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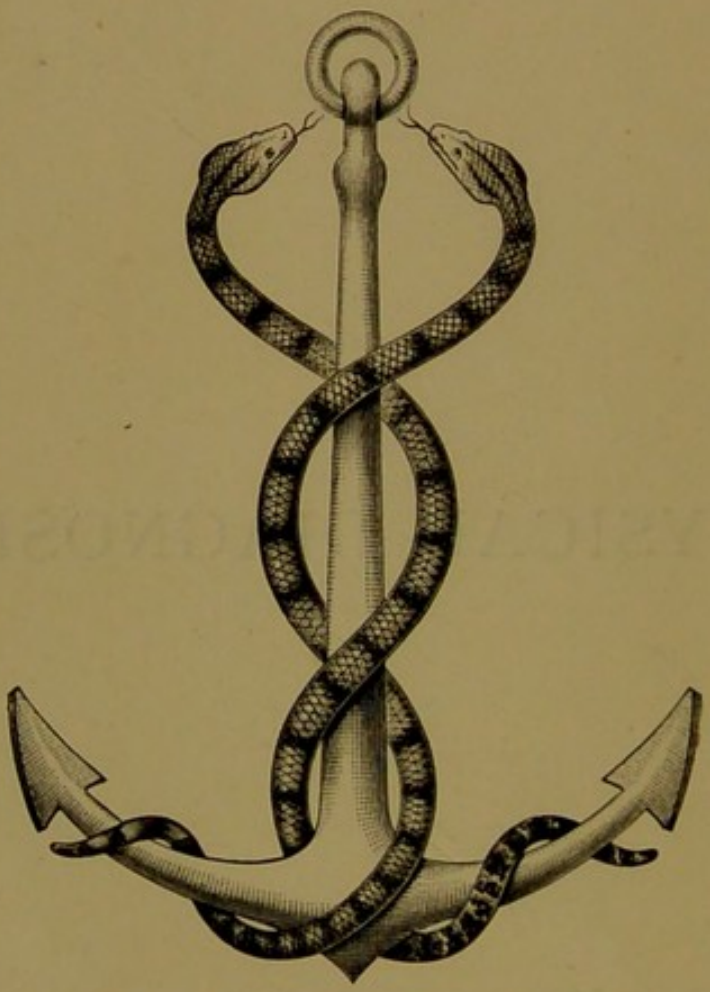
PHYSICAL DIAGNOSIS



NUNQUAM ALIUD NATURA, ALIUD SAPIENTIA DICIT.

GIBSON AND RUSSELL'S

PHYSICAL DIAGNOSIS

THIRD EDITION REVISED & REWRITTEN

BY

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WITH 144 ILLUSTRATIONS

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TO THE

PHYSICIANS TO THE ROYAL INFIRMARY

PAST AND PRESENT

WITH WHOM WE HAVE BEEN ASSOCIATED

IN CLINICAL WORK

PREFACE TO THE THIRD EDITION

THE Third Edition of this work has been revised and in great part rewritten, to bring it up to the requirements of modern medicine. New Sections have been added—on the "Examination of the Blood," the "Examination of the Gastric Contents," "Intestinal Parasites," the "Cranial Nerves," and on "Clinical Bacteriology"; while the illustrations have been largely increased.

The Section on the "Sense of Sight, with Examination of the Eye," has been revised by Dr. George Mackay, Ophthalmic Surgeon to the Royal Infirmary, who was originally responsible for its preparation.

Thanks are due to Dr. Noël Paton, Dr. Norman Walker, Dr. Lovell Gulland, and Dr. Logan Turner, for kindly advice and assistance.

EDINBURGH, *May* 1902.

PREFACE TO THE FIRST EDITION

In the following work are embodied the results of several years' experience in teaching methods of clinical investigation. We have limited ourselves almost entirely to the consideration of physical examination, as this is the department of practical medicine in which students require most assistance.

While we have no desire to avoid our united responsibility for this work, it is right to indicate the portions for which we individually must be held to be chiefly responsible. The sections devoted to the Integumentary, Urinary, and Nervous Systems, together with the Examination of the Pulse, are by Dr. Gibson; the Circulatory, Respiratory, and Alimentary Systems are by Dr. Russell.

No attempt has been made, except in a few instances, where the original writings will be found to be of special use, to acknowledge the various sources from which we have derived our knowledge of physical diagnosis. To do so fully would be practically impossible, as it would not only entail an enumeration of most of the works dealing with the subject from the time of Laennec to the present, but also render it necessary to estimate the influence of intercourse with many minds, as teachers, pupils, and friends.

We have gratefully to acknowledge our indebtedness to Dr. M'Bride, Surgeon to the Ear and Throat Department of the Royal Infirmary, for the sections on the Ear, Throat,

Pharynx, Naso-Pharynx, and Larynx; and to Dr. Mackay, Assistant Ophthalmic Surgeon to the Royal Infirmary, for those devoted to the Eye. These will, we believe, materially add to the value of the book.

We would also cordially express our obligations to Dr. Noël Paton for his kindness in revising the section dealing with the Urinary System, and for aiding us with many practical suggestions in regard to it.

We further wish to tender our thanks to Mr. William Keiller, F.R.C.P.Ed., for his efforts to realise our aim in the diagrams illustrating topographical anatomy and the physical conditions producing clinical phenomena.

EDINBURGH, *March* 1890.

CONTENTS

CHAPTER I.

PAGE

Introductory and Explanatory 1

CHAPTER II.

Methods and Terms 4

CHAPTER III.

Temperature 12

CHAPTER IV.

Integumentary System 18

CHAPTER V.

Circulatory System 31

CHAPTER VI.

Examination of the Blood 103

CHAPTER VII.

Respiratory System 126

CHAPTER VIII.

PAGE

THE ALIMENTARY SYSTEM, INCLUDING THE ABDOMINAL VISCERA 186

CHAPTER IX.

THE URINARY SYSTEM 239

CHAPTER X.

THE NERVOUS SYSTEM 302

CHAPTER XI.

THE SPECIAL SENSES 361

CHAPTER XII.

CLINICAL BACTERIOLOGY 417

APPENDIX 433

INDEX 437

LIST OF ILLUSTRATIONS

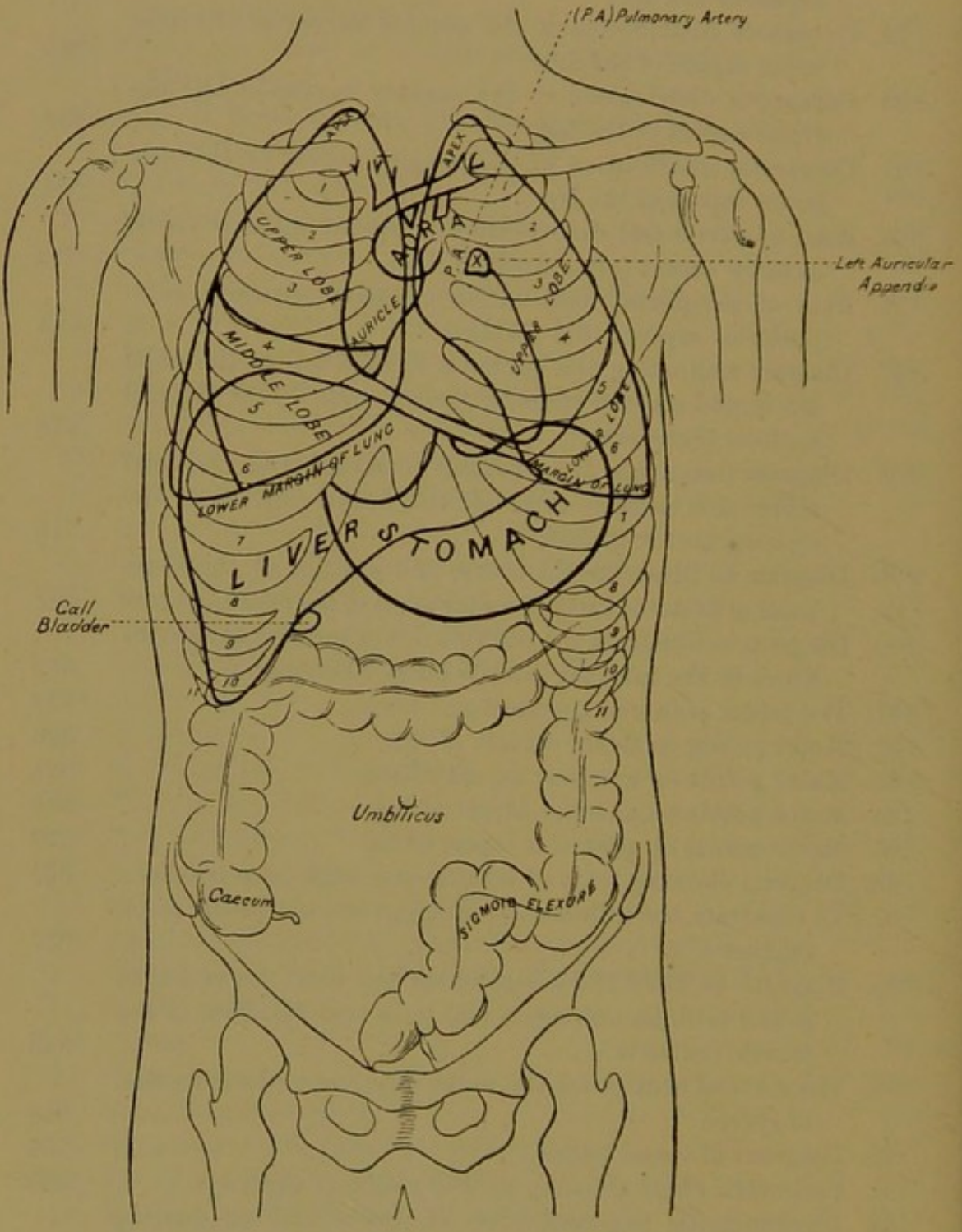
		PAGE
FRONTISPIECE I.	— Relations of Thoracic and Abdominal Viscera, Anterior view	xviii
FRONTISPIECE II.	— Relations of Thoracic and Abdominal Viscera, Posterior view	xix
FIG.		
1.	To illustrate the theory of percussion	8
2.	Febricula	13
3.	Typhoid fever with sustained temperature and lysis	14
4.	Typhoid fever, showing a regular diurnal remission	15
5.	Acute pneumonia, showing fall by crisis	16
6.	Showing intermittent type of temperature	16
7.	Showing relapsing type of temperature—Wunderlich	17
8.	Hair from case of tinea tonsurans	27
9.	Hair from favus	28
10.	*Microsporon furfur*	29
11.	Position of cardiac orifices in relation to the surface of the chest	32
12.	Showing percussion area of heart and liver	42
13.	Showing præcordial areas	44
14.	Normal first and second sounds—Diminished first sound	46
15.	Normal first and second sounds—Accentuated first sound	47
16.	Normal first and second sounds—Reduplicated first sound	48
17.	Normal first and second sounds—Accentuated second sound	50
18.	Normal first and second sounds—Reduplicated and accentuated second sound	51
19.	Diagrammatic representation of the cardiac cycle (after Gairdner)	54
20.	Presystolic murmur	55
21.	Systolic murmur	55
22.	Diastolic murmur	55
23.	Presystolic and systolic murmurs coexisting	56
24.	Systolic and diastolic murmurs coexisting	56
25.	Presystolic, systolic, and diastolic murmurs coexisting	56

FIG. PAGE
26. Showing aortic systolic murmur from stenosis . . 57
27. Showing aortic diastolic murmur from incompetence . 57
28. Showing mitral systolic or regurgitant murmur from failure of valve to close orifice 60
29. Showing diastolic or presystolic mitral murmur due to adhesion of mitral cusps 60
30. Area of audition of aortic systolic murmur (Gibson) . 62
31. Area of audition of aortic diastolic murmur (Gibson) . 63
32. Area of audition of aortic and mitral systolic murmurs (Gibson) 64
33. Area of audition of mitral systolic greater than area of audition of mitral diastolic murmur (Gibson) . . 65
34. Area of audition of presystolic mitral greater than area of audition of mitral systolic murmur (Gibson) . . 66
35. Area of audition of presystolic and diastolic mitral and systolic mitral and tricuspid murmurs (Gibson) . . 66
36. Area of audition of presystolic, systolic and diastolic mitral, and presystolic and diastolic tricuspid murmurs (Gibson) 72
37. Tracing taken with Marey's sphygmograph from the radial artery in a case of mitral incompetence . . . 88
38. Tracing taken with Sommerbrodt's sphygmograph from the radial artery in a case of mitral incompetence . . 88
39. Tracing from pulse of healthy man 90
40. Diagram of pulse tracing in health 90
41. Tracing taken from a case of advanced atheroma of the arteries by means of Marey's sphygmograph . . 92
42. Tracing taken with Marey's sphygmograph from the radial artery in a case of mitral stenosis . . . 93
43. Tracing taken with Marey's sphygmograph from the radial artery in a case of aortic incompetence . . . 95
44. Tracing taken with Marey's sphygmograph from the radial artery in a case of aortic stenosis 96
45. Diagram of fully dicrotic pulse 97
46. Diagram of hyperdicrotic pulse 97
47. Diagram of pulse of low tension 98
48. Diagram of pulse of high tension 98
49. Tracing from the internal jugular vein in a case of tricuspid incompetence 101
50. Squares in field of Thoma-Zeiss hæmacytometer . . 104
51. Oliver's hæmoglobinometer 107
52. Normal blood film 113
53. Blood—pernicious anæmia 114

