, 11
- Alcoholic cirrhosis of liver, 220
- pseudo-tabes, 512
- Alcoholism, chronic, 141
- — treatment, 142
- Alexia, 487
- Alexine, 14
- Alimentary tract, diseases of, 151
- Amaurosis, 495, 523
- uræmic, 393
- Amblyopia, tobacco, 496
- Amboceptors, 14
- Amnesia, verbal, 488
- Amœba dysentericæ, 76
- Amphoric breathing, 329
- resonance, 328
- Amyloid disease of the kidney, 404
- — of liver, 228
- Amyotrophic lateral sclerosis, 447
- Anæmia, 297
- aplastic, 301
- pernicious, 300
- Anæmia, pernicious, morbid anatomy, 301
- — symptoms, 302
- — treatment, 302
- secondary, 298
- splenic, 322
- Anæsthesia, dissociated, 435
- hysterical, 522
- Anaphylaxis, 17
- Anarthria, 455, 478
- Aneurysm, 287
- etiology and morbid anatomy, 288
- of abdominal aorta, 294
- of sinuses of Valsalva, 291
- of thoracic aorta, 289
- Angina abdominis, 280
- pectoris, 279
- — symptoms, 280
- — diagnosis and treatment, 281
- — vasomotoria, 280
- Angina sine dolore, 280
- Ankle-clonus, 424
- Ankle-drop, 145, 512
- Ankylostoma duodenale, 205
- Anorexia nervosa, 176
- Anosmia, 493
- Anthraco-sis, 359
- Anthrax, 89
- symptoms, 90
- treatment, 91
- Anthrax-cœdema, 90
- Antibodies, 13
- Antigens, 13
- Antisera, 13
- Antitoxins, 13
- Antityphoid serum, 26
- Aortic aneurysm, 289
- — physical signs, 291
- — symptoms, 290
- — treatment, 293
- — varieties, 292
- regurgitation, 267, 270, 271
- stenosis, murmur of, 267
- — pulse of, 270
- Aphasia, 485
- types of, 488
- Aphtha epizootica, 93
- Aplastic anæmia, 301
- Apoplectiform attacks, 393, 489, 492
- Apoplexy, 474
- splenic, 89
- Appendicitis, 183
- diagnosis, 185
- morbid anatomy, 184
- perforation in, 184



- Appendicitis, symptoms, 184  
 — treatment, 185  
 Appendix, vermiform, anatomy, 183  
 Argyll-Robertson pupil, 441, 492, 497  
 Arrhythmia, cardiac, 281  
 Arsenical poisoning, 146  
 Arteries, diseases of, 286  
 Arteriosclerosis, 286  
 — etiology and morbid anatomy, 286  
 — symptoms and treatment, 287  
 Arteriosclerotic kidney, 402  
 Arthritis, rheumatoid, 129  
 — — acute, 132  
 — — pathology, 130  
 — — symptoms, 131  
 — — treatment, 132  
 — gonorrhœal, 98  
 — rheumatic, 65  
 — scarlatinal, 38  
 Arthropathy, tabetic, 442  
 Artificial pneumothorax, 368  
 Ascaris lumbricoides, 204  
 Ascites, 223, 226, 236  
 — diagnosis, 238  
 — etiology, 236  
 — signs, 237  
 — treatment, 238  
 Asphyxia, local, 531  
 Asthma, 343  
 — symptoms, 344  
 — treatment, 345  
 Ataxia, Friedreich's, 450  
 — hereditary, 450  
 — locomotor, 439  
 Ataxic gait, 427, 441  
 — paraplegia, 449  
 Atelectasis, congenital, 347  
 Atheroma, 287  
 Athetosis, 479  
 Atrophy of liver, acute yellow, 212, 216  
 Atrophy, optic, 496  
 — progressive muscular, 445  
 Auditory nerve, 506  
 Aura, epileptic, 516  
 Auricular fibrillation, 262, 283  
 — flutter, 283  
 Auscultation of heart, 246  
 — of lungs, 328  
 Automatism, post-epileptic, 517  
 Axons, 413  
  
 Babinski's sign, 444, 475  
 Bacillus anthracis, 89  
 — botulinus, 147  
 — cholerae, 70  
 — diphtheriae, 54  
 — dysenteriae, 73  
 — enteritidis, 147  
 — icteroides, 78  
 Bacillus influenzae, 48  
 — leprae, 115  
 — mallei, 92  
 — of Oppler-Boas, 170  
 — pestis, 68  
 — tetani, 96  
 — tuberculosis, 112  
 — typhosus, 20  
 — of whooping-cough, 44  
 Banti's disease, 322  
 Bell's paralysis, 505  
 Bence-Jones albumosuria, 385  
 Beri-beri, 126  
 — morbid anatomy and symptoms, 127  
 — treatment, 127  
 Bile, formation of, 212  
 — secretion of, 213  
 Bile-ducts, diseases of, 230  
 — tumours, 231  
 Bile salts, tests for, 387  
 Bilharzia hæmatobia, 208  
 Biliary calculi, 231  
 — cirrhosis of liver, 223  
 — colic, 232  
 — — treatment, 233  
 — fistula, 233  
 Bilious remittent fever, 84  
 Bilirubin-calcium, in gall-stones, 232  
 Biuret reaction, 160, 385  
 "Black vomit," 79  
 Blackwater fever, 85  
 — — treatment, 87  
 Blindness, 495, 496  
 Blood, colour index, 296  
 — diseases of, 293  
 — examination of, 293  
 — in chlorosis, 299  
 — in diabetes, 133  
 — in leukæmia, 303  
 — in pernicious anæmia, 301  
 — in secondary anæmia, 298  
 — in urine, tests for, 391  
 — crises, 302  
 — occult, test for, 166  
 Bone-marrow, in leukæmia, 304  
 — in pernicious anæmia, 301  
 Bothriocephalus latus, 198  
 $\beta$ -oxybutyric acid, 134, 136  
 Bradycardia, 285  
 — paroxysmal, 278  
 Brain, abscess, 471  
 — anatomy of, 455  
 — basal ganglia of, 456  
 — blood supply, 457  
 — cysts of, 474, 476  
 — diseases of, 455  
 — embolism, 477  
 — functions of, 459  
 — lesions at different levels, 462  
 — hæmorrhage into, 472