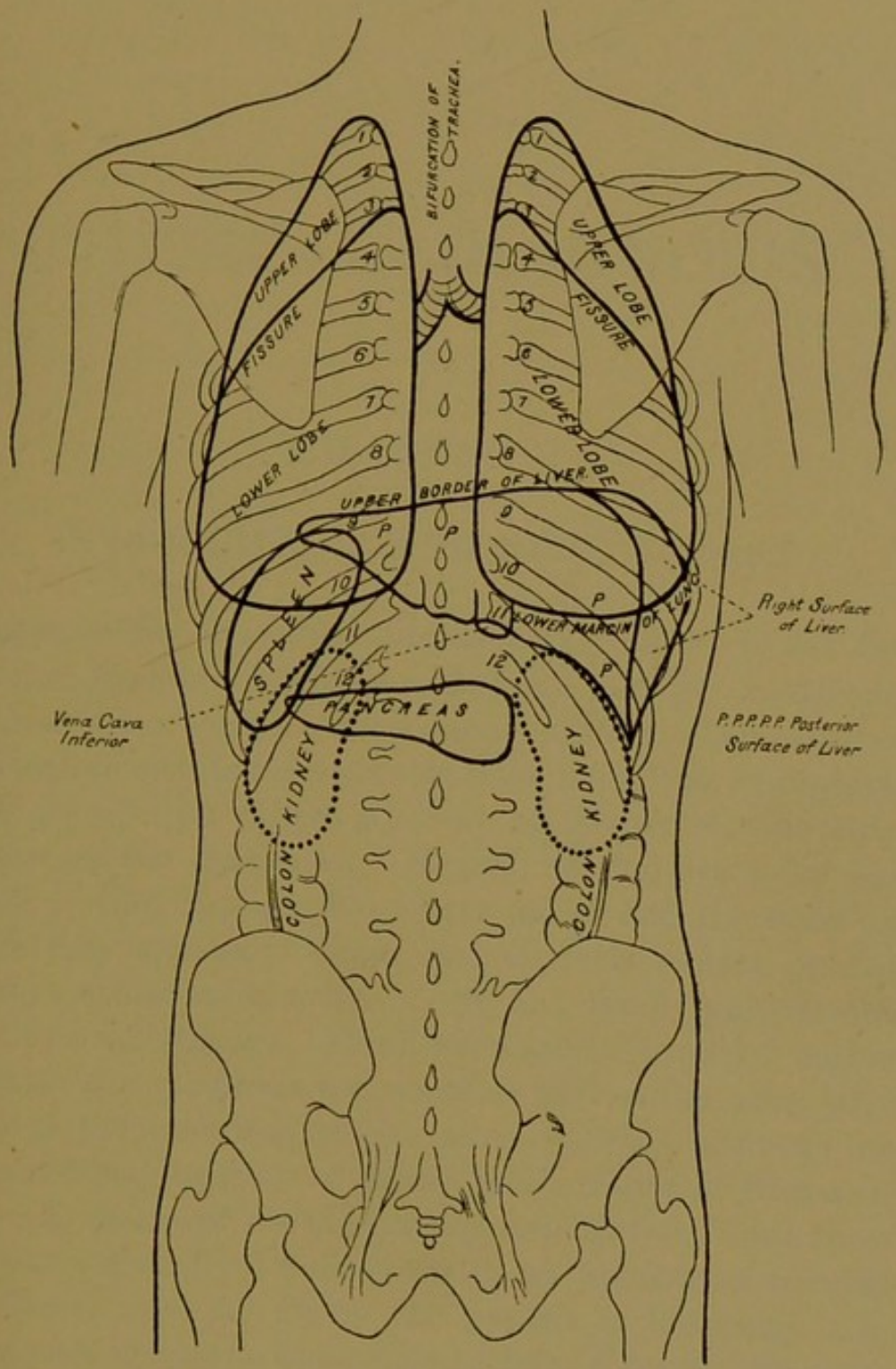
FIG.		PAGE
54.	Blood—spleno-medullary leucocythæmia	115
55.	Blood—lymphatic leucocythæmia	116
56.	Varieties of the asexual forms of the simple tertian malarial parasites found in the peripheral blood	123
57.	Anterior thoracic and abdominal regions	127
58.	Posterior thoracic and abdominal regions	128
59.	Normal chest (Gee)	131
60.	Rickety chest (Gee)	132
61.	Pigeon-breast (Gee)	132
62.	Emphysematous chest (Gee)	132
63.	Showing retraction of one side of the chest (Gee)	132
64.	Tracings from cases of Cheyne-Stokes breathing (Gibson)	137
65.	Vertical transverse section in mammary line, showing relations of lung and liver (after Symington)	145
66.	Diagram showing moderate dulness over tuberculous infiltration	147
67.	Diagram showing moderate dulness over superficial pneumonic patch	147
68.	Diagrammatic representation of varieties of vesicular breathing (after Wyllie)	159
69.	Bronchial breathing (modified from Wyllie)	161
70.	Broncho-vesicular breathing (modified from Wyllie)	162
71.	Diagrammatic representation of amphoric breathing	164
72.	Representing the production of rhonchi, and of crepitations in the bronchi and alveoli	165
73.	Crepitations (after Wyllie)	166
74.	To illustrate the graphic method of representing the physical signs of pneumonia	170
75.	Representing pneumonia of the inferior lobe	171
76.	To illustrate the graphic method of representing the physical signs of pleurisy with effusion	173
77.	Showing moderate pleural effusion with bronchial breathing and crepitations over the compressed and congested portion of lung	174
78.	Showing phthisis at various stages in one lung	176
79.	Showing the condition of parts in hydro-pneumothorax from a perforation in the pleura	179
80.	Showing a moderately distended stomach	199
81.	Taken from the post-mortem room, showing the U shape often assumed by the enlarged stomach	200
82.	From the post-mortem table, showing enlargement of the liver and distention of the stomach	208

FIG. PAGE
83. The microscopic appearance of the gastric contents from a case of gastrectasis 220
84. *Amœba coli* (after Lösch) 230
85. Tape-worm form of *Tœnia saginata s. mediocanellata* (Leuckart) 231
86. Head of *Tœnia saginata* in contracted and extended condition (Leuckart) 232
87. *Tœnia solium* (Leuckart) 232
88. *Bothriocephalus latus* (Leuckart) 233
89. *Distomum hepaticum* (Leuckart) 234
90. *D. hæmatobium*, male and female (Leuckart) . . 234
91. Ovum of *Bilharzia* (Leuckart) 234
92. Free embryo of *Bilharzia* (Leuckart) . . . 235
93. *Ascaris lumbricoides*, female (Leuckart) . . . 235
94. *Oxyuris vermicularis* (Leuckart) 236
95. Male *Ankylostomum duodenale* 236
96. *Ankylostomum duodenale*, male and female (Blanchard) . 237
97. *A. duodenale* (Sonsino) 237
98. *Trichocephalus dispar, in situ* (Leuckart) . . . 237
99. *Trichina spiralis* (Leuckart) 238
100. Ureometer of Russell and West 256
101. Ureometer of Doremus and Thursfield . . . 257
102. Uric acid 261
103. Uric acid 261
104. The spectrum of hæmoglobin 280
105. Phenyl-glucosazon 295
106. Urate of sodium 295
107. Urate of ammonium 296
108. Calcium oxalate 296
109. Ammonio-magnesium phosphate (prismatic form) . 296
110. Ammonio-magnesium phosphate (feathery crystals) . 296
111. Leucin and tyrosin 298
112. Cystin 298
113. Cholesterin 298
114. Squamous epithelium 298
115. Columnar epithelium from urethra . . . 298
116. Round epithelium from kidney 300
117. Hyaline (*a*) and blood (*b*) tube-casts . . . 300
118. Hard hyaline (*a*), epithelial (*b*), and granular (*c*) tube-casts 300
119. Transverse section of spinal cord 302
120. Cutaneous distribution of the sensory nerves of the head . 305

FIG.		PAGE
121.	Cutaneous distribution of the sensory nerves of the anterior aspect of the arm	308
122.	Cutaneous distribution of the sensory nerves of the posterior aspect of the arm	309
123.	Cutaneous distribution of the sensory nerves of the anterior aspect of the leg	310
124.	Cutaneous distribution of the sensory nerves of the posterior aspect of the leg	311
125.	Area of peripheral distribution of spinal sensory roots, anterior aspect (after Kocher)	312
126.	Area of peripheral distribution of spinal sensory roots, posterior aspect (after Kocher)	313
127.	Diagram indicating the probable localisation of the chief motor and sensory areas in the cerebral cortex (lateral surface) (Schäfer)	316
128.	Diagram indicating the probable localisation of the chief motor and sensory areas on the mesial surface of the cerebral cortex (Schäfer)	316
129.	Diagram to illustrate the course and position of the fibres passing from the cortical motor areas to the crus cerebrum	318
130.	Diagram to illustrate the position of the motor and sensory fibres in the internal capsule (after Sherrington)	319
131.	The motor points of the head and neck	322
132.	Motor points on flexor surface of arm	323
133.	Motor points on extensor aspect of arm	324
134.	Motor points on anterior aspect of leg	325
135.	Motor points on posterior aspect of leg	326
136.	Diagram showing seats of interference with deep reflexes	334
137.	To illustrate the influence of different lesions on the pupil reflexes	337
138.	Diagram to show the situation of the chief nerve nuclei in the medulla oblongata and pons near the floor of the fourth ventricle	343
139.	The cortical centres for the understanding and production of speech	356
140.	Diagram of visual paths	359
141.	Perimetric chart showing field of vision of right eye	393
142.	Diagrammatic representation of the visual conducting paths and fields of vision	398
143.	Larynx in quiet respiration (M'Bride)	409
144.	Film preparations of micro-organisms	418



FRONTISPIECE I.—Relations of Thoracic and Abdominal Viscera. Anterior view.



FRONTISPIECE II.—Relations of Thoracic and Abdominal Viscera. Posterior view.

PHYSICAL DIAGNOSIS

CHAPTER I

INTRODUCTORY AND EXPLANATORY

In approaching the subject of diagnosis, we have to ask the question, How is a diagnosis made? and in reply, it may be said that it is a complex, not a simple process. The evidence on which it rests is derived from three main lines of inquiry. First, we ask the patient what he complains of; the complaints are the *Symptoms*. Second, we inquire as to the length of time the symptoms have lasted, and the order in which they appeared; these, and all facts bearing on the patient's constitution, mode of life, and family antecedents, constitute the *History*. Third, we examine the part or region to which the symptoms are referable, and at the same time examine the condition of the other systems, although no complaint may be made of them; this is *Physical Examination*. By means of the information derived from these three sources we then form a diagnosis or an opinion as to what the disease is under which the patient is labouring. In many cases it is quite impossible to form a correct diagnosis without considering the facts derived from all three sources; in other cases, however, it is possible to form an absolutely correct diagnosis from the physical examination alone, and an opinion formed in this way is what is strictly speaking

physical diagnosis. In *all* cases, however, it is necessary to make a physical examination of the patient before forming a diagnosis, and it is with a view to help the student of clinical medicine in this important and difficult department that this book is written. In it we have in the main followed the lines which, in our experience as Tutors in Clinical Medicine in the Royal Infirmary of Edinburgh, have appeared to be most useful.

Physical Examination embraces and is confined to facts which are apparent to the senses of sight, touch, and hearing, and to these may be added smell.

The application of sight is known as *inspection*, touch as *palpation*, and hearing as *auscultation* or *percussion*, according to whether the sounds are produced by the organs themselves, or by tapping the different regions of the trunk.

It is of course necessary to be acquainted with what is to be seen, felt, and heard on examination of the different regions of the healthy body before the abnormal can be appreciated. It is necessary to be familiar with the normal shape of the abdomen, in order to describe the alterations which occur in disease; to know the character and position of the normal cardiac impulse, in order to appreciate deviations from health; and to have an intimate acquaintance with the normal sounds heard on auscultation, before studying the sounds present in disease.

Students ought, therefore, to avail themselves to the utmost of every opportunity of examining healthy individuals, particularly healthy chests, both in hospital and dispensary practice; they will thus become familiar with the normal and the variations which occur within the limits of health.

Physical diagnosis, however, includes not only investigations by means of the senses; it also inquires into the physical causes or conditions which produce the various phenomena. For instance, on inspection, an abdomen is found to be distended; by the aid of percussion and palpation, and the

application of an elementary knowledge of physics, it is decided whether it is distended with fluid in the peritoneal cavity or gas in the intestines. When this point is settled, we are in a position to state the various causes which may lead to the condition, but it is often impossible from this alone to form an opinion as to the cause in any particular case; this may only be possible after a careful consideration of the history and all the symptoms of the case. The possibility of forming a diagnosis from physical examination alone is distinctly limited, but no diagnosis ought ever to be attempted without making a physical examination. The symptoms and the clinical history of individual diseases will be found in the ordinary text-books of medicine, and they will not be dealt with at length in this volume.

CHAPTER II

METHODS AND TERMS

General Inspection.—By general inspection is meant the observation of the patient's appearance when he first presents himself, and during the time attention is directed to his complaints. The trained eye learns much in this way as to the temperament, constitution, possible habits, and the system, which may be at fault; and the student has to train himself to be an observer of these. Mental notes are made as to the state of nutrition, the gait and carriage, the colour of the skin and hair, the apparent age, the expression of the face and eye, the voice, any apparent abnormalities in exposed parts, and so on.

If the patient be seen in bed, similar observations have, so far as possible, to be made. In addition, however, are to be noted the decubitus, whether on the back or on the side, whether lying flat down or partially raised, whether the knees are drawn up or the legs extended, the look of exhaustion or of suffering, of consciousness or unconsciousness, of apathy or interest; the respiration, whether accelerated, or slow, or irregular; whether the nostrils move, or a look of suffering accompanies inspiratory efforts; if cough be present, whether it is "soft" or "hard," whether there is perspiration, whether the face is pale, flushed, or cyanotic. All these points will be noted almost automatically after a time if the observing faculties are carefully cultivated, and by that time their significance and value will be appreciated, but until then no reliable conclusion can be

drawn from them, and meanwhile they, or any other abnormal appearances, ought simply to be recorded.

Local Inspection.—In addition to the foregoing, the inspection of the various regions, especially of the thorax and abdomen, is necessary. In this are included shape, degree of plumpness or emaciation, movements, and so forth. It may also be made to include *mensuration*, that is, the determination of the size of the part.

Palpation.—By means of the hand or hands placed on a part are noted its degree of resistance, its hardness or softness, whether fluctuation be present or not, the degree and kind of movement, the presence of abnormal sensations, and similar facts. Under this head may be included *vocal fremitus*, a term which is confined to the examination of the lungs. It is the sensation which is felt by the hand when placed flat on the thorax while the patient speaks, or repeats the numeral *one, one, one*, or any combination of numerals.

The physical explanation of the phenomenon is that speaking sets up vibrations in the column of air in the trachea; these vibrations are propagated down the column of air in the bronchi and air vesicles, and, passing through the lung tissue and chest wall, are palpable to the hand placed on the chest. The intensity of the vocal fremitus varies greatly in different individuals, and depends on factors which will be considered under palpation of the lungs. For its production it is of course necessary that there should be no obstruction in the bronchi, which would arrest the passage of the vibrations.

Percussion.—This term is employed to denote the method of determining the physical condition of the viscera by the sound elicited on tapping the surface of the body.

Percussion may be carried out by tapping on the body directly, either by means of the finger or by means of a specially constructed hammer — the plessor. This direct method is termed *immediate* percussion. The body may, on the other hand, be tapped indirectly through the medium

of the finger laid flat upon the surface, or of a specially constructed instrument called a plexímeter. This method is known as *mediate* percussion, and is most commonly employed. By far the best instruments to employ in percussion are the fingers. In using the hammer and plexímeter, delicate gradations in the sense of resistance are lost—a point of very great importance. To carry out percussion, the first or second finger of the left hand is placed upon the part to be investigated, care being taken that the finger to be struck be in accurate apposition with the skin. The middle finger of the right hand being flexed at the first phalangeal joint, almost to a right angle, the observer strikes upon the plexímeter finger with a motion from the metacarpo-phalangeal joint or wrist. The motion must not be from the elbow, or the percussion becomes unnecessarily rough and shakes the patient.

Percussion is divided into gentle and strong, the difference depending upon the strength of the stroke.

Gentle percussion is always used when applicable. It is used when the parietes are thin or the organ to be defined lies superficially, or if there are structures of different physical characters in close proximity—as, for example, the intestine containing air behind the solid but thin anterior edge of the liver, or the solid liver behind the inferior edge of the right lung.

Strong percussion is used if the parietes be thick from muscle or fat, or if the organ whose limits are to be defined lies behind some other structure,—as, for instance, the left border of the heart, which is covered by lung.

For a thorough understanding of the results obtained by this method of investigation it is necessary to consider the conditions under which sound is produced and conducted.

Sound consists of undulations produced in a body which has been caused to vibrate. These are transmitted through various media, and conducted to the auditory apparatus. They thus reach the brain, where they are translated into

different sounds. Sound-vibrations are longitudinal, that is to say, they occur in lines parallel to the direction in which they are conducted, and they are therefore to be regarded as waves of condensation and rarefaction.

There are two kinds of sounds—musical sounds, or *notes*, and non-musical sounds, or *noises*. The former consist of undulations possessing regular wave-lengths, and recurring rhythmically or periodically, and have a fundamental tone. The latter consist of vibrations without any definite length of wave and having no fundamental tone.

If a sound be produced by striking upon the membrane of a drum the sound obtained will have a certain *tone*, for not only does the membrane vibrate but the contained air vibrates in consonance. Such a condition of matters is only possible in the healthy body on percussion of the abdomen, when a sound can be elicited from a simple cavity, such as the stomach, containing air. The sound then has tone, and is a musical note. Very different, however, are the conditions on striking over the chest. Vibrations are then initiated in the chest wall and in a greatly subdivided column of air contained within the bronchi and alveoli. Vibrations of the air column in consonance is impossible, and the sound obtained is not a musical sound, it does not have a fundamental tone. As most percussion sounds are non-musical in character, the term note should be avoided in the description of the percussion sound. The sound elicited on percussion varies in intensity, duration, pitch and quality.

The **intensity** or loudness of the sound depends upon the energy of the individual vibrations. The sound is louder, therefore, in proportion to the amount of force employed in the production of the vibrations. Thus the stronger the percussion stroke the louder the sound obtained, but in addition the loudness of the sound will vary in proportion to the capacity of the body for vibration. With an equal force a louder sound will be obtained over an air-containing

body than over a solid body, for the air-containing body possesses a greater capacity for being thrown into vibration.

The **duration** of the sound is a somewhat complex perception, which gives the observer the impression of a shallow or deep sound; the greater the duration of the sound the greater the impression of "depth" or "fulness"; the shorter the duration of the sound the more will it give the impression of "shallow" or "empty." The duration of the percussion sound depends upon the length of the column of air thrown into vibration. With firm percussion a full percussion sound will be got when a relatively long column of air is thrown into vibration; thus in the chest, on percussion over the whole thickness of the lung, as in the second right interspace. On passing downwards to the fourth and fifth interspaces the sound becomes more shallow as the lung thins off and is replaced in part by the solid liver, the column of air within the lung being thus diminished in length. It must not be forgotten that the strength of the percussion stroke may influence the duration of the sound. If the stroke be not sufficiently firm to set into vibration the whole subjacent column of air the sound obtained will be of shorter duration than if a firmer stroke be employed. This may be best illustrated by the accompanying diagram (Fig. 1). Firm percussion at *c* will give a much fuller sound than at *a*, but gentle percussion at *c*, throwing only a portion of the column of air into vibration, may give a shallower sound than firm percussion at *b*, throwing the whole column into vibration. An excellent demonstration of the duration of the percussion sound may be got by comparing the sound elicited on firm percussion in the second right interspace,

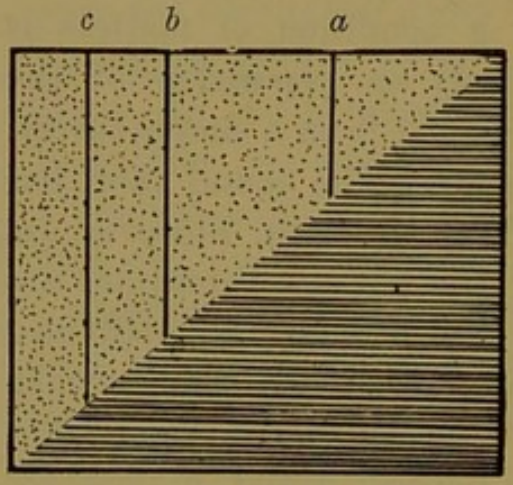


FIG. 1.

where the sound is full, with the sound in the fifth and sixth interspaces, where it is shallower.

The **pitch** of the sound depends upon the number of vibrations taking place in a given period of time; the greater the number of vibrations the higher the pitch. The percussion sound has four recognised gradations of pitch—(1) The tympanitic, or lowest pitched, is the sound got on percussion over the stomach or intestine when air-containing. (2) The normal thoracic percussion sound, or subtympanitic, is the sound obtained on percussion over the healthy lung. It is a degree higher in pitch than the tympanitic. (3) The tracheal sound, a grade higher in pitch, may be obtained by percussion over the trachea, with the mouth slightly open. (4) The osteal sound, the highest in pitch, can be elicited by percussion over bone or cartilage. These gradations in pitch can all be studied on the normal subject by percussion over the abdomen, third intercostal space, trachea, and skull.

The **quality** of the percussion sound depends upon the physical properties of the body thrown into vibration. There are two extremes of quality, clear or resonant and dull. A clear or resonant sound is got when percussing over an air-containing body, a dull sound when percussing over a solid body. Between these two extremes there are various gradations which will be fully considered under Percussion of the Chest.

The Sense of Resistance.—In the study of percussion attention must be directed to the *sense of resistance*, an accurate knowledge of which is of the greatest importance. On percussion over a solid body, such as the thigh, a marked sense of resistance will be noticed with both plessor and pleximeter fingers. This sense of resistance depends upon two factors, the consistency of the body and the volume of the body thrown into vibration. The greater the consistency the greater the sense of resistance; thus a greater sense of resistance is got from a solid than from an air-containing body. The greater the volume of the body the

greater the sense of resistance; thus two solid organs may give a different sense of resistance. This is well exemplified on percussion over the area of absolute hepatic dulness and over the area of absolute splenic dulness, the larger volume of the liver giving a greater sense of resistance than the smaller volume of the spleen.

Auscultation.—By auscultation the sounds produced by some of the organs are heard. It is chiefly devoted to the auscultation of the sounds produced in the respiratory organs during respiration, and in the heart and large vessels as the blood is flowing through them. The sounds are due to vibrations set up in these organs and propagated through the chest and stethoscope to the ear.

It may be practised by putting the ear directly to the part, when it is called *immediate auscultation*; but it is more commonly performed through the medium of the stethoscope, and is then called *mediate*. In using this instrument one or two precautions are necessary. Grasp it between two fingers and the thumb, and place it perfectly flat on the part at which it is proposed to listen; adapt your own position and body so that the ear can be brought flat upon it without moving it. If this is not carefully attended to, the stethoscope rides on its edge, hurts the patient, and the conduction of the sound is interfered with. The auscultator should assume as comfortable a position as possible, and he should avoid having his head hanging down. Further, he should be careful to lean with the hand not on the patient, but on the bed, and not to let the weight of the head rest on the stethoscope—in fact, to lean as lightly as possible on it.

Vocal resonance may be included under this head. When the stethoscope is applied over the lungs, and the patient asked to speak, as described under Vocal Fremitus, a sound is found to be conducted through the stethoscope to the ear, and it is to this conduction of the voice that the term is applied.

Combined percussion and auscultation, and other special methods, will be referred to when describing the physical examination of the various organs.

Succussion is the term applied to a splashing sound heard sometimes at a considerable distance, at other times only on auscultation, when the patient's body is shaken. It indicates the presence of fluid and air, and is of special importance when produced in the pleural cavity. It was first described by Hippocrates, and often has his name prefixed to it.

CHAPTER III

TEMPERATURE

The temperature of the body is usually taken in the axilla; in young children it is better, as a rule, to take it in the groin. The normal temperature in these situations is about 98°·4. The ordinary clinical thermometer ought to be left in for five minutes. If the half-minute thermometer be used, it is usually placed in the mouth under the tongue, and it requires to be carefully cleansed after being used. It must be remembered that the temperature is almost a degree higher in the mouth than in the axilla. Care has to be taken that the axilla is dry; for, if wet or moist, the condensation of vapour on the glass prevents its heating, and the mercury does not rise.

The temperature of the axilla is normally one degree below that of the rectum. In some special cases it is necessary to take it there, as the temperature of internal parts may be elevated, while that of the surface may be even lowered. This occurs, for instance, in some cases of malignant scarlet fever, as well as in other malignant fevers.

Normally the temperature is slightly higher in the evening than in the morning, and this normal habit is usually adhered to in disease, although they may both be on a level much above normal, or the variations may greatly exceed the normal variation. Occasionally, however, in diseased conditions the order is reversed, and the temperature is higher in the morning than in the evening.

The temperature is a factor of much importance in disease, and as its height varies it may be necessary not only to take it both morning and evening, but at shorter intervals. As a rule, however, morning and evening observations are sufficient, and, save in exceptional circumstances, more frequent observations are not practicable.

In nearly all acute inflammatory diseases the temperature is elevated. In children, gastro-intestinal disturbances, due to indigestion, often produce a rise of temperature of several degrees. We have also seen this in old people, but it is rare in them. Certain individuals have a very unstable temperature equilibrium, a slight catarrh or "cold" being sufficient to raise their temperature, and to lead to considerable discomfort; while in others it requires a more serious condition to disturb the equilibrium.

The length of time the temperature is raised depends on the duration and the nature of the illness, and is often the most reliable index as to its progress. The onset of acute disease is heralded by, amongst other symptoms, a more or less sudden rise in temperature, its course by a more or less continuously elevated one, and its decline by a gradual or sudden return to normal.

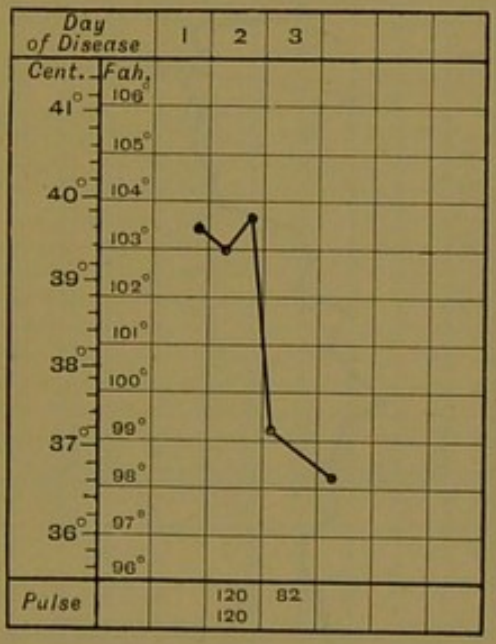


FIG. 2.—Febricula.

Febriculæ are short illnesses lasting from twelve to forty-eight hours, usually inaugurated by a slight rigor, or a feeling of chilliness, and associated with a rise of temperature of several degrees. The chart (Fig. 2) is from a case of this kind.

In illnesses of longer duration, the temperature curve presents some well-marked types.

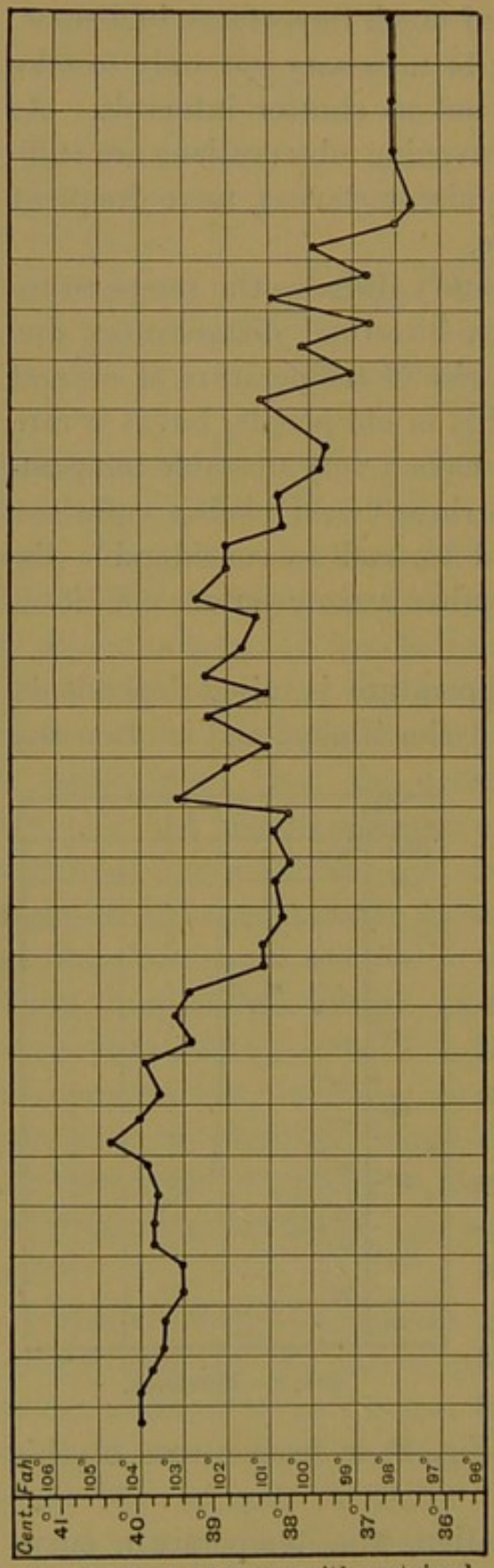


FIG. 3.—Typhoid fever, with sustained temperature and lysis—Recovery.