- Brain, hæmorrhage into, cortical, 475.  
 — irritation of, 462  
 — softening, 476  
 — syphilis, 481  
 — thrombosis, 476  
 — tumours, 483  
 Break-bone fever, 50  
 "Breast-pang," 279  
 Breath-sounds, types of, 328, 329  
 Bright's disease, acute, 394  
 — — chronic, 397  
 — — differential diagnosis, 400  
 "Brill's disease," 26  
 Broca's convolution, 460, 486  
 Bromism, 518  
 Bronchi, diseases of, 338  
 Bronchial breathing, 329  
 Bronchiectasis, 341, 342  
 — treatment, 343  
 Bronchiolitis exsudativa, 344  
 Bronchitis, acute, 338  
 — — diagnosis, 374  
 — — symptoms and treatment, 339  
 — capillary, 339  
 — chronic, 340  
 — — symptoms and treatment, 340  
 — — varieties, 341  
 — putrid, 341  
 Bronchophony, 330  
 Bronchopneumonia, 356  
 — diagnosis, 374  
 — morbid anatomy and symptoms, 357  
 — treatment, 358  
 Bronchorrhœa, 341  
 Broncho-vesicular breathing, 330  
 Brood capsules, of hydatids, 200  
 Bubonic plague, 67  
 Bulbar paralysis, 452  
 — — acute, 455  
 — — symptoms, 453  
 — — treatment, 455
- Cachexia, malarial, 83, 86  
 — saturnine, 145  
 — strumipriva, 315  
 Calculi, biliary, 231  
 — renal, 408  
 Calmette's reaction, 365  
 Cancer of intestine, 189  
 — — treatment, 190  
 — of liver, 225  
 — of lungs, 369  
 — of œsophagus, 157  
 — of pancreas, 235  
 — of peritoneum, 243  
 — of rectum, 189  
 — of stomach, 168  
 Cancrum oris, 153  
 Capillary bronchitis, 329  
 Capsule, external, 420  
 Capsule, internal, 422  
 Carbohydrates, metabolism of, 210  
 Casts, urinary, 388  
 Catarrh of intestine, 178  
 — — in children, 180  
 — of stomach, acute, 160  
 — — chronic, 161  
 Catarrhal jaundice, 230  
 Caudate nucleus, 430  
 Cerebral diplegia, 481  
 — embolism, 476  
 — hæmorrhage, 472  
 — — cortical, 475  
 — — symptoms, 474  
 — — treatment, 475  
 — irritation, 462  
 — localisation, 459  
 — palsies, infantile, 480  
 — paraplegia, 481  
 — sinuses, thrombosis, 469  
 — syphilis, 481  
 — — treatment, 482  
 — thrombosis, 476  
 — tumours, 483  
 — — diagnosis and localisation, 484, 485  
 — — symptoms, 483  
 — — treatment, 484  
 Cerebro-spinal fever, epidemic, 50  
 — — — complications, 52  
 — — — Kernig's sign in, 52  
 — — — morbid anatomy, 51  
 — — — lumbar puncture in, 52  
 — — — symptoms, 51  
 — — — treatment, 52  
 Cestoda, 194  
 — life-history, 195  
 "Chalk-stones," 120, 121  
 Chancre, hard, 101  
 Charbon, 89  
 Charcot's disease of the joints, 442  
 Charcot-Leyden crystals, 314, 344  
 Chest, examination of, 326  
 Cheyne-Stokes breathing, 278  
 — — in uræmia, 393  
 Chiasma, optic, 495  
 Chicken-pox, 35  
 Child-crowing, 335  
 Chlorosis, 297, 298  
 — etiology and symptoms, 299  
 — treatment, 300  
 Choked disc, 484  
 Cholangitis, 230  
 Cholecystitis, 230  
 Cholera, etiology, 70  
 — morbid anatomy, 70  
 — symptoms, 71  
 — treatment, 72  
 — infantum, 181  
 — nostras, 181



- Chorea, 526  
 — etiology and pathology, 526  
 — symptoms, 527  
 — treatment, 528  
 — insaniens, 528  
 — post-hemiplegic, 479  
 Chvostek's sign, 521  
 Chyluria, filarial, 207  
 Circle of Willis, 457  
 Circulatory system, diseases of, 244  
 Cirrhosis of kidney, 402  
 — of liver, 220  
 — — biliary, 223  
 — of lung, 358  
 — of stomach, 162  
 Claudication, intermittent, 279  
 Clonus, ankle, 424  
 — knee, 424  
 Cocainism, 143  
 Cochlear nerve, 506  
 Colic, biliary, 232  
 — lead, 145  
 — renal, 409  
 Colitis, ulcerative, 182  
 — mucous, 182  
 Collapse of lung, 347  
 Colles' law, 100  
 Colour index of blood, 296  
 Column of Burdach, 416  
 — of Clarke, 415  
 — of Goll, 416  
 Coma, alcoholic, 142  
 — diabetic, 136  
 — — treatment, 138  
 — epileptic, 517  
 Comma bacillus, 70  
 Compensation in heart disease, 260  
 — failure of, 261  
 — — treatment, 274  
 Complement, 14  
 — deviation of, 105  
 Condylomata, 101, 102  
 Congestion of lungs, 349  
 — of liver, passive, 229  
 — passive, in heart disease, 268  
 Conjugate deviation, 475, 481  
 Constipation, 191  
 — treatment, 192  
 Convulsions, epileptic, 516  
 — hysterical, 523  
 — infantile, 520  
 — — treatment, 520  
 Co-ordination, course of fibres serving, 420  
 Copula, 14  
 Coronary arteries, diseases of, 276  
 Cor villosum, 251  
 Cough, varieties of, 331  
 Cracked-pot sound, 328  
 Craniotabes, 124  
 Crepitus, pneumonic, 330, 352  
 Cretinism, 314  
 — symptoms and treatment, 315  
 Crises of locomotor ataxia, 442  
 Crossed paralysis, 462, 479, 505  
 Croup, 59  
 — catarrhal, 333  
 — false, 333  
 — spasmodic, 333  
 — diphtheritic, 56  
 Curschmann's spirals, 344  
 Cysticerci, 195  
 Cysticercus bovis, 197  
 — cellulosæ, 196  
 Cysts, hydatid, 200  
 — — of liver, 227  
 Cytases, 14  
 Cytoryctes variolæ, 30  
 Dandy fever, 50  
 Decussation of the fillet, 419  
 — of the pyramids, 417  
 — sensory, 419  
 Degeneration, reaction of, R.D., 424  
 — secondary, 424  
 — — morbid changes of, 425  
 Delirium, forms of, 9  
 — cordis, in chorea, 527  
 — tremens, 142  
 Dementia, paralytic, 490  
 Dendrons, 413  
 Dengue, 49  
 — symptoms, 50  
 Deviation of complement, 105  
 — conjugate, 475, 501  
 — secondary, 499  
 Diabetes insipidus, 139  
 — mellitus, 132  
 — — blood in, 133  
 — — complications, 136  
 — — pancreas in, 133  
 — — pathology, 134  
 — — symptoms, 135  
 — — treatment, 137  
 Diabetic coma, 136  
 — puncture, 135  
 — tabes, 137  
 Diacetic acid, test for, 387  
 Diaphragm, paralysis of, 510  
 Diarrhœa, 190  
 — lienteric, 179  
 Diazo-reaction, 24  
 Dibothrium latum, 198  
 Dietl's crises, 412  
 Digestion, gastric, 157  
 Dilatation of heart, 265  
 — of left ventricle, 265  
 — of right ventricle, 266  
 — of stomach, 173  
 Dioxydiamidoarsenobenzol, 106  
 Diphtheria, 54  
 — bacteriology, 54



- Diphtheria, complications, 57  
 — diagnosis, 57  
 — etiology, 54  
 — morbid anatomy, 55  
 — serum disease in, 59  
 — symptoms, 56  
 — treatment, 58  
 — varieties, 57  
 Diplegia, cerebral, 481  
 Diplococcus intracellularis meningitidis, 51  
 — pneumoniae, 351  
 Diplopia, 499  
 Disorders of cardiac rhythm, 281  
 — of nutrition, 118  
 Disseminated sclerosis, 488  
 — — diagnosis, 490  
 — — symptoms, 489  
 Distomum hæmatobium, 208  
 Doremus' ureameter, 382  
 Dracunculus medinensis, 207  
 Dropsy, cardiac, 263  
 — causes of, 236  
 — of gall-bladder, 233  
 — renal, 395, 398  
 — — treatment, 396  
 Ductless glands, diseases of, 313  
 — — in diabetes, 135  
 Duodenal ulcer, 166  
 Dura mater, hæmatoma of, 464  
 Dysarthria, 453, 478  
 Dyschromatopsia, hysterical, 523  
 Dysentery, 73  
 — amœbic, 76  
 — — morbid anatomy and symptoms, 76  
 — — treatment, 77  
 — bacillary, 73  
 — — complications, 75  
 — — etiology, 73  
 — — morbid anatomy, 74  
 — — symptoms, 74  
 — — treatment, 75  
 Dyspepsia, 175  
 — treatment, 177  
 Dysphonia clericorum, 334  
 Dystrophy, muscular, 533  
  
 Eberth's bacillus, 20  
 Ehrlich's side-chain theory, 15  
 Eighth nerve, 506  
 Elephantiasis, tropical, 207  
 Emboli in endocarditis, 257  
 Embolism, cerebral, 476  
 Emetine in amœbic dysentery, 77  
 Emphysema, 341, 345  
 — atrophic and compensatory, 346  
 — hypertrophic, 346  
 — symptoms, 347  
 Empyema, 373  
 — treatment, 376  
 Empyema of gall-bladder, 233  
 Encephalitis, diffuse, 471  
 — hæmorrhagic, 470  
 — suppurative, 470  
 Encephalopathy, saturnine, 146  
 Endarteritis, obliterative, 481  
 Endocarditis, acute, 254  
 — — etiology, 254  
 — — morbid anatomy, 255  
 — — symptoms, 256  
 — — treatment, 258  
 — chronic, 258  
 — congenital, 256  
 — malignant, 255  
 — — varieties, 257  
 — ulcerative, 255  
 Entamœba histolytica, 76  
 Enteric fever, 19  
 — — bacteriology, 20  
 — — characters of ulcers, 23  
 — — complications, 22  
 — — diagnosis, 24  
 — — etiology, 20  
 — — morbid anatomy, 21  
 — — perforation in, 23  
 — — rash, 22  
 — — specific treatment, 26  
 — — symptoms, 21  
 — — treatment, 24  
 — — varieties, 22  
 — — Widal's reaction in, 23  
 Enteritis, 178  
 — symptoms, 179  
 — treatment, 180  
 — catarrhal, 178  
 — croupous, 181  
 — phlegmonous, 181  
 — ulcerative, 181  
 Epilepsy, 515  
 — diagnosis, 518  
 — Jacksonian, 519  
 — minor, 517  
 — pathology, 515  
 — symptoms, 516  
 — treatment, 518  
 Epileptiform attacks, 278, 489, 492  
 Epiploexy, 224  
 Ergotism, 148  
 Erysipelas, 60  
 — symptoms, 60  
 — treatment, 61  
 Esbach's albuminimeter, 384  
 Exophthalmic goitre, 317  
 External capsule, 422  
 Extra-systole, 282  
  
 Facial hemiatrophy, 503  
 — neuralgia, 513  
 — paralysis, 504  
 — — hemiplegic, 478  
 — — treatment, 506



- Facial spasm, 506  
 Facies Hippocratica, 240  
 Fæcal ulcers, 182  
 Farcy, 92  
 Fatty heart, 276  
   — liver, 229  
 Fehleisen, streptococcus of, 60  
 Fermentation test for sugar, 386  
 Festination, 530  
 Fever, 1  
   — course of, 8  
   — delirium in, 9  
   — general treatment of, 10  
   — morbid anatomy of, 4  
   — physiology of, 4  
   — types of, 5  
   — typhoid state in, 8  
   — use of alcohol in, 11  
   — æstivo-autumnal, 84  
   — bilious remittent, 84  
   — blackwater, 85  
   — enteric, 19. *See* Enteric Fever  
   — glandular, 47  
   — hæmoglobinuric, 85  
   — malarial, 81  
   — Malta, 80  
   — Mediterranean, 80  
   — paratyphoid, 24  
   — relapsing, 28  
   — rheumatic, 64  
   — scarlet, 36  
   — splenic, 89  
   — thermic, 149  
   — typhoid, 19. *See* Enteric Fever  
   — typhus, 26  
   — yellow, 77  
 Fibrillary twitchings, 446  
 Fibrillation, auricular, 262, 283  
 Fibroid degeneration of heart, 277  
   — phthisis, 364  
 Fibrosis, arterio-capillary, 286  
 Fifth nerve, 501  
 Filaria Bancrofti, 206  
   — medinensis, 207  
   — nocturna, 207  
   — sanguinis hominis, 206  
 Filariasis, 207  
 Fillet, decussation of the, 419  
 Fistula, biliary, 233  
 Fits, apoplectic, 474  
   — apoplectiform, 393, 489, 492  
   — epileptic, 516  
   — epileptiform, 278, 489, 492  
   — uræmic, 392  
 Fixed ideas, 524  
 Flaccid paralysis, 427  
 Flatworms, 208  
 Flechsig's tract, 416  
 Fleshworms, 202  
 Floating kidney, 412  
 Flukes, 208  
 Food poisoning, 147  
 Foot and mouth disease, 93  
 Fourth nerve, 498  
 Framboesia, 108  
 Fremitus, hydatid, 227  
   — vocal, 327  
 Friction, pericardial, 248, 252  
   — pleural, 330, 371  
 Friedländer's pneumobacillus, 351  
 Friedreich's ataxia, 450  
   — sign, 253  
 Gait, ataxic, 427  
   — of disseminated sclerosis, 427  
   — in paralysis agitans, 531  
   — spastic, 427  
 Gall-bladder, diseases, 230  
   — dropsy and empyema, 233  
   — inflammation, 230  
   — tumours, 231  
 Gall-stones, 231  
   — characters and symptoms, 232  
   — in intestinal obstruction, 186  
 Gangrene, of lungs, 368  
   — symmetrical, 531  
 Garrod's test for uric acid, 122, 382  
 Gastralgia, 176  
 Gastric crises, 442  
   — ulcer, 162  
   — — characters, 163  
   — — etiology, 163  
   — — pathology, 164  
   — — perforation in, 167  
   — — subphrenic abscess in, 167  
   — — symptoms, 165  
   — — treatment, 167  
 Gastritis, acute, 160  
   — — symptoms and treatment, 161  
   — chronic, 161  
   — membranous, 161  
   — phlegmonous, 161  
   — toxic, 161  
 Gastrodynia, 176  
 Gastroptosis, 174  
 Gastrorrhagia, 171  
 Gastrostaxis, 172  
 Gastosuccorrhœa, 176  
 Gastroxynsis, 176  
 Gaucher's disease, 322  
 General paralysis, 490  
   — — symptoms, 491  
   — — treatment, 492  
 Gerhardt's sign, 363  
 German measles, 42  
 Girdle pains of locomotor ataxia, 440  
 Glanders, 92  
   — diagnosis, 93  
   — etiology and morbid anatomy, 92  
   — symptoms, 92  
 Glandular fever, 47  
 Globus hystericus, 523