1. First, there is the type of **continued or sustained fever,** followed by gradual decline and return to normal, this mode of decline being known as *lysis.* It is typically seen in some cases of typhoid, but is not the type invariably shown by this fever. The annexed tracing (Fig. 3) is from such a case.

2. The chart (Fig. 4) is also from a case of typhoid fever, in which the variations in each period of twenty-four hours are considerable. This type of temperature might be called the **regularly remittent.** It is often present in septicæmia and in tuberculosis.

The temperature during the first days of the fever is not shown in either chart, as neither case came under observation sufficiently early, and this is the common experience. It is further to be noted that while the foregoing are types of temperature curve in typhoid, the duration of individual cases varies greatly, some being shorter than that represented in Fig. 4, while others are longer.

3. An **irregularly remittent** temperature is seen in phthisis and prolonged pyæmia, the variations of level being more extreme and much more irregular than in the preceding.

4. **Sustained** temperature, with a sudden fall to normal at the end of six to eight days, characterises acute croupous pneumonia. This sudden fall to normal is known as *crisis*, and is shown in Fig. 5.

While this sudden fall is characteristic of pneumonia, and indicates a favourable termination of the acute process, a similar fall in typhoid fever, for instance, would indicate a grave change for the worse, and probably a fatal issue from collapse, due to intestinal hæmorrhage or some equally serious condition such as perforation.

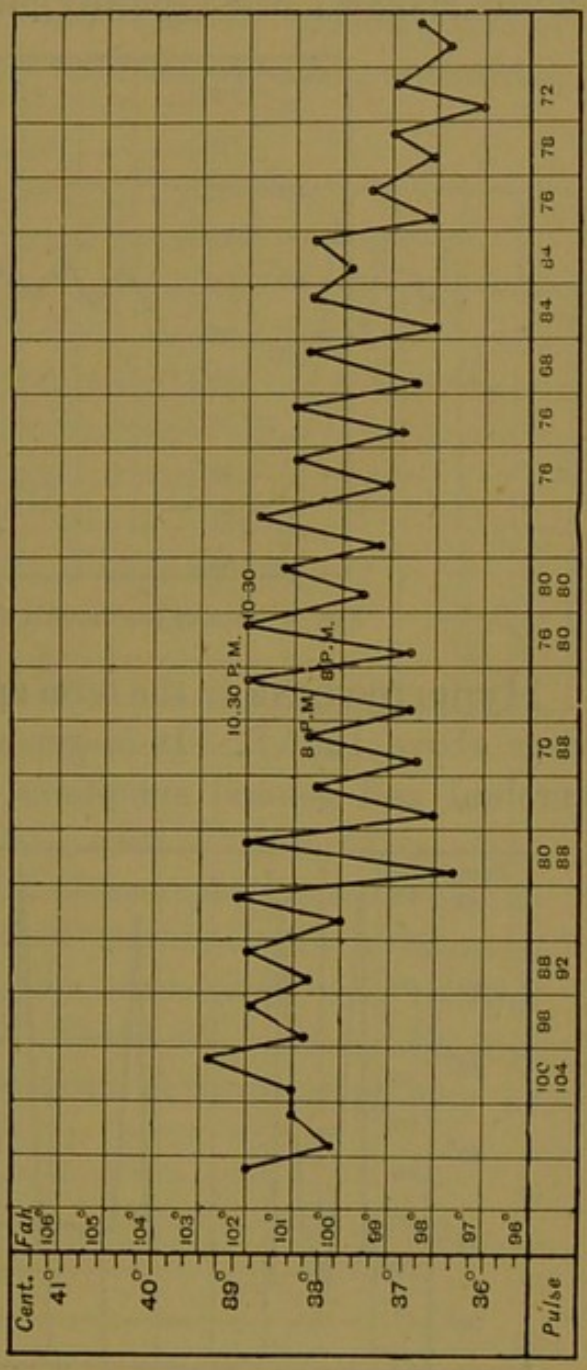


FIG. 4.—Typhoid fever, showing a regular diurnal remission—Recovery.

5. **Intermittent** is the type of temperature present in malarial fevers. Here there is either a break of one day between the febrile paroxysms, when it is called quotidian, of two days, when it is called tertian, or of three days, when it is termed quartan. The chart from Wunderlich (Fig. 6) represents these varieties.

6. **Relapsing** is the type of temperature represented in Fig. 7. There is an interval of several days between the febrile periods; it is typically present in relapsing fever.

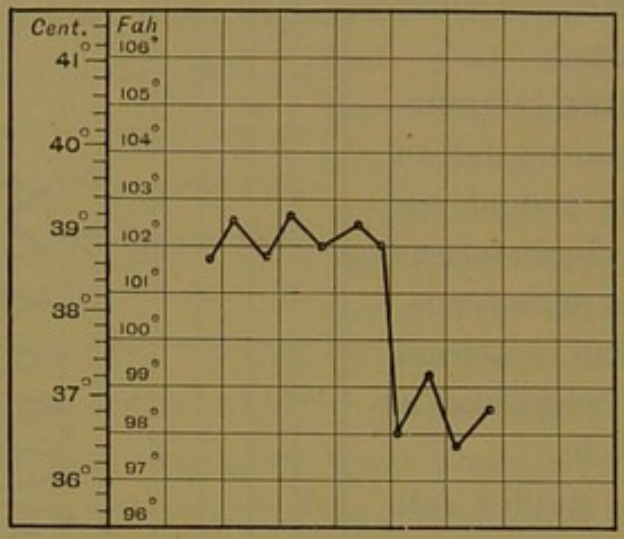


FIG. 5.—Acute pneumonia showing fall by crisis.

Hyperpyrexia is the term applied when the temperature rises above 105° F. It is generally accompanied by severe cerebral and general symptoms, and occurs in rheumatic,

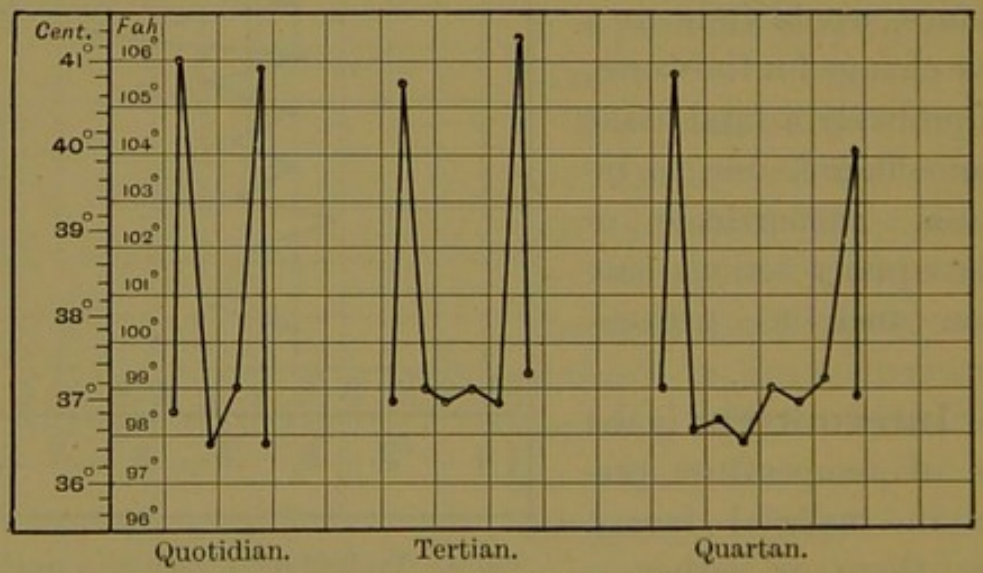


FIG. 6.—Showing intermittent type of temperature.

scarlet, typhoid, and other fevers, and such conditions as insolation.

When the temperature in acute or chronic disease suddenly rises above what it has reached for some days, it indicates

the involvement of a hitherto unaffected part. We have seen this in rheumatic fever when the heart became affected, in gangrene of the lung with the onset of inflammation of the pleura, in purulent pleurisy with the onset of pericarditis, and so forth.

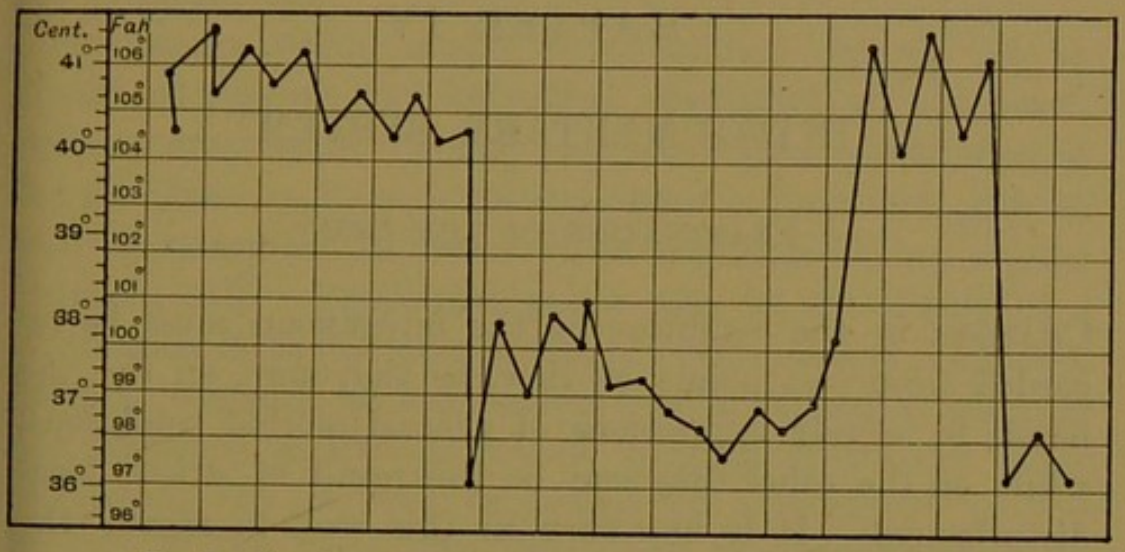


FIG. 7.—Showing relapsing type of temperature.—Wunderlich.

Relation of Temperature and Pulse rate.—The frequency of the pulse usually increases *pari passu* with the rise of temperature. This is not invariably the case, however. In typhoid fever, for instance, the temperature may be 102° or more, while the pulse is only about 80°. In tuberculous meningitis in children a like want of proportion may be present in the earlier stages.

CHAPTER IV

INTEGUMENTARY SYSTEM

Examination of the Skin

Changes in the condition of the integument must be regarded from two points of view, as they may, on the one hand, be merely expressions of general morbid conditions, while on the other they may be the result of strictly local disturbances. It is necessary in this way to regard the cutaneous and subcutaneous tissues as sharing in the state of the body at large, or as being the seat of more or less restricted lesions. These considerations lead to a division of the subject into the two heads of *general conditions* and *special affections* of the skin.

General Conditions

As very many disorders of the internal organs produce changes in the condition of the cutaneous and subcutaneous tissues, careful examination of these textures should in every case be made. The points to which attention should be directed are—

The colour of the skin.
The moisture of the skin.
The condition of the subcutaneous tissues.

In addition to these points the temperature of the skin is of the highest importance; this, however, has been dealt with in the previous section.

Changes in the Colour of the Skin.—The healthy tint of the European skin may undergo modifications in many directions.

Pallor of the Skin.—Paleness is due to deficiency of blood in the cutaneous capillaries, and this may be temporary or permanent.

Temporary pallor occurs from cardiac failure, as in emotional syncope and enfeebled conditions of the heart, such as fatty heart. It may also occur from constriction of the arterioles, as in such mental states as fear and pain, as well as under the influence of external conditions, as, for example, cold. Permanent pallor may be the result of a diminution in the number of the red corpuscles of the blood, or of a deficiency of hæmoglobin—as in chlorosis, anæmia, and leukæmia, as well as chronic Bright's disease and other affections. Or it may be caused by a reduction in the amount of the blood, due directly to hæmorrhages and indirectly to deficient assimilation.

Pallor of the skin is, as a rule, attended by paleness of the mucous membranes, which are apt to be dry. The causes which induce it sometimes lead to dropsy, and when this is the case the pallor becomes increased.

Redness of the Skin.—Redness of the skin is due to hyperæmia. Temporary excitement, such as is frequently caused by an interview with a medical man, may cause dilatation of the arterioles and rapid as well as powerful action of the heart, attended by flushing of the skin; this, however, rapidly subsides.

A more permanent redness is one of the most frequent symptoms of acute diseases, as seen in the flush over the malar bones in acute pneumonia, when it is of a dusky tint, and in hectic fever, where it is of a brighter hue.

A dull diffused redness of the face, with arborescent markings of a purple or blue colour, caused by stasis in the venous radicles, and commonly termed *venous stigmata*, is common in chronic disease of the heart, involving the

systemic veins, and is also very frequently a symptom of chronic alcoholism.

Blueness of the Skin, or **Cyanosis**, is the result of venous stasis, the dilated veins containing blood which is deficient in oxygen.

This condition is found in malformation, as well as disease, of the heart or pericardium. In the malformations of the heart which permit the venous and arterial blood to mingle together, the blood which is supplied to the systemic arteries is partly venous. Valvular lesions cause venous stasis by backward pressure from the left ventricle, or from inability on the part of the right cavities to forward the blood in the proper direction. Muscular degeneration of the heart is similar in its results, and the pressure of pericardial effusion occasionally produces cyanosis.

Cyanosis may be due also to affections of the respiratory system. Temporary venous stasis is very frequently observed during fits of coughing, which prevent the proper circulation of the blood through the lungs. Permanent venous stasis may result from prevention of the access of air by any narrowing of the larynx, trachea, or bronchi, *e.g.* in laryngeal diphtheria. It may also be caused by lessening of the aërating surface of the lungs, and this may occur in such changes of the texture of the lung as emphysema, or in compression over a large area of the branches of the pulmonary artery, the result of external pressure from effusion into the pleura.

Local cyanosis is occasionally to be observed as the result of pressure on large veins.