- Glomerulo-nephritis, 395  
 Glosso-labio-laryngeal paralysis, 452  
 Glosso-pharyngeal nerve, 508  
 Glottis, œdema of, 333  
 Glycogenic function of liver, 134, 210  
 Goitre, 316  
   — symptoms, 316  
   — exophthalmic, 317  
   — — symptoms, 318  
   — — treatment, 319  
 Gonorrhœal arthritis, 98  
   — infection, 98  
   — — symptoms and treatment, 99  
 Gout, 118  
   — chronic, 121  
   — etiology, 118  
   — irregular, 121  
   — morbid anatomy, 120  
   — pathology, 118  
   — symptoms, 120  
   — suppressed, 121  
   — treatment, 122  
 Gowers' table of spinal localisation, 432  
   — tract, 416  
 Grain poisoning, 148  
 Grand mal, 516  
 Granular kidney, 402  
 Grocco's sign, 372  
 Gummata, 101, 102  
   — cortical, 482  
   — structure of, 101, 102  
  
*Hæmamoeba malarie*, 81  
*Hæmatemesis*, 171  
   — diagnosis, 172  
   — treatment, 173  
*Hæmatoma* of dura mater, 464  
*Hæmatorrhachis*, 431  
*Hæmaturia*, 390  
   — treatment, 391  
*Hæmoglobinuria*, 391  
   — paroxysmal, 311  
*Hæmoglobinuric fever*, 85  
*Hæmolysis*, 104  
*Hæmomenas*, 82  
*Hæmophilia*, 309  
   — treatment, 310  
*Hæmoptysis*, 350  
   — in phthisis, 362  
*Hæmorrhage*, cerebral, 472  
   — — morbid anatomy, 473  
   — — symptoms, 474  
   — meningeal, 431, 464, 473  
   — pancreatic, 235  
 Haffkine's anti-cholera vaccine, 73  
 Hanot's disease, 223  
 Haptophore group, 15  
 Harrison's groove, 125  
 Heart, dilatation, 265  
   — diseases of, 244  
 Heart disease, compensation in, 260  
   — disorders of rhythm, 281  
   — examination of, 245  
   — fatty, 276  
   — fibroid degeneration, 277  
   — hypertrophy, 263  
   — syphilis, 277  
   — valvular diseases, 259, 266  
   — — — prognosis, 271  
   — — — symptoms, 268  
   — — — treatment, 272  
 Heart-block, 277  
 Heat-exhaustion, 149  
 Heller's test for albumin, 384  
 Hemeralopia, 495  
 Hemianæsthesia, 477, 484  
   — hysterical, 523  
 Hemianopia, 496  
 Hemiatrophy, facial, 503  
 Hemisrania, 514  
 Hemiplegia, 427, 478  
   — infantile, 480  
   — treatment, 480  
 Hepatic abscess in dysentery, 76  
   — intermittent fever, 233  
 Hepatisation of lung, 352  
 Hereditary ataxia, 450  
 Hobnailed liver, 221  
 Hodgkin's disease, 305  
   — — symptoms, 306  
   — — treatment, 307  
 "Hutchinson" teeth in syphilis, 104  
 Hydatid fremitus, 227  
 Hydatids, 200, 227  
   — of liver, 227  
 Hydræmia, 297  
 Hydrarthrosis, gonorrhœal, 99  
 Hydrocephalus, 468  
   — acute, 466  
   — treatment, 469  
 Hydrochloric acid, tests for free, 159  
 Hydronephrosis, 407  
   — symptoms and treatment, 408  
 Hydrophobia, 94  
   — symptoms, 95  
   — diagnosis and treatment, 95  
 Hydropneumothorax, 377  
 Hydrothorax, 378  
 Hyperacousis, 505  
 Hyperchlorhydria, 165, 176  
 Hypernephromata, 411  
 Hyperosmia, 493  
 Hyperpyrexia in acute rheumatism, 65  
 Hypertonus, muscular, 425  
 Hypertrophy of heart, 263  
 Hypochlorhydria, 162, 176  
 Hypoglossal nerve, 509  
 Hypostatic congestion, 349  
 Hysteria, 522  
   — diagnosis, 518, 524  
   — psychical disturbances in, 524



- Hysteria, symptoms, 522  
 — treatment, 524  
 Hystero-epilepsy, 523  
 Hysterogenetic zones, 523
- Icterus, 213  
 — neonatorum, 216  
 Immune body, 14  
 Immunity, active, 12  
 — high tide of, 17  
 — passive, 13  
 — theories of, 15  
 "Inclusion bodies" in scarlet fever, 36  
 Inco-ordination, 426  
 — in chorea, 527  
 — in locomotor ataxia, 441  
 Incubation period of infectious diseases, 19  
 Infantile paralysis, 436  
 — — treatment, 438  
 Infarction of lungs, 350  
 Infectious diseases, 19  
 Influenza, 47  
 — bacteriology and morbid anatomy, 48  
 — complications, 49  
 — symptoms, 48  
 — treatment, 49  
 Insular sclerosis, 488  
 Intention tremor, 489  
 Intermission of pulse, 283  
 Intermittent claudication, 279  
 — fever, hepatic, 233  
 Internal capsule, 417, 420  
 Interstitial pneumonia, 358  
 Interstitial nephritis, 402  
 Intestinal obstruction, 186  
 — — diagnosis, 188  
 — — symptoms, 187  
 — — treatment, 188  
 — — chronic, 188  
 "Intestinal stasis," 192  
 Intestines, catarrh of, 178  
 — — acute, in children, 180  
 — diseases of, 178  
 — syphilis, 181  
 — tuberculosis, 182  
 — tumours, 189  
 — ulceration, 181  
 Intoxications, 141  
 Intussusception, 186, 187  
 Iridoplegia, 497  
 — reflex, 441, 497  
 Irregularity, cardiac, 281
- Jacksonian epilepsy, 519  
 Jaundice, 213  
 — catarrhal, 230  
 — etiology, 214  
 — fatal, 216  
 Jaundice, forms of, 214  
 — obstructive, symptoms, 215  
 — — treatment, 216
- (Kala-azar), Leishmaniasis, 110  
 Kernig's sign, 52  
 Kidney, amyloid, 404  
 — arteriosclerotic, 402  
 — calculus of, 408  
 — cirrhosis, 402  
 — floating, 412  
 — granular, 402  
 — large white, 399  
 — movable, 412  
 — tuberculosis, 410  
 — tumours, 411  
 Kidneys, diseases of, 380  
 — inflammation, 394, 397  
 Klebs-Loeffler bacillus, 54  
 Knee-jerk, 423  
 Koplik's spots, 41
- Lactic acid, test for, 159  
 Landry's paralysis, 438  
 Lardaceous disease of kidney, 404  
 Laryngismus stridulus, 335  
 — — etiology and symptoms, 336  
 Laryngitis, catarrhal, 332  
 — chronic, 334  
 — oedematous, 333  
 Laryngoscope, use of, 332  
 Larynx, diseases of, 332  
 — paralysis, 337  
 — syphilis, 335  
 — tuberculosis, 334  
 — tumours, 335  
 Lateral sclerosis, 443  
 — — amyotrophic, 447  
 Lathyrism, 148  
 Lead colic, 145  
 — palsy, 145  
 Lead-poisoning, 144  
 — symptoms, 145  
 — treatment, 146  
 Leishmania Donovanii, 110  
 Lenticular nucleus, 420  
 Leprosy, 115  
 — anæsthetic, 116  
 — symptoms, 116  
 — treatment, 117  
 — tubercular, 116  
 Leptomeningitis, 429, 431  
 — cerebral, 465  
 Leucocythæmia, 303  
 Leucocytosis, 296  
 — in cerebro-spinal meningitis, 52  
 — in enteric fever, 23  
 Leucopenia, 297  
 Leukæmia, 303  
 — morbid anatomy, 303  
 — symptoms, 304