The colour of the skin in cyanosis varies from a pale bluish tint to a dark blackish-blue. When it is intense, the mucous membranes—as, for instance, the lips and gums—are also coloured, even the conjunctiva sharing in the venous stasis, and showing distinctly the distended veins. Pressure in this condition drives away the colour, which returns again immediately. It differs in this way from the colouring

which results from the prolonged administration of nitrate of silver. The veins of the neck and arms are frequently distended, and may undulate with the movements caused by respiration and circulation. The skin is cool, and the nose and ears, as well as the feet and hands, are, as a rule, cold. There is a tendency to arborescent networks on the surface of the lower extremities, and sometimes intense cyanosis is accompanied by œdema of the feet and ankles.

Yellowness of the Skin.—A yellow tint of the skin, constituting *icterus* or *jaundice*, is present in many cases either of temporary or permanent interference with the functions of the liver. As examples of obstructive causes of jaundice may be mentioned—catarrh of the duodenum and bile ducts; bodies within the ducts, such as gall-stones or parasites; and pressure on the ducts by external causes, such as accumulation of fæces in the bowel. Among the permanent causes, stricture and tumours of the bile ducts, and pressure upon them by tumours of other viscera, deserve passing notice.

The second class, sometimes termed hæmato-hepatogenous jaundice, includes the discoloration which sometimes follows the inhalation of anæsthetics, and also that which occurs in some acute general diseases, the absorption of some drugs, such as phosphorus and antimony, the virus of snake-bites, the poisons of many acute specific diseases, such as typhus, enteric, relapsing, malarial, yellow, and scarlet fevers, as well as epidemic jaundice. In this class of case there is an increased destruction of blood corpuscles with a consequent liberation of hæmoglobin. The bile pigment is not, however, formed in the blood, as was at one time supposed, when the condition was known as "hæmatogenous" jaundice. The hæmoglobin is carried to the liver and there converted, the resulting bile containing a high proportion of bilirubin and a proportionally small amount of bile acid. The bile thus formed being of a very viscid nature, there is difficulty in its excretion, the pressure in the smaller bile ducts rises,

and the secreted bile is absorbed by the lymphatics and passes into the circulation. If the destruction of blood corpuscles be on so large a scale that the liver cannot deal with the liberated hæmoglobin, or the liver function be diminished, hæmoglobinuria results; hence its relation to hæmato-hepatogenous jaundice.

Bronzing of the Skin.—Changes in the colour of the skin may be caused also by disease of the suprarenal capsules. In this condition (*i.e.* Addison's disease) the skin assumes a brown or bronze colour, more especially in those parts of the body which normally are darker than the rest, *e.g.* the axillæ and inner surface of the thighs. Bronzing of the skin is also found in some rare cases of diabetes with hypertrophic cirrhosis of the liver.

Bronzing, but to a less extent, is found in many cases of pregnancy, and it may be a symptom of pelvic disease in women. It is in this case termed *chloasma*.

A minor form of pigmentation is the lenticular form known commonly as freckles, which occurs on the parts of the surface exposed to the sun.

Greyness of the Skin.—The use of drugs may induce changes of colour; the prolonged use of nitrate of silver is sometimes followed by a grey discoloration known as *argyria*, due to reduction of silver in the tissues, which must not be mistaken for cyanosis. True pigmentation often results from the too long-continued use of arsenic, and often affects all the areas of the disease for which the drug was originally prescribed.

In special affections of the skin increased pigmentation is usually associated with long-continued inflammation or certain specific affections such as syphilis.

The Moisture of the Skin.—The amount of perspiration must be noted. It is greater at all times in the axillæ, perineum, palms, and soles than over the rest of the body. The perspiration is increased or diminished by mental conditions. Its amount is habitually greater in those who

inherit the rheumatic constitution, while in acute rheumatism it is profuse during the febrile stages of the disease. It constitutes the most important symptom of certain stages of disease, such as the sweating stage of ague, the crisis of continued fevers, the febrile attacks of tuberculosis, and the condition of collapse. Increased perspiration may occur locally in nervous diseases, being limited to the part supplied by the affected nerves. The perspiration is diminished in the early stages of fevers, and in such diseases as diabetes and interstitial nephritis.

The perspiration has a characteristic odour in uræmia, and the sweat of the axillæ and other regions may have a fœtid smell from its decomposition.

In jaundice the perspiration may have a yellow tint from the bile pigments which it contains, and other colours have been observed in some rare cases.

In increased perspiration the skin may be dotted all over with very small vesicles corresponding to the sudoriparous glands.

Condition of the Subcutaneous Tissues.— The subcutaneous textures vary in their conditions as to nutrition. The amount of fat deposited in those tissues may be greatly increased or very deficient. An increase in its amount is often hereditary, but may be due to errors of diet and exercise. Fat may be deficient as a constitutional habit of body, or may be a symptom of wasting disease, and if it should have appeared suddenly it points to the latter as the probable cause.

The subcutaneous tissues may be swollen, from the presence of the serum of the blood, and this constitutes the condition known as œdema or anasarca.

The position of the œdema may point to its cause, for, as a rule, in cardiac anasarca, the swelling begins at the ankle, while in that of acute Bright's disease it appears primarily below the lower eyelid. The affected part may only appear to be a little larger than usual, but, on the other hand, the

skin may seem to be greatly distended, and if this be so it is pale, smooth, and shining. If the finger be pressed upon the swollen part, an indentation is produced, which fills up gradually when the finger is removed.

The causes of œdema may be cardiac, vascular, or renal.

Cardiac dropsy may be primary, and due to the retardation of the return of venous blood by regurgitation into the veins, or it may be secondary to chronic pulmonary diseases.

Vascular œdema may be due to disease of the coats of a vein, such as thrombosis following phlebitis, or to pressure on one of the venous trunks, or to irritation of the vasodilator nerves of the part, as in angio-neurotic œdema.

Renal dropsy, as has been mentioned above, makes its appearance usually first in the face, especially in the eyelids. It soon becomes general, and is accompanied by dropsy in the various serous cavities. The colour of the skin is pale and pasty-looking.

Anasarca may be produced by deficiencies in the quality of the blood, which may be primary in anæmia, or occur in the course of wasting diseases.

The subcutaneous textures in the disease known as myxœdema are invaded by a gelatinous substance containing mucin. In this disease the whole surface of the body is swollen. The face becomes large, swollen, and expressionless, the hands blunt and clumsy, and along with these conditions there is hebetude of the nervous system. In myxœdema there is no indentation on pressure with the finger, and it affects every part of the body alike.

The subcutaneous tissues may be swollen from the presence of air. This is known as subcutaneous emphysema, and may occur under various conditions. In the lung of the child interlobular emphysema is a possible and by no means uncommon accident, as the result of frequent violent and

paroxysmal cough. The air then passes in the connective tissue surrounding the bronchi to the root of the lung, and thence diffuses into the mediastinum, neck, trunk, and general surface of the body. In the adult, there being no distinct interlobular connective tissue, such an accident is impossible. The air can then pass into the mediastinum only from an ulcerative lesion of the trachea or bronchi, or of the œsophagus. In some rare cases the air may pass into the subcutaneous tissue directly from the respiratory or alimentary tract. Thus in phthisis, where there is cavity formation, the surfaces of the pleura having become adherent, the ulcerative process may pass through the parietal pleura, and a sudden effort of coughing produce emphysema of the chest wall. In emphysema the skin is pale and elevated above its surroundings. The part is soft and yielding, quite unlike the brawny sensation given by œdema. There is no pitting on pressure as in œdema, the indentation at once disappearing. On handling, there is palpable and at times audible crackling. When the air has passed by way of the mediastinum the swelling first appears in the root of the neck, passing upwards and downwards over the chest wall, obliterating the normal outline.

Special Affections

It is quite beyond the scope of a volume such as this to enter into a consideration of the diagnosis of the special affections of the skin. The subject is one in which, to attain any skill, systematic knowledge is necessary as well as clinical experience. The diagnosis of a given case depends largely upon the results of inspection, but palpation gives useful information, while in many cases the microscope affords invaluable aid. For the consideration of the special affections of the skin the student is referred to the manuals of dermatology.

Parasites of the Skin

Animal Parasites.—Of the wingless insects which attack man, it is not necessary to describe the common forms, such as *Pulex irritans* (the common flea), *Pulex penetrans* (the sand flea), and *Acanthia lectularia* (the bed bug).

The **Pediculus**, or louse, gives rise to the condition known as *Pediculosis* or *phtheiriasis*. The **Pediculus corporis** lives in the clothes, but feeds upon the blood of the subject. The body of the insect is oval in outline, pale grey in colour. It is provided with a proboscis, which it inserts into the cutaneous follicles in order to suck the blood of the victim. **Pediculus capitis** lives on the scalp. It is smaller than the *Pediculus corporis*, has a triangular head and dark markings on the body. It deposits its eggs on the hairs, each egg being attached to the hair by a short stalk and being furnished with a lid. The **Pediculus pubis** is found in the genital regions, the axillæ, and on the eyebrows. It is smaller than the other varieties, is of a brownish colour, and has a more rounded body.

The **Acarus scabiei** gives rise to the condition known as *scabies*. The parasite is oval in shape, and is provided with strong jaws and four pairs of legs. The male is smaller than the female. The eggs are oval in outline. The female, after impregnation, excavates an oblique tunnel in the horny layer of the skin and lays her eggs as she advances. The favourite seats for these ravages are the thin skin of the webs of the fingers, the anterior borders of the axillæ, the genitals in males, and the areolæ of the nipples in females.

The **Acarus folliculorum** lives in dilated hair follicles and sebaceous glands. As a rule its presence gives rise to no serious results. It has on its head a pair of antennæ and a suctorial proboscis. The body is segmented.

The **Leptus autumnalis**, or harvest mite, is a reddish coloured mite which lives in grass and shrubs. During

harvest time it may pass to man, and cause troublesome irritation of the skin by its attacks.

Vegetable Parasites.—A number of skin affections are due to the presence of vegetable parasites, which in their growth affect the skin and its appendages.

Tinea tonsurans, or ringworm as a clinical disease, may result from one of two parasites,—the **Microsporon audouini**

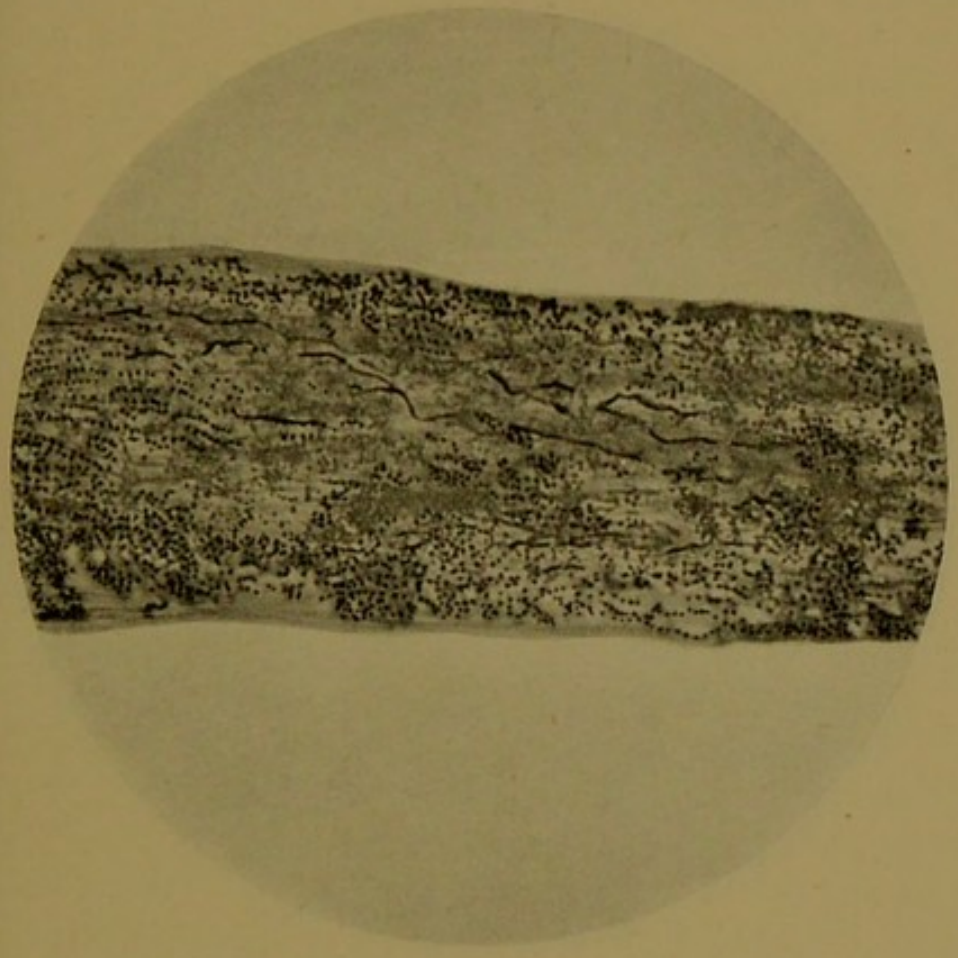


Fig. 8.—Hair from case of Tinea tonsurans. Showing *Microsporon audouini*, stained. × 200.

(small-spored variety) or the **Trichophyton megalosporon** (large-spored variety). The spores of the two fungi differ in size, but a still more characteristic difference is found in the arrangement of the spores: those of the microsporon are arranged irregularly in a *mosaic*, those of the trichophyton in the form of chaplets of beads or *rosaries*.

The affected hairs are short, dull, have lost their elasticity,

and are bent or twisted in all directions, or, in the large-spored variety, are broken off so short that the patch appears quite bald.

If ringworm be suspected the hairs or scales from the body should be carefully examined under the microscope; the hairs or scales should be soaked for a short time in diluted liquor potassæ before examination, when the para-

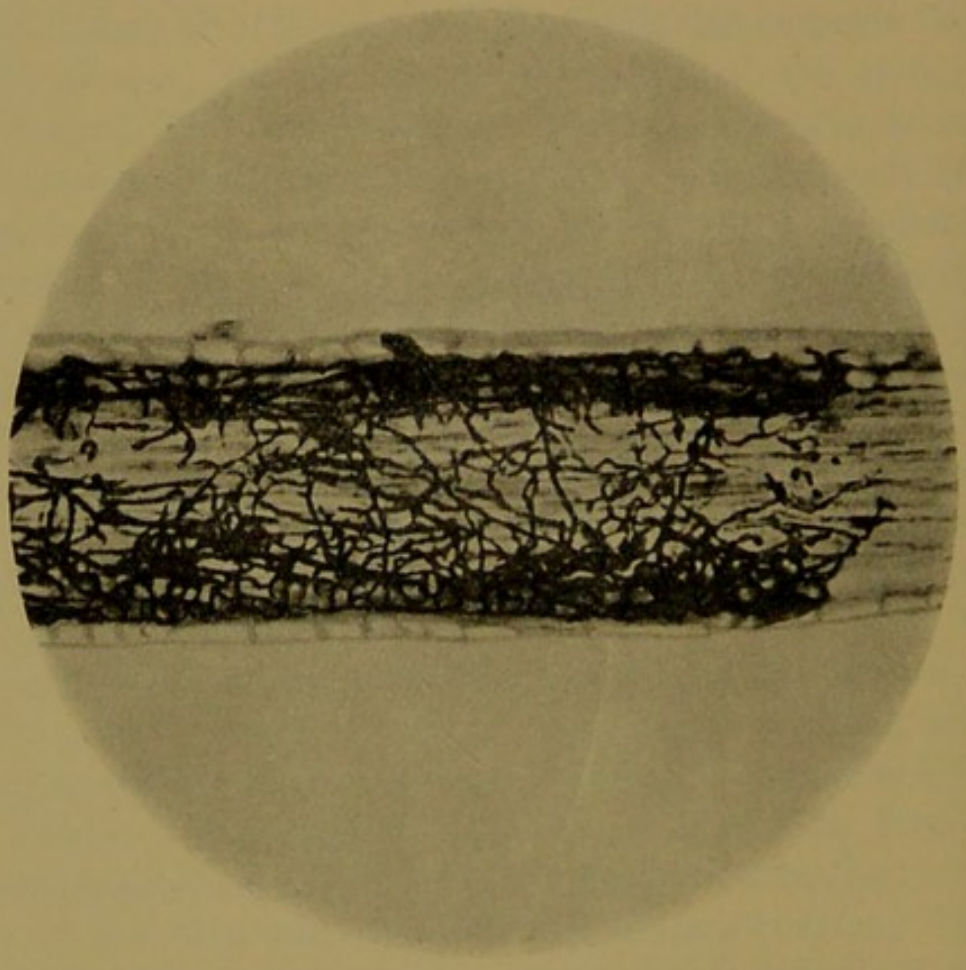


Fig. 9.—Hair from Favus. Showing *Achorion Schönleinii*, stained. × 200.

site, if plentiful, can be demonstrated. Care must be taken not to mistake the drops of oil emulsion, formed by the combination of the potash with the oily matter of the hair, for spores.

In doubtful cases the hair should be stained. The hair is first treated with a saturated solution of gentian-violet in aniline water for thirty minutes, and afterwards transferred

to Gram's solution of iodine (iodine 1, iodide of potash 2, water 300) for two minutes. The hair is then placed on a slide and firmly dried with blotting-paper, and a drop of aniline oil, containing enough iodine to give it a light mahogany colour, is applied. This removes surplus colour from the cells of the hair, while leaving it in the fungus, which can be readily seen. If a permanent preparation is

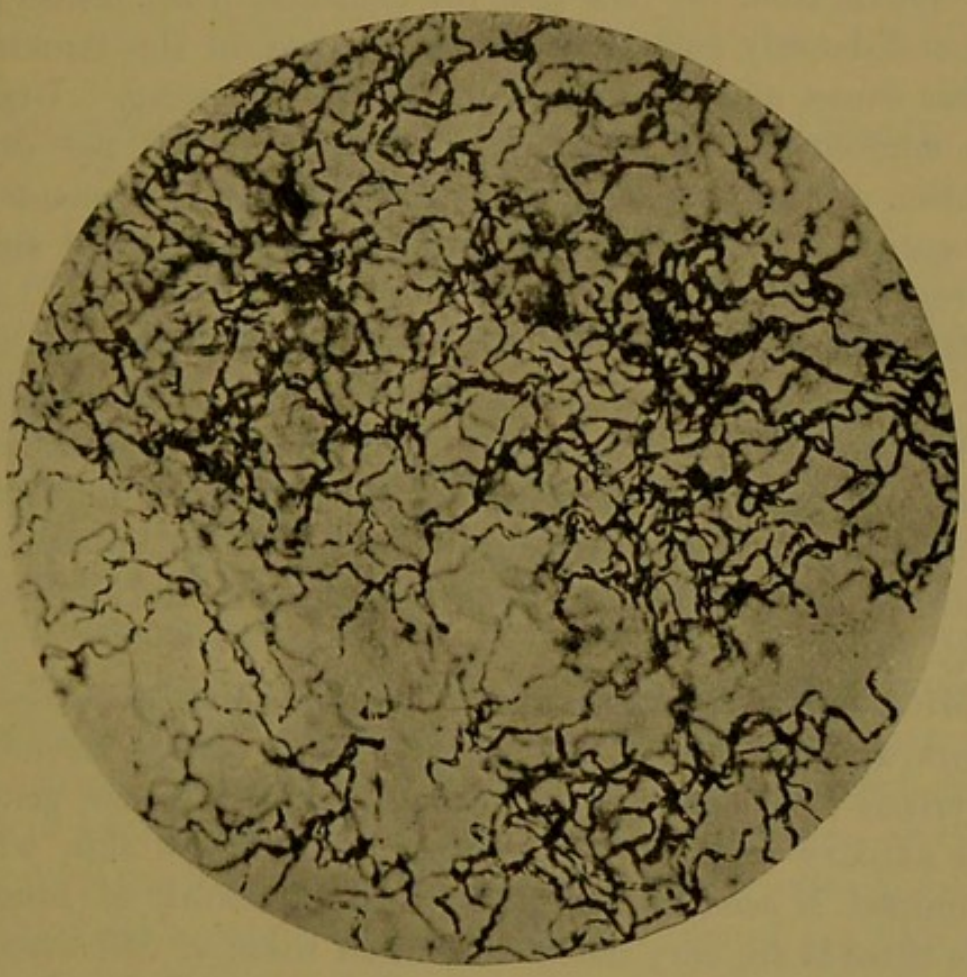


FIG. 10.—*Microsporon furfur*, stained. × 200.

desired, the aniline oil is washed out with xylol or benzol and the hair mounted in Canada balsam. Fig. 8 shows the appearance of the fungus from a stained preparation.

Achorion Schönleinii.—The fungus which is the cause of the disease known as *favus* differs from that of ringworm both in its method of growth and of attacking the hair. The hairs affected are not broken off, but have a stiff, lustre-

less appearance. Under the microscope the appearance differs from that found in ringworm. The elements of the fungus are longer than in ringworm, and entirely fill the hair so as to completely conceal its normal structure; there is no sign of medullary canal. The scutulum or crust, formed for the most part of fungus elements, is a sulphur-yellow mass of varying size, and cup-shaped or depressed in the centre, from the fact that the elements in the centre are dry and densely packed, while at the margin the structure is less dense, and more moist and actively growing. Under the microscope the scutulum, if soaked in 10 per cent. solution of caustic potash, shows long branching filaments of elongated bodies (mycelium) united to form rows, and small spores (Fig. 9).

Microsporon furfur is a fungus which has its growth in the superficial layers of the skin, giving rise to the condition known as *Pityriasis versicolor*. When the scales from a patch are examined in a drop of liquor potassæ under the microscope the fungus is seen, the mycelium as long branching filaments with groups of spores enclosed in the meshwork (Fig. 10).

Microsporon minutissimum, a fungus which gives rise to *Erythrasma*, is rare in this country. The disease resembles pityriasis versicolor, but is invariably limited to the genital and axillary regions. The horny layer of the skin when examined is seen to contain a dense feltwork of fungus. The threads are very much finer than those of *Microsporon furfur*, and if the scale is broken up and made into a cover-glass preparation the mycelium breaks up into bacillary-like segments. The spores are scanty and of smaller size than those of *Microsporon furfur*.

CHAPTER V

CIRCULATORY SYSTEM

Anatomical Relations of the Heart and Great Blood Vessels

For purposes of physical examination it is convenient to regard the heart as presenting a base, an apex, and three borders. The *base*, as seen from the front, is formed from right to left by the superior limit of the right auricle and its appendix, and the junction of the pulmonary artery with the conus arteriosus of the right ventricle. It extends approximately from the level of the third left costal cartilage, a short distance from the edge of the sternum, to the corresponding rib on the right side, or to the interspace above —that is, the second interspace,—and to about an inch to the right of the sternum. The *apex* is under the fifth left space or behind the sixth rib in the mammary line, and is formed by the left ventricle. The *right border* extends from the third right costal cartilage to the junction of the fifth or sixth costal cartilage with the sternum. This border is convex, the convexity being directed outwards, so that in the third and fourth spaces it is from $1\frac{1}{2}$ to 2 in. from the midsternal line: it is formed exclusively by the wall of the right auricle. The *inferior border* extends in a slightly curved line from the junction of the fifth or sixth right costal cartilage with the sternum to the apex: it is formed by the wall of the right ventricle and the apex of the left. The *left border* extends from the apex to the level of the

third left costal cartilage, where it joins the base: it is formed by the wall of the left ventricle, and is situated about $3\frac{1}{2}$ in. from the midsternal line.

The groove marking the boundary between the right auricle and ventricle is represented by a line drawn from the junction of the third left costal cartilage with the

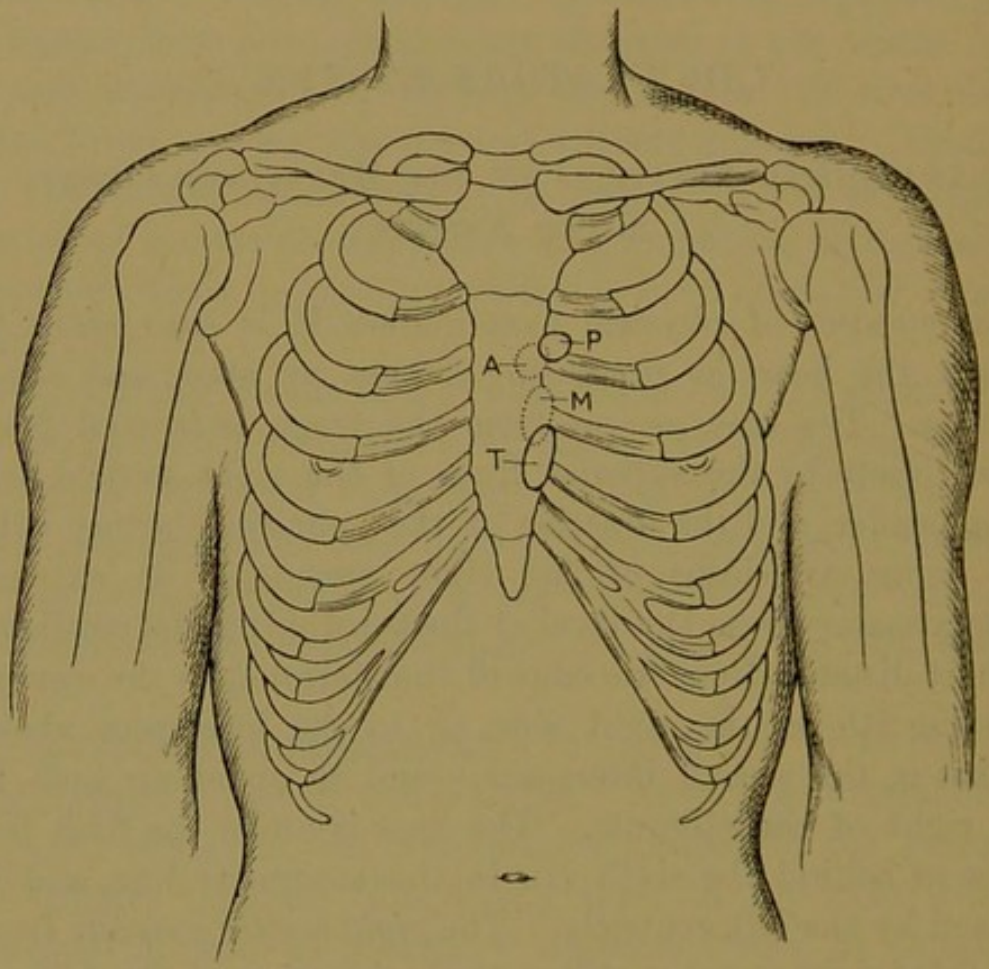


FIG. 11.—Position of cardiac orifices in relation to the surface of the chest.

sternum, downwards and to the right, to the junction of the fifth or sixth right costal cartilage with the sternum.

Posteriorly the base of the heart is at the level of the fifth dorsal vertebra, and the apex at the level of the eighth.

If the position of the four cardiac orifices be marked as in Fig. 11, it will be seen that they are all included within a very limited area of the surface of the thorax. They may be given thus—

Aortic orifice.—Sternum adjoining third *left* cartilage and a small portion of the second left intercostal space. One quarter of it is covered by the pulmonary orifice.

Pulmonary orifice.—Third *left* cartilage and sternum adjoining.

Mitral orifice.—*Left* half of sternum at level of the fourth cartilage.

Tricuspid orifice.—Middle of sternum from fourth to fifth cartilages.

Two of these, the pulmonary and tricuspid orifices, are comparatively near the chest wall; while the other two, the aortic and mitral, occupy a much deeper position in the chest.

The **left auricular appendix** is opposite the second left space, and to the outer side of the pulmonary artery, which it touches.

The **right auricular appendix** lies behind the right half of the sternum, on a level with the third cartilage.

These and some of the following relations are represented in the frontispieces.

The **arch of the aorta** lies behind the sternum, its transverse portion crossing about an inch from the suprasternal fossa, that is, on a level with the second rib; on the right side it projects somewhat into the second space and under the second cartilage.

The **innominate,** the **left common carotid,** and **left subclavian** arteries arise from the transverse portion of the arch; the first passing upwards to the back of the right sterno-clavicular articulation, and the second to that of the left.

The **superior vena cava** lies to the right side of the arch, opposite the inner ends of the first and second cartilages and intercostal spaces.

The **left innominate vein** is in contact with the upper

part of the aortic arch, its superior limit being nearly on a level with the upper edge of the sternum.

The **pulmonary artery** lies behind the sternal end of the second left intercostal space for about an inch.