- Lienteric diarrhœa, 179  
 Lissauer's tract, 416  
 Little's disease, 481  
 Liver, abscess, 217  
   — — pyæmic and tropical, 218  
   — acute yellow atrophy, 212, 216  
   — amyloid, 228  
   — cancer, 225  
   — cirrhosis, 220  
   — — biliary, 223  
   — — portal, 220  
   — — — symptoms, 222  
   — — treatment, 224  
   — diseases of, 210  
   — fatty, 229  
   — functions of, 210  
   — glycogenic function of, 134, 210  
   — hydatid disease, 227  
   — passive congestion, 229  
   — syphilis, 224  
   — waxy, 228  
 Localisation, cerebral, 459  
   — of cerebral abscess, 472  
   — — tumours, 484  
   — spinal, Gowers' table, 432  
 Lock-jaw, 96  
 Locomotor ataxia, 439  
   — — symptoms, 440  
 Luetin reaction in syphilis, 105  
 Lumbago, 129  
 Lumbar puncture, 52  
 Lumbricus teres, 204  
 Lungs, abscess, 368  
   — anatomy of, 325  
   — cirrhosis, 358  
   — collapse, 347  
   — congestion, 349  
   — diseases of, 345  
   — examination of, 326  
   — fibrosis, 358  
   — gangrene, 368  
   — infarction, 350  
   — œdema, 348  
   — tuberculosis, 359  
   — tumours, 369  
 Lustig's anti-plague serum, 70  
 Lymphadenoma, 305  
 Lymphatic glands, in leukæmia, 304  
   — leukæmia, 303  
 Lymphatism, 307  
 Lymph-scrotum, 207  
 Lysogenesis, 14  
  
 Macrocytosis, 296  
 Macrophages, 16  
 Maidismus, 148  
 Main en griffe, 446  
 Malarial cachexia, 83, 86  
   — fever, 81  
   — — diagnosis, 86  
   — — etiology, 81  
 Malarial fever, intermittent, 83  
   — — morbid anatomy, 83  
   — — pernicious, 85  
   — — remittent, 84  
   — — symptoms, 83  
   — — treatment, 87  
 Malignant pustule, 89, 90  
 Malinering, diagnosis, 518  
 Mallein, 93  
 Malta fever, 80. *See* Mediterranean  
 Mania a potu, 142  
   — post-epileptic, 517  
 Mast cells, 297  
 M'Burney's point, 183, 184  
 Measles, 40  
   — complications, 42  
   — morbid anatomy, 41  
   — rash, 41  
   — treatment, 42  
   — varieties, 42  
 Mediastinal tumours, 378  
 Mediterranean fever, 80  
   — — etiology and symptoms, 80  
   — — treatment, 81  
 Medulla oblongata, diseases of, 452  
 Megaloblasts, 296  
 Ménière's disease, 507  
 Meningeal hæmorrhage, 431, 480  
 Meningitis, cerebral, 464  
   — — treatment, 468  
   — — tuberculous, 466  
   — epidemic cerebro-spinal, 50  
   — gummatous, 482  
   — posterior basic, 53  
   — spinal, 429  
   — — acute, 429  
   — — — treatment, 430  
   — — chronic, 430  
   — — — treatment, 431  
 Meningo-myelitis, 433, 435  
 Metabolism of carbohydrates, 210  
   — of proteids, 211  
 Metasyphilitic diseases, 103  
 Metchnikoff's theory of immunity, 16  
 Micrococcus lanceolatus, 351  
   — melitensis, 80  
   — rheumaticus, 64  
 Microcytosis, 296  
 Microgyria, 480  
 Microphages, 16  
 Migraine, 514  
 Milne's treatment of scarlet fever, 39  
 Mitral pulse, 271  
   — stenosis, murmur of, 267  
   — regurgitation, murmur of, 267  
 Möbius' sign, 318  
 Monoplegia, 427  
 Morbilli, 40. *See* Measles  
 Morbus coxæ senilis, 131  
 Morbus maculosus Werlhofii, 312  
 Moro's reaction, 365



- Morphinism, 143  
 Motor path, 417  
   — tracts in cord, 415  
 Mouth, diseases of, 151  
 Mucous colitis, 182  
 Multiple neuritis, 515  
   — sclerosis, 488  
 Mumps, 46  
 Murexide test, 383  
 Murmurs, aneurysmal, 248  
   — cardiac, 246, 267  
   — endocardial, propagation of, 266  
   — exocardial, 248  
   — hæmic, 248  
   — valvular, characters, 267  
 Muscles, diseases of, 533  
 Muscular atrophy, peroneal type, 446  
   — — progressive, 445  
   — dystrophy, 533  
   — — diagnosis, 535  
   — — hereditary, 534  
   — — juvenile, 534  
   — — facio-scapulo-humeral, 534  
   — sense, 426  
 Myalgia, 129  
 Mydriasis, 497  
 Myelitis, acute, 433  
   — — treatment, 435  
 Myelocytes, 297  
 Myocardium, diseases of, 276  
   — — treatment, 278  
 Myosis, 441, 497  
 Myotonia congenita, 535  
 Myotonic reaction, 536  
 Myxœdema, 313  
   — symptoms and treatment, 315  
  
 Negri's bodies in rabies, 94  
 Nematoda, 201  
 Neosalvarsan, 107  
 Nephritis, acute, 394  
   — — complications, 396  
   — — etiology, 394  
   — — morbid anatomy, 394  
   — — symptoms, 395  
   — — treatment, 396  
   — chronic, interstitial, 402  
   — — symptoms, 402  
   — — parenchymatous, 399  
   — — treatment, 405  
   — differential diagnosis, 400  
   — scarlatinal, 39  
 Nephrolithiasis, 408  
 Nerve cell, structure of, 413  
   — deafness, 507  
 Nerves, cranial, diseases of, 493  
   — spinal, diseases of, 510  
   — fifth, 501  
   — fourth, 499  
   — glosso-pharyngeal, 508, 509  
   — hypoglossal, 509  
   — ninth to twelfth, 508  
     — oculomotor, 496  
     — olfactory, 493  
     — optic, 493  
     — phrenic, 510  
     — pneumogastric, 508, 509  
     — seventh, 503  
     — sixth, 499  
     — spinal accessory, 509  
     — third, 496  
     — vagus, 508, 509  
 Nervous system, diseases of, 413  
   — — functional diseases of, 513  
 Neuralgia, 513  
   — treatment, 513  
   — trigeminal, 513  
 Neurasthenia, 525  
   — treatment, 526  
 Neuritis, 510  
   — multiple, 511  
   — — symptoms and treatment, 512  
   — optic, 484, 495  
 Neuroglia, structure of, 414  
 Neurone, structure of, 413  
 Neuroses of stomach, 175  
 Night-blindness, 495  
 Night-sweats of phthisis, 363, 367  
 Nissl's bodies, 414  
 Nodal rhythm, 285  
 Nodes, syphilitic, 104  
 Noma, 153  
 Normoblasts, 296  
 Nutmeg liver, 229  
 Nutrition, disorders of, 118  
 Nyctalopia, 495  
 Nystagmus, 489, 501, 507  
  