The **left pneumogastric, phrenic,** and **superficial cardiac nerves** lie in front of the transverse portion of the aortic arch, and the left *recurrent laryngeal* turns up under and behind it.

Posteriorly the aorta reaches the spine to the left of the body of the fourth dorsal vertebra, and at the lower part of the body of the fifth it becomes the descending thoracic aorta.

Physical Examination of the Circulatory System

Inspection

Inspection of the circulatory system is best carried out with the patient in a semiprone position, the chest being well exposed and in a good light. The præcordia (the region of the chest which comes into relation with the heart), the epigastric region, the upper part of the sternum, the supraclavicular and suprasternal regions are in turn inspected for any alteration in shape or size, and for pulsations, normal or abnormal.

Form of the præcordia.—Apart from the common alterations in shape, such as the depression at the lower end of the sternum seen in shoemakers, which have no direct pathological bearing upon the condition of the circulatory system, certain important alterations in form are met with.

Abnormal bulging occurs when the heart is much enlarged, especially when the enlargement has come on during early life, when the chest wall is soft and yielding. When there is effusion into the pericardial sac the præcordia may show considerable fulness, the fulness being most marked in the intercostal spaces.

Depression or retraction of the præcordia is sometimes

seen. In adherent pericardium there is a retraction of the interspaces surrounding the apex-beat and of the left epigastric region. The retraction is not at the apex, as so frequently stated, but in the interspaces around. This condition must be distinguished from the yielding of the interspaces about the left border of the heart so frequently seen in cases of cardiac enlargement.

Præcordial pulsation.—On inspecting the normal præcordia, a shock or impulse will, as a rule, be noticed at each beat of the heart, situated in the fifth interspace, $\frac{1}{2}$ in. to $\frac{3}{4}$ in. internal to the mammary line, that is, about 3 in. from the midsternal line. This impulse is known as the "apex-beat," and is normally produced by the impulse of the left ventricle. The term "apex-beat" has been very loosely applied to the point of maximum cardiac pulsation. It should be restricted to the lowest and most external point where cardiac pulsation is visible. The normal cardiac impulse then is situated in the fifth interspace, its extent in a vertical direction is a single interspace, in a lateral direction about an inch; its character is a gentle pulsation, its rhythm is regular, and its time in the cardiac cycle systolic.

Displacement of the apex-beat may take place from various causes. In pathological conditions the exact position of the pulsation should be accurately defined. Any pathological condition within the thorax or abdomen, which produces displacement of the heart as a whole, will cause dislocation of the apex-beat. Thus the apex-beat will be displaced *downwards* in aneurysm of the aorta when the aneurysmal tumour is large, and in emphysema of the lung. An abdominal tumour, tympanitis or ascites, will cause an *upward* displacement, and a similar displacement may take place in retraction of the right lung from chronic fibroid changes. In effusion into the pleura or pneumothorax the heart may be displaced to one or other *side*. When there is marked effusion into the right pleura the heart is carried to

the left; when the effusion is into the left pleura, or when there is retraction of the right lung, the heart, and as a consequence the apex-beat, is displaced to the right, and pulsation may be visible to the right of the sternum. By far the most common causes of displacement of the apex-beat are to be found in pathological changes in the heart and pericardium. In hypertrophy and dilatation of the left ventricle the apex-beat is displaced downwards and outwards to a very marked degree. In hypertrophy and dilatation of the right ventricle the breadth of the heart is increased and the apex-beat is displaced outwards, without such marked downward displacement as in left-sided hypertrophy and dilatation.

In congenital transposition of the viscera the apex-beat, instead of being on the left side of the thorax, is found on the right.

Extra-apical Pulsations. — Besides the apex-beat other pulsations may be observed on inspecting the præcordia.

Pulsation of the right ventricle is frequently present. It is seen in the third, fourth, and fifth left spaces, in the last of these extending outwards to within an inch or so of the apex. This results from thinness of the chest walls, or is caused by dilatation of the right ventricle, which may follow upon mitral stenosis or regurgitation, or may be due to obstruction to the pulmonary circulation, as in emphysema; but it also occurs in conditions of debility and anæmia. Emphysema is the least common cause of pulsation in the interspaces mentioned, as in it the heart becomes covered by lung, and in addition is displaced downwards, so that the pulsation is more commonly epigastric.

Pulsation in the second left space is not infrequently present. It may be caused by a distended pulmonary artery, at other times by a dilated conus arteriosus of the right ventricle, or by retraction of the lung from over the pulmonary artery, as in chronic fibroid phthisis.

Extent of the Pulsation.—The præcordial pulsation may be confined to a single interspace, as in the normal condition, or may be diffused over a wide area, as in cases of ventricular hypertrophy. It may be taken as a general rule that if the heart be in its normal position, pulsation extending to or beyond the mammary line indicates hypertrophy or dilatation. In cases where the chest wall is very thin or the left lung retracted from fibroid change, the pulsation may be very diffused, but if the heart be normal in size the pulsation will not extend beyond a point slightly internal to the mammary line.

Character and rhythm of pulsation.—The character of præcordial pulsation varies considerably. Besides variations in force, there is an undulatory or wave-like pulsation, which may be present in pericardial effusion or in dilatation of the right ventricle, if the ventricle is in contact with the parietes.

The rhythm of pulsations at different points can be satisfactorily demonstrated by placing small flags on the chest wall. It can then be seen whether the pulsations are synchronous with one another, or what their precise relations are in point of time.

Negative Evidence from Inspection.—By this we mean the absence of positive evidence, as the absence of bulging, or of pulsation. The importance of such negative evidence must not be under-estimated. When no impulse is visible it should so be stated. Negative evidence is not, however, necessarily of much importance, and this is particularly so in muscular and fat people, where, in consequence of the thickness of the parietes, there may be no præcordial pulsation visible.

Extra-cardiac Regions.—Pulsation in the **epigastric region** is common, and does not necessarily indicate disease. It is seen pathologically in displacement downwards of the heart, and in dilatation and hypertrophy of the right ventricle. Epigastric pulsation is not necessarily ventricular. It may

be seen in tricuspid regurgitation from pulsation in the dilated hepatic veins. Any tumour in the epigastric region, such as a pyloric carcinoma, may show communicated pulsation from the abdominal aorta. In some conditions the abdominal aorta itself may be seen pulsating a little below the xiphisternum.

Pulsation, systolic in time, may be visible at the upper **part of the sternum** and **episternal notch** in cases of aneurysmal dilatation of the aorta. In the **supraclavicular** regions considerable information may be got from the condition of the vessels, arterial and venous, their fulness or otherwise, the presence or absence of pulsation, and, if present, its character; but these points may be better discussed under examination of the vessels.

Palpation of the Præcordia

The præcordia is palpated by placing the hand flat on the chest. By this means we feel the movements of the heart as they are transmitted through the parietes. In the normal condition the systolic impulse against the thoracic wall is felt at the apex, while at the base the shock of the closure of the semilunar valves can be felt when the thoracic wall is thin or the cardiac action excited. When the organ is feeble, as in myocarditis, failure from long standing, valvular disease, or nervous interference with its action, its impulse is feeble, and may only be felt by making the patient bend forwards; but the impulse may also be feeble from the presence of fluid or air in the pericardial sac, or from emphysema of the lungs, and even pneumothorax, pleurisy with effusion, and hydrothorax may diminish the force of the impulse; when the organ is *hypertrophied* the measured thud is very characteristic.

Pulsation is felt over a wide area when the heart is enlarged, and it usually corresponds with the area over which it is visible.

Both the systolic impulse and the vibrations set up by the closure of the semilunar valves are felt at the apex in many persons, more especially in those with thin chests, or who are clinically known as of a nervous temperament.

Irregularity in the time and force of the impulse occurs in many morbid conditions, more especially in mitral lesions, and in fatty, debilitated, and dilated hearts. It is also present in some nervous affections.

Thrills are sometimes to be felt over the præcordia, either at the apex or at the base. The sensation has been compared to the feeling experienced when the hand is placed on the back of a cat when it is purring; it has accordingly been called *frémissement cataire.* They are most commonly produced at the mitral, the aortic, or the pulmonary orifices. At the mitral orifice they may be systolic, or diastolic, or distinctly presystolic, and are felt at the apex: at the aorta or pulmonary artery they are almost invariably systolic, and are felt at the base. The physical causes of thrills can be best discussed along with the causes of cardiac murmurs, as the same physical causes which result in the production of a murmur will, if sufficiently marked, produce a thrill. For their production the blood has to flow with considerable force, and as a consequence of this they disappear if the nutrition of the cardiac muscle becomes much impaired, and reappear with its rehabilitation.

Cardiography.—The movements of the præcordia can be registered by means of the cardiograph. Tracings obtained by this graphic method show the sequence and duration of the cardiac movements; they give clear indications of such abnormal vibrations as those causing reduplication of the sounds of the heart, but afford less distinct evidence of the vibrations producing murmurs.

Percussion of the Heart

The percussion area of the heart is divided into superficial and deep, according as it is desired to delimit the part of the

organ uncovered by lung, or to find the precise size of the organ itself. The former is the *superficial* or *absolute dulness*, the latter the *deep* or *relative dulness*.

The **superficial dulness** corresponds with the part of the heart uncovered by lung, and lies within the notch in the left lung, and between it and the margin of the right lung. What has really to be done in defining the superficial dulness is to find the edge of the lungs. Percussion must be *very light*, as the edge of lung overlying the heart is very thin at its margin, and all trace of clearness in the sound must disappear before it is inferred that the margin of the lung is passed. Normally the superficial dulness extends from the fourth to the sixth left costal cartilage, and extends outwards to about the junction of the cartilages with the ribs. The part of the heart under the sternum which is uncovered by lung is not so readily defined, but can be made out by comparing the percussion sound on the sternum above and below it.

The **clinical value** of the superficial dulness is not great, and many clinicians have entirely abandoned its investigation. When, however, the heart is much enlarged, or when there is much pericardial effusion, the area of this dulness is extended as the organ approaches the chest wall and pushes the lung aside. On the other hand, when the volume of the lung is increased, as in emphysema, the area of dulness is diminished, for the edge of the left lung approaches nearer to the middle line and covers more of the heart. Occasionally confusion may arise from a tympanitic sound being obtained over the area of superficial cardiac dulness. This is especially apt to occur if too strong percussion be used. The tympanitic resonance of the sound is given by the air-containing stomach, lying behind the heart, and if the patient be otherwise normal has no pathological significance.

The **deep dulness** of the heart is of very great importance and value to the clinician, for by means of it he can

determine its exact size and position in the thorax. The percussion in this case must be strong, and it requires to be understood that the change in the percussion sound as we pass from lung only, to lung with heart beneath it, is at first very slight, indeed almost imperceptible to the untrained ear, although it becomes more distinct the nearer we approach the sternum, and as the depth of lung overlying the heart diminishes. Much help will be obtained in percussion of the heart by carefully noting the sense of resistance on percussion. Perhaps the greatest difficulty in the percussion of this organ is the acquirement of confidence in our observations, for the organ as it lies in the thorax is covered by a greater extent of thoracic wall than is quite appreciated by clinicians as a rule. It is also to be remembered that the large vessels above the base of the heart, and under the sternum and adjoining cartilages and spaces, act as a solid body to percussion. As a consequence of this, their dulness is continuous with the cardiac dulness, and we make a rule of defining both. The method to be used is as follows:—Begin to percuss, in the outer half of the first or second space on the left side, and percuss strongly, always moving inwards, until a slight alteration in the sound is perceived, and at that spot make a mark; the second, third and succeeding spaces are percussed in the same manner, always being mindful to begin far out, so as to get a pure lung sound to start from, and in each space a mark is made where dulness begins. If it is desired, a similar proceeding may be followed with the ribs, but as a matter of fact it is superfluous, for if we have a mark in each space (a dot is sufficient) all that is necessary is to join them by means of a line, and thereby a very accurate outline of the left edge of the heart, as it lies in the pericardium, and of the vessels at its base, is obtained. In this way the true apex of the heart is found, a point of importance when auscultation is followed, and which is to be distinguished from the præcordial pulsation in other positions, often erroneously called the apex-beat. A like proceeding is adopted to define

the right edge of the vessels at the base and the right edge of the heart. With reference to the latter, however, it is to be remembered that the depth of lung between the parietes and the part of the heart to the right of the sternum is considerable, and that the difference in sound when we pass from lung, to lung with underlying heart, is correspondingly

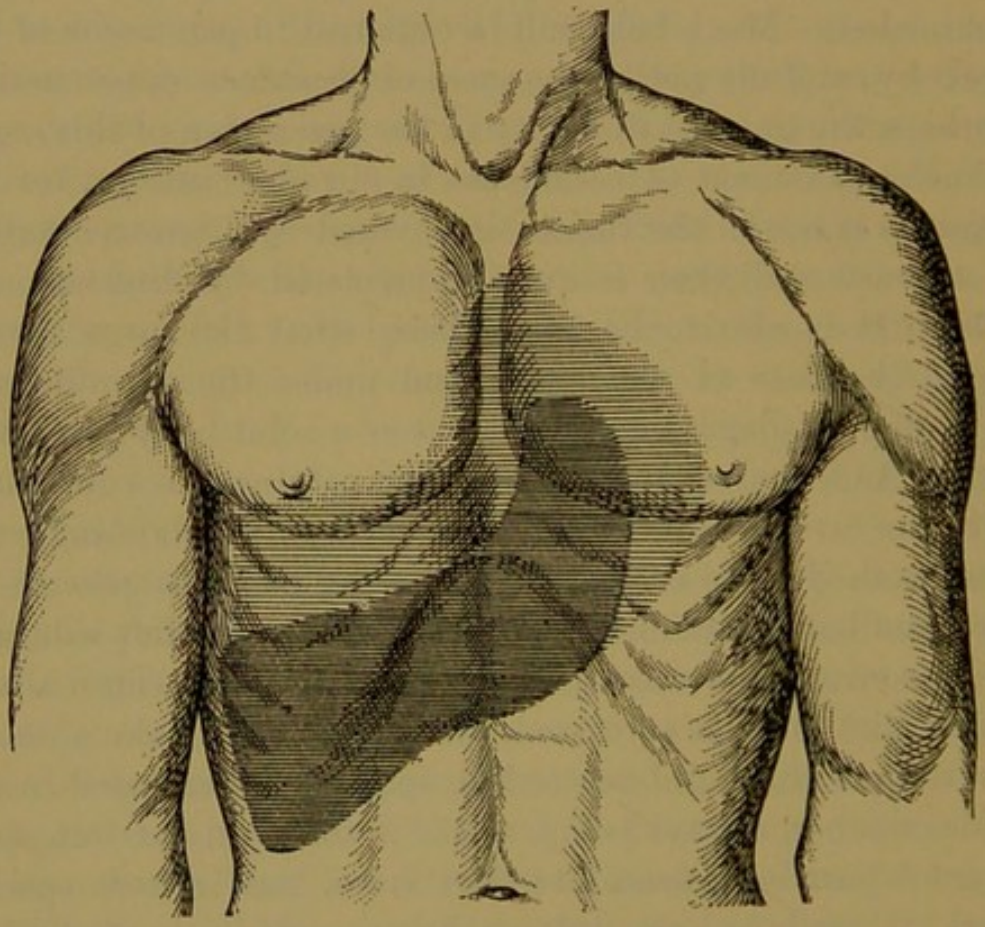


FIG. 12.—Showing percussion area of heart and liver. Deep shading, the area of absolute dulness; light shading, the area of deep or relative dulness.

slight. Lastly, there is to be considered the delimitation of the lower border of the organ. If the right and left borders have been completed, a line drawn to join their inferior limits gives this border with sufficient accuracy for all practical purposes. The delimitation of the border by percussion is usually regarded as impracticable owing to the organ being in such close apposition to the liver. This is,

however, by no means invariably the case, for there can often be made out a distinct difference in the percussion sound and sense of resistance over the two organs, which, by observations verified on the dead body, is found to accurately indicate their respective limits.