 Obstruction of bowels, 186  
 "Occult blood," tests for, 166  
 Ocular muscles, paralysis of, 497  
   — — spasm of, 501  
 Oculomotor nerve, 496  
 Œdema of glottis, 333  
   — of lung, 348  
 Œgophony, 372  
 Œsophagitis, 155  
 Œsophagus, spasm of, 156  
   — stricture of, 156  
   — tumours of, 157  
 Oidium albicans, 152  
 Olfactory nerves, 493  
 Oligæmia, 297  
 Oligochromæmia, 297  
 Oligocythæmia, 297  
 Ophthalmoplegia, 471, 501  
 Opisthotonos, 97  
 Oppler-Boas bacillus, 170  
 Opsonic index, 18  
 Opsonins, 14  
 Optic atrophy, 496  
   — chiasma, 495, 496



- Optic nerves, diseases of, 495  
 — neuritis, 484, 495  
 — thalamus, 419, 420  
 Osteo-arthritis, 131  
 Oxyuris vermicularis, 205
- Pachymeningitis, 429  
 — hæmorrhagica, 464  
 — internal hypertrophic, 431  
 Palpitation, 285  
 Pancreas, diseases of, 234  
 — in diabetes, 133  
 Pancreatitis, 234  
 Paragraphia, 488  
 Paralysis, 427  
 — agitans, 529  
 — alternate, 479  
 — bulbar, 452  
 — cerebral, 462  
 — — of childhood, 480  
 — crossed, 462, 479, 505  
 — of diaphragm, 510  
 — facial, 504  
 — general, 490  
 — glosso-labio-laryngeal, 452  
 — hysterical, 523  
 — infantile, 436  
 — Landry's, 438  
 — laryngeal, 337  
 — ocular, 497  
 — post-diphtheritic, 57  
 — pseudo-hypertrophic, 533  
 — syphilitic spinal, 444  
 Paralytic dementia, 490  
 Paraplegia, 427  
 — ataxic, 449  
 — cerebral, 481  
 — spastic, 443  
 Parasites, internal, 194  
 Parasyphilitic diseases, 103  
 Parathyroids, 316  
 Paratyphoid fever, 24  
 Parkinson's disease, 529  
 Parosmia, 493  
 Parotitis, epidemic, 46  
 Paroxysmal bradycardia, 278  
 — hæmoglobinuria, 311  
 — tachycardia, 284  
 Passive congestion, in heart disease, 263  
 Pasteur's method in rabies, 96  
 Pectoriloquy, 330  
 Peliosis rheumatica, 312  
 Pellagra, 148  
 Pemphigus, syphilitic, 103  
 Perforation, in appendicitis, 184  
 — in enteric fever, 23  
 — in gastric ulcer, 167  
 Pericardial friction, 248, 252  
 Pericarditis, 251  
 — diagnosis, 253  
 Pericarditis, suppurative, 251  
 — symptoms, 251  
 — treatment, 254  
 Pericardium, adherent, 253  
 Perihepatitis, 241  
 Peripheral neuritis, 511  
 Perisplenitis, 241  
 Peristaltic unrest, 175  
 Peritoneum, diseases of, 236  
 Peritonitis, acute, 239  
 — — etiology, 239  
 — — symptoms, 240  
 — — treatment, 241  
 — chronic, 241  
 — malignant, 243  
 — perforative, 240  
 — subphrenic, 240  
 — tuberculous, 242  
 — — treatment, 243  
 Pernicious anæmia, 300  
 Peroneal type of muscular atrophy, 446  
 Pertussis, 43. *See* Whooping-cough  
 Pestis, 67. *See* Plague  
 Petit mal, 517  
 Phagocyte count, 18  
 Phagocytosis, 14  
 Phagolysis, 16  
 Phantom tumour, 523  
 Phenyl-hydrazin test for sugar, 386  
 Phrenic nerve, 510  
 Phthisis, 359  
 — acute, diagnosis, 374  
 — bronchopneumonic, 360  
 — chronic, 360  
 — — complications, 364  
 — — physical signs, 361, 363  
 — — symptoms, 361, 362  
 — — treatment, 365  
 — fibroid, 364  
 — pneumonic, 360  
 Pigeon breast, 125  
 Pigmentation in Addison's disease, 320  
 — arsenical, 147  
 Pituitary body, diseases of, 323  
 Plague, 67  
 — bubonic form, 68  
 — morbid anatomy and symptoms, 68  
 — pneumonic form, 69  
 — treatment, 70  
 Plasmodium malarie, 81  
 Pleura, diseases of, 369  
 Pleurisy, 369  
 — diagnosis, 373  
 — physical signs, 371  
 — symptoms, 371  
 — treatment, 376  
 Pleurodynia, 129  
 Plumbism, 144  
 Pneumobacillus, Friedländer's, 351



- Pneumococcus, 351  
 Pneumogastric nerve, 508, 509  
 Pneumonia, catarrhal, 356  
   — chronic, 358  
   — — symptoms, 359  
   — interstitial, 358  
   — lobar, 351  
   — — complications, 355  
   — — etiology and morbid anatomy, 351  
   — — physical signs, 352  
   — — prognosis, 355  
   — — symptoms, 353  
   — — treatment, 355  
   — — varieties, 354  
   — lobular, 356  
 Pneumonokoniosis, 359  
 Pneumothorax, 376  
   — artificial, 368  
   — treatment, 377  
 Podagra, 118  
 Poikilocytosis, 296  
 Polioencephalitis, 470  
 Poliomyelitis, acute anterior, 436  
 Polychromatophilia, 296  
 Polygraph, 250  
 Porencephaly, 471, 480  
 Post-epileptic states, 517  
 Posterior basic meningitis, 53  
 Precipitins, 14  
 Premature beats, 282  
 Progressive muscular atrophy, 445  
 Projection, erroneous, 499  
 Proteids, metabolism of, 211  
 Proteoses, test for, 160  
 Pseudo-angina, 281  
 Pseudo-hypertrophic paralysis, 533  
 Pseudo-tabes, alcoholic, 512  
 Ptomaine poisoning, 147  
 Ptosis, 497  
 Pulmonic regurgitation, murmur of, 268  
   — stenosis, murmur of, 267  
 Pulse, examination of, 249  
   — in cardiac diseases, 270  
   — intermittent, 283  
 Pulsus alternans, 284  
   — paradoxus, 253  
 Purin bodies, 119  
 Pus, tests for, 388  
 Purpura, 311  
   — hæmorrhagica, 312  
   — variolosa, 32  
 Pyæmia, 62  
 Pyæmic abscess of liver, 218  
 Pyelitis, 405  
   — symptoms, 405  
   — treatment, 407  
 Pyelonephritis, 405  
 Pylephlebitis, suppurative, 218  
 Pylorus, hypertrophic stenosis of, 175  
 Pyonephrosis, 405  
 Pyopneumothorax, 377  
 Pyramidal system, 417  
   — tracts of spinal cord, 415  
 Pyramids, decussation of, 417  
 Pyrosis, 174  
 Quinsy, 154  
 Rabies, 94  
 Râles, 330  
 Rash in chicken-pox, 35  
   — in dengue, 50  
   — in enteric fever, 22  
   — in epidemic cerebro-spinal meningitis, 52  
   — in measles, 41  
   — in röteln, 43  
   — in scarlet fever, 37  
   — in small-pox, 31  
   — in typhus fever, 27  
 Rashes in fevers, 19  
 Raynaud's disease, 531  
   — — symptoms, 531  
   — — treatment, 532  
 Reaction of degeneration, R.D., 424  
 Receptors, 15  
 Reflexes, 422  
 Reflex iridoplegia, 441, 497  
 Regurgitation, aortic, murmur of, 267  
   — — pulse of, 271  
   — mitral, murmur of, 267  
   — tricuspid, 262  
   — — murmur of, 267  
   — — symptoms, 269  
 Relapsing fever, 28  
   — — complications, 29  
   — — symptoms, 29  
   — — treatment, 30  
 Relative insufficiency of cardiac valves, 261  
 Renal calculus, 408  
   — — etiology and symptoms, 409  
   — — treatment, 410  
   — colic, 409  
   — tuberculosis, 410  
   — — treatment, 411  
   — tumours, 411  
 Rennin, test for, 160  
 Resonance, amphoric, 328  
   — vocal, 330  
 Respiratory organs, diseases of, 325  
 Retinitis, 495  
   — albuminuric, 398  
 Retropulsion, 530  
 Rhachitic rosary, 125  
 Rhachitis, 123  
 Rheumatic fever, 64  
 Rheumatism, acute, 64  
   — — complications and diagnosis, 66  
   — — etiology, 64



- Rheumatism, acute, morbid anatomy, 65  
 — — symptoms, 65  
 — — treatment, 67  
 — chronic, 128  
 — gonorrhœal, 99  
 — muscular, 129  
 Rheumatoid arthritis, 129  
 Rhonchi, 330  
 "Rice-water" stools, 71  
 Rickets, 123  
 — late, 125  
 — symptoms, 124  
 — treatment, 126  
 Rigidity, early and late, 425, 474, 479  
 Risus sardonius, 97  
 Romberg's sign, 441  
 Rôtheln, 42  
 Roundworms, 201, 204  
 Rubella, 42  
 Rubro-spinal tract, 416  
 Rupia, 102  
 Russo's test in enteric fever, 24  
 Rhythm, disorders of cardiac, 281  
  
 Saccharomyces albicans, 152  
 Salvarsan, 106  
 Sapræmia, 62  
 Saturnine cachexia, 145  
 — encephalopathy, 146  
 Saturnism, 144  
 Scarlatina, 36  
 Scarlet fever, 36  
 — — complications, 38  
 — — etiology, 36  
 — — morbid anatomy, 37  
 — — rash and symptoms, 37  
 — — treatment, 39  
 — — varieties, 38  
 Sclerosis, amyotrophic lateral, 447  
 — disseminated, 488  
 — insular, 488  
 — lateral, 443  
 — multiple, 488  
 — of nervous tissues, 427  
 Scurvy, 308  
 — treatment, 309  
 — infantile, 309  
 Secondary deviation, 499  
 Sensory decussation, 419  
 — path, 418  
 — tracts in cord, 416  
 Septic diseases, 62  
 — — symptoms, 63  
 — — treatment, 64  
 Septicæmia, 62  
 Serum, antibacterial, 13  
 — antitoxic, 13  
 — antityphoid, 26  
 — disease, 59  
 Serum treatment of anthrax, 91  
 — — of bacillary dysentery, 76  
 — — of diphtheria, 58  
 — — of enteric fever, 25  
 — — of cerebro-spinal fever, 53  
 — — of erysipelas, 61  
 — — of exophthalmic goitre, 319  
 — — of pernicious anæmia, 303  
 — — of plague, 70  
 — — of scarlet fever, 39  
 — — of septicæmia, 64  
 — — of tetanus, 97  
 Serum-anaphylaxis, 17  
 Seventh nerve, 503  
 Shaking palsy, 529  
 Sick headache, 514  
 Siderosis, 359  
 Silicosis, 359  
 Sinus arrhythmia, 282  
 — thrombosis, cerebral, 469  
 Siriasis, 149  
 Sixth nerve, 499  
 Sleeping sickness, 108  
 Small-pox, 30  
 — complications, 33  
 — etiology, 30  
 — morbid anatomy, 31  
 — symptoms, 31  
 — treatment, 33  
 — varieties, 32  
 Snuffles, syphilitic, 103  
 Softening of the brain, 476  
 Spasm, 425  
 — of cardia, 176  
 — facial, 506  
 — hysterical, 523  
 — of ocular muscles, 501  
 — œsophageal, 156  
 Spasmodic torticollis, 528  
 Spastic gait, 427  
 — paralysis, 427  
 — paraplegia, 443  
 — — etiology and symptoms, 444  
 — state, 425  
 Speech, cortical centres for, 486  
 — staccato, 489  
 Sphygmomanometer, 250  
 Spinal accessory nerve, 509  
 — cord, anatomy, 414  
 — — columns of, 415, 416  
 — — diseases of, 428  
 — — system diseases of, 436  
 — — — classification, 439  
 — — — diagnosis, 451  
 — — — tumours of, 436  
 — localisation, Gowers' table, 432  
 Spinal meningitis, 429  
 Spirillum Obermeieri, 28  
 Spirochæte pallida, 100  
 — pallidula, 108



- Spirochæte pertenuis*, 108  
*Spirochæta pallidum*, 100  
 Spleen, diseases of, 321  
   — in leukæmia, 304, 305  
   — in lymphadenoma, 306  
   — in malaria, 83  
   — movable, 321  
 Splenic anæmia, 322  
   — — symptoms, 322  
   — apoplexy, 89  
   — fever, 89  
   — tumours, characters of, 323  
 Splenisation of lung, 352  
 Splenomedullary leukæmia, 303  
 Splenomegaly, primary, 322  
   — tropical, 110  
 Sputum, varieties of, 331  
 Squinting, 499  
 Staccato speech, 489  
 Status epilepticus, 517  
   — lymphaticus, 307  
 Stellwag's sign, 318  
 Stenocardia, 279  
 Stenosis, aortic, 260, 267  
   — — pulse of, 270  
   — mitral, murmur of, 267  
   — pulmonic, 267  
   — of pylorus, hypertrophic, 175  
 Stercoral or fæcal ulcers, 182  
 Still's disease, 132  
 Stokes-Adams syndrome, 278  
 Stomach, atony of, 176  
   — cancer, 168  
   — — diagnosis, 170  
   — — treatment, 171  
   — — symptoms, 169  
   — catarrh, 160, 161  
   — cirrhosis of, 162  
   — dilatation, 173  
   — — treatment, 174  
   — functions of, 157  
   — hour-glass contraction, 163  
   — neuroses, 175  
   — pyloric stenosis, hypertrophic, 175  
   — ulcer, 162  
   — contents, examination of, 157  
 Stomatitis, 151  
   — aphthous, 152  
   — gangrenous, 153  
 Stools, of bacillary dysentery, 74  
   — choleraic, 71  
   — enteric, 23  
 Strabismus, 499  
 Strangulation of bowel, 186  
*Streptococcus erysipclatis*, 60  
*Streptotrichosis*, 87  
 Subphrenic abscess, 240  
   — — in gastric ulcer, 167  
 Succussion, gastric, 174  
   — uccussion, Hippocratic, 328, 377  
 Sugar, tests for, 385  
 Sunstroke, 149  
 Suppression of urine, obstructive, 410  
 Suprarenal glands, diseases of, 319  
 Syncope, local, 531  
 Syphilides, 102  
 Syphilis, 99  
   — cerebral, 481  
   — congenital, 103  
   — diagnosis, 104  
   — of heart, 277  
   — of intestines, 181  
   — of larynx, 335  
   — of liver, 224  
   — salvarsan in, 106  
   — tertiary, 102  
   — treatment, 105  
 Syringomyelia, 435  
 System diseases of spinal cord, 436  
   — — — classification, 439  
   — — — diagnosis, 451  
 Tabardillo, 26  
 Tabes, diabetic, 137  
   — dorsalis, 439  
   — mesenterica, 242  
 Tabetic arthropathy, 442  
 Tache cérébrale, 467  
 Tachycardia, 284  
*Tænia cucurbitina*, 196  
   — echinococcus, 199  
   — grisea, 198  
   — lata, 198  
   — mediocanellata, 197  
   — saginata, 197  
   — solium, 196  
*Tæniada*, 196  
 Tapeworms, 194  
   — symptoms, 198  
 Taste, nerves of, 502  
 Tendon reflexes, 423  
 Tetanus, 96  
   — symptoms, 97  
   — treatment, 97  
 Tetany, 521  
 Thalamus, optic, 419, 420  
 Thermic fever, 149  
 Third nerve, 496  
 Thomsen's disease, 535  
 Threadworms, 205  
 Thrombosis, cerebral, 476  
   — of cerebral sinuses, 469  
 Thrush, 152  
 Thyroid gland, diseases of, 313  
 Tic douloureux, 513  
 Tinnitus aurium, 507  
 Tongue in bulbar paralysis, 453  
 Tonsillitis, acute, 153  
   — chronic, 155



- Töpfer's method for free HCl, 159  
 Tophi, gouty, 120, 121  
 Torticollis, muscular, 129  
   — spasmodic, 528  
 Toxophile receptors, 15  
 Toxophore group, 15  
 Tracheal tugging, 291  
 Tracts of spinal cord, motor, 415  
   — — — sensory, 416  
 Trematoda, 208  
 Tremor, intention, 489  
 Trichina spiralis, 202  
 Trichiniasis, 203  
 Trichocephalus dispar, 206  
 Tricuspid regurgitation, 262, 267, 269  
 Trigeminal neuralgia, 513  
 Trismus, 96  
 Tropical abscess of liver, 218  
 Trophic realms of Wyllie, 417  
 Trousseau's sign, 521  
 Trypanosoma gambiense, 109  
 Trypanosomiasis, 108, 109  
 Tubercle bacillus, 112  
   — miliary, structure of, 112  
 Tuberculin, diagnostic use of, 365  
   — in treatment of phthisis, 366  
 Tuberculosis, 111  
   — morbid anatomy, 112  
   — of intestines, 182  
   — of kidney, 410  
   — of larynx, 334  
   — of lungs, 359  
   — meningeal, 466  
   — peritoneal, 242  
   — acute miliary, 113  
   — — — pulmonary form, 114  
 Tuberculous meningitis, 466  
 Tumours of brain, 483  
   — of intestines, 189  
   — of kidney, 411  
   — of larynx, 335  
   — of lungs, 369  
   — of mediastinum, 378  
   — phantom, 523  
   — of the spinal cord, 436  
   — splenic, 323  
 Typhoid fever, 19  
   — state, 8  
 Typhus fever, 26  
   — — etiology and symptoms, 26  
   — — rash and treatment, 27  
 Tubular breathing, 329  
 Uffelmann's test for lactic acid, 159  
 Ulcer, duodenal, 162, 166  
   — dysenteric, 74  
   — enteric, 23  
   — faecal or stercoral, 182  
   — gastric, 162  
 Ulcerative endocarditis, 255, 257  
 Uræmia, 392  
   — diagnosis, 393, 518  
   — latent, 410  
   — symptoms and varieties, 392  
 Urea, estimation of, 381  
   — formation of, 212  
 Ureameter, Doremus', 382  
 Uric acid, 382  
 Urine, albumin in, 384, 389  
   — albumose in, 385  
   — bile in, 387  
   — blood in, 390  
   — casts in, 388  
   — chlorides in, 383  
   — deposits, 381, 388  
   — in diabetes, 133  
   — examination of, 380  
   — sugar in, 385  
 Urobilin, 213  
 Vaccination, 34  
 Vaccine treatment, 17  
   — — of cholera, 73  
   — — of enteric fever, 26  
   — — of goitre, 316  
   — — of gonorrhœal arthritis, 99  
   — — of plague, 69  
   — — of phthisis, 366  
   — — of rheumatoid arthritis, 132  
 Vaccines, sensitised, 18  
 Vagus nerve, 508, 509  
 Valvular lesions of heart, 266 *et seq.*  
 Varicella, 35  
 Variola, 30. *See* Small-pox  
 Varioloid, 32  
 Vermiform appendix, anatomy, 183  
 Vesicular breathing, 328  
 Vestibular nerve, 506  
 Vestibulo-spinal tract, 416  
 Visual field, retraction of, 496, 523  
 Vocal fremitus, 327  
   — resonance, 330  
 Volvulus, 186  
 Vomiting, nervous, 175  
 Von Graefe's sign, 318  
 Von Pirquet's reaction, 365  
 Wassermann reaction, 104  
 Weir-Mitchell treatment, 177  
 Whooping-cough, 43  
   — bacteriology and etiology, 43  
   — symptoms, 44  
   — complications and treatment, 45  
 Widal's reaction, 23  
 Willis, circle of, 457  
 Winter cough, 340  
 Wintrich's sign, 363  
 Wolff-Junghaus test for gastric cancer, 170  
 Wool-sorter's disease, 89, 91  
 Word blindness, 487, 507



Word centres in cortex, 487

— deafness, 487

Worms, intestinal, 194

Wrist-drop, 145, 512

Writing centre in brain, 486

Wry-neck, spasmodic, 528

Xanthopsy, 205, 215

Yaws, 108

Yellow fever, 77

— — etiology and morbid anatomy, 78

— — symptoms, 78

— — treatment, 79

Yersin's anti-plague serum, 70

