The preceding diagram (Fig. 12) represents the superficial and deep dulness obtained by percussion after the method given above.

The area of cardiac dulness is increased in all conditions where the organ is enlarged. There is marked increase in the breadth and in the vertical extent of the dulness in hypertrophy and dilatation, especially of the left side of the heart. In such affections of the right side of the heart the transverse extent of the dulness is increased, the vertical not being so much altered. In effusion into the pericardium, as seen in pericarditis, the increase in the cardiac dulness is in every direction the outline of the dulness taking the form of the pericardium. The outline thus formed is somewhat pear-shaped, the narrow upper portion of the dulness forming the stalk of the pear. This characteristic outline may, of course, be modified if pericardial adhesions be present.

The position of cardiac dulness is of course altered if there is any alteration in the position of the organ, as when it is displaced to the right side, or downwards and outwards, or downwards only, or upwards. These displacements have been referred to under inspection and palpation. It is unnecessary to dwell in detail on the position of the dulness in all these, but the displacements must be kept in mind, and accuracy in determining their limits will be attained by practice.

Auscultation of the Heart

When the stethoscope is placed on the præcordia the sounds produced by the heart are heard. They are two in number, and are commonly represented by the two monosyllables *lŭp-dŭp.*

The first sound corresponds with ventricular systole, and for purposes of physical diagnosis may be regarded as mainly caused by the closure and tension of the auriculo-ventricular valves, that is, the mitral and tricuspid, and only in an unimportant degree by the sonorous vibration emitted by the contracting muscular substance. At the same time it is to be understood that the strength of the muscle determines

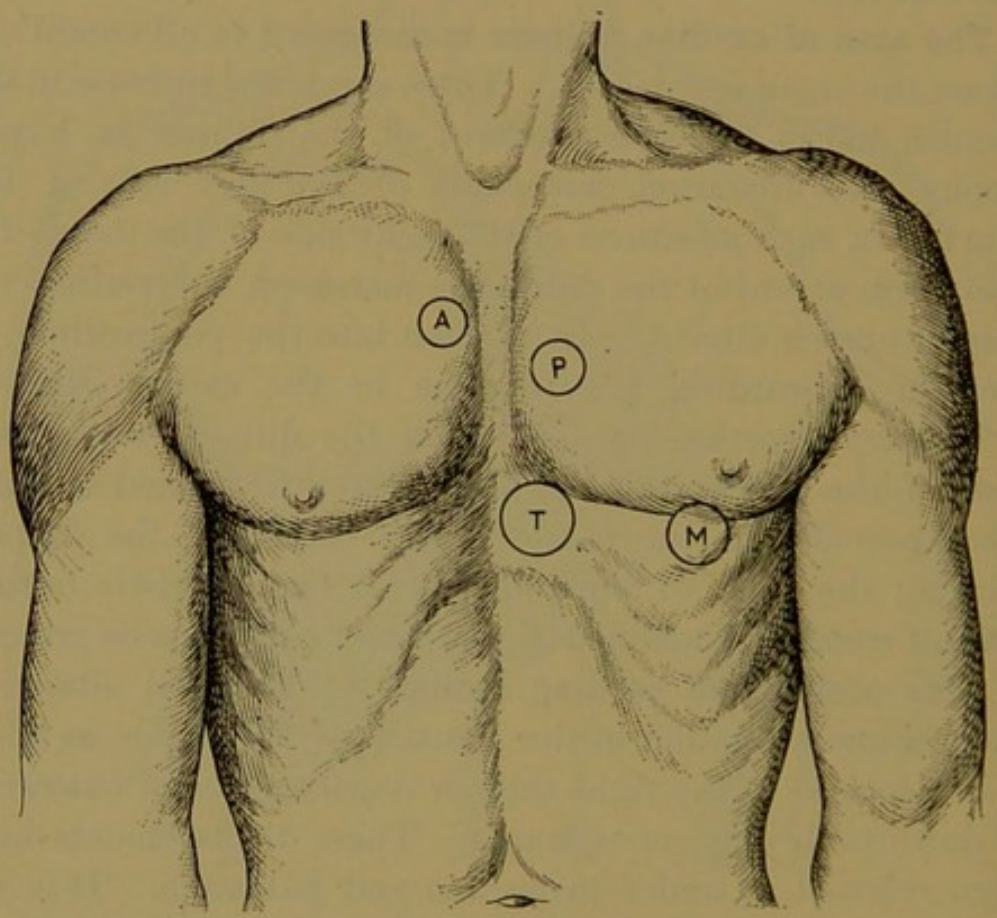


FIG. 13.—Showing præcordial areas. *A*. Aortic area; *P*. Pulmonary area; *T*. Tricuspid area; *M*. Mitral area.

the vigour of the contraction, and therefore the vigour with which the auriculo-ventricular valves close.

The second sound is caused by the closure and tension of the semilunar valves (the aortic and pulmonary), and marks the *beginning* of ventricular diastole.

The first or systolic sound is heard best at the apex and over the right ventricle, and is less distinctly audible at the base.

The second or diastolic sound is, on the other hand, best heard over the base of the heart; the aortic second sound being heard best in the second right space, while the pulmonary second sound is heard best in the third space on the left side. The second sound is normally of greater intensity and higher in pitch in the pulmonary than in the aortic area.

For convenience in describing auscultatory signs, the præcordia is divided into *four areas*, called respectively the mitral, tricuspid, aortic, and pulmonary. The *mitral area* is represented in Fig. 13 as occupying an area about 2 in. in diameter, the centre being the apex. The *tricuspid area* embraces the area from the third to the fifth intercostal spaces on the left side and the adjoining sternum, the centre of which is shown in the figure. The *aortic area* is usually given as the inner end of the second right cartilage or second space; in the figure it has been placed in the former situation. The *pulmonary area* is the inner end of the third left intercostal space.

The relative intensity of the two sounds at the apex, over the right ventricle midway between the apex and the edge of the sternum, over the lower half of the sternum, and in the aortic and pulmonary areas, ought to be carefully studied, for, although there is considerable variation in different healthy individuals, valuable indications are sometimes obtained from such observations in abnormal conditions.

On careful examination in the healthy subject it will be found that the mitral first sound is lower in pitch and somewhat longer in duration than the tricuspid, and that the pulmonary second sound is of greater intensity and higher in pitch than the aortic.

Quantitative alterations in the cardiac sounds may affect either the first or second sound, and occur under varying conditions.

Diminution in the intensity of the First Sound as

heard at the apex occurs in all conditions in which the strength of the left ventricle is impaired. This is often notably the case in febrile diseases; to a certain extent in cachectic and wasting diseases, although not to the same extent as in febrile diseases, in anæmia and debility, from various causes; and in fatty heart, whether due to fatty degeneration of the muscle of the ventricle or to deposit of fat on the surface and between the muscular fibres. In fat persons, especially women, the sounds may be faint from the thickness of the chest wall overlying the heart, but in persons of this type the strength of the organ is also not infrequently impaired.

It is of the utmost practical importance to be able to estimate the power of the left ventricle by auscultation, and the student ought to avail himself of every opportunity of listening to hearts and analysing the loudness of the sounds at the various points already indicated. To form a correct estimate, it is necessary to auscultate over the *apex*,—not the apex-beat necessarily, for the "apex-beat" is frequently caused by the right ventricle. The position of the true apex, when there is doubt, must be determined by percussion. If, instead

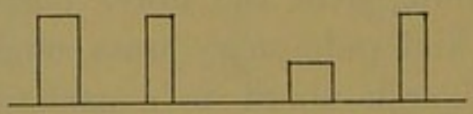


FIG. 14.

Normal first and second sounds. Diminished first sound.

of auscultating at the true apex, we do so over the part of the chest which covers the right ventricle, the first sound may be loud, although the muscle of the left ventricle is degenerated; this is because the sound produced by the right side of the heart is as a matter of course heard best immediately over it, and because the muscle of the right ventricle may be comparatively strong, although the left is weak. The state of the pulse, as regards both frequency and strength, depends mainly on the frequency and vigour of the contractions of the

left ventricle, and it ought always to be examined before a definite opinion is formed as to the condition of the ventricle. The neglect of these precautions leads to many errors. Further, it must not be forgotten that temporary excitement, such as the excitement caused in the majority of persons by being subjected to physical examination, stimulates the heart to increased vigour, and therefore to a louder first sound and a stronger pulse. If, therefore, an opinion be formed by a rapid examination under such circumstances, it will most probably be incorrect. Even when the first sound is so weak as to be inaudible at the apex, the second sound may continue to be distinctly audible there, and may give the erroneous impression of being accentuated. When the first sound is thus inaudible, all that is heard at the apex is of course the *dŭp-dŭp-dŭp* of the second sound, with a longer or shorter silence in between. Instead of the somewhat grave tone of the second sound, it often becomes clicking in character.

Increase in the intensity of the First Sound.—This may present two varieties. In one form the sound is

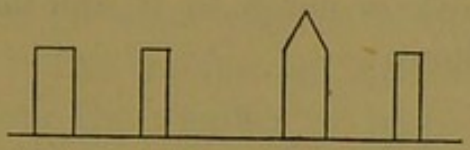


FIG. 15.

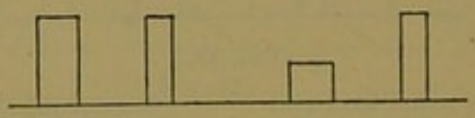
Normal first and second sounds. Accentuated first sound.

long and grave (as distinguished from short and sharp), a like impression being conveyed to the mind of the observer by the character of the impulse communicated through the stethoscope to the head. When the sound has this character, it is followed by an abnormally loud second sound. It occurs when the left ventricle is hypertrophied and its muscle well nourished. It is present most typically in cases of chronic interstitial nephritis, and in some cases of aortic stenosis and aortic aneurysm.

In the second variety the first sound is short and sharp, and high in pitch, and may be met with when there is some dilatation of the left ventricle, the muscle contracting shortly and sharply with the irritability of weakness. It may occur in persons with healthy hearts who have thin chests, and from comparatively unimportant causes, as mental emotion or slight excitement, flatulence or other symptom of indigestion, or a little extra physical exertion. The individuals who are classed as "neurotic" frequently present this peculiarity of the cardiac sounds. It also occurs in febriculæ, and may be present at the onset of any acute febrile disease.

Modifications in the tone of the First Sound.—The first sound is liable to various modifications in tone, some of which may be noted. It may be short and sharp, long and grave, flopping, or impure. The two last are not infrequently mistaken for murmurs, and sometimes indicate a condition in which murmur may be developed later or on exertion.

Alterations in Rhythm—Reduplication of the First Sound.—This is best understood by considering it as the first sound with a break or notch in it, and may be represented graphically as follows:—



A. *B.*

FIG. 16.

Normal first and second sounds. Reduplicated first sound.

It can never be a true doubling; there can never be too distinct individual first sounds heard, for the time occupied would be almost the entire cardiac systole. The doubling is apparent rather than real, and results from an asynchronism of the maximum intensity of the right and left first sounds. There is some difficulty in absolutely determining the cause of the phenomenon, but the two main views entertained as

to its mode of production are—(1) That it is caused by non-synchronous closure of the mitral and tricuspid valves; (2) that it is due to non-synchronous tension of the cusps of the same valves. It occurs in lesions of the auriculo-ventricular valves, usually when compensation has failed. It is well, however, to note that the phenomenon is rare. Frequently what was at first considered to be a reduplicated first sound resolves eventually into a presystolic murmur preceding a single sound.

Irregularity presents much variety both in time and strength. No two successive sounds may correspond, or there may be a few intense sounds followed by a number of weak ones. It is a marked feature in many cases of mitral lesion, of fatty heart, and in some cases of debility, especially during or following acute illness.

Intermittence is the missing of a contraction, and therefore of the sound, by a heart which is otherwise beating regularly. It is to be distinguished from irregularity. Its cause is not always apparent, but it may occur in dyspepsia in gouty persons, while at other times it appears to be a neurosis, and in some persons is habitual and of no practical bearing. The number of intermissions should be noted in relation to the number of beats as 1 in 10, 12, or whatever the number may be.

Diminution in the intensity of the Second Sound occurs in all conditions where there is diminished blood pressure in the aorta or pulmonary artery. The diminished blood pressure may result from a deficient propulsive power on the part of the ventricle, deficient access of blood to the ventricle, so that a diminished amount is propelled into the vessel at each systole, or to lessened resistance in the peripheral arteries. The aortic second sound is diminished in such conditions as mitral obstruction, mitral regurgitation or lung disease, a diminished volume of blood being under those circumstances propelled into the aorta. It is also diminished in intensity in affections which weaken the

power of the left ventricle, as pyrexia, myocarditis, fatty degeneration. Again, when the arterioles are relaxed, the pressure in the systemic system is lowered, and the aortic second sound is thus diminished in intensity. Diminished intensity of the pulmonary second sound is by no means common. Theoretically it should be present in tricuspid incompetence, but as a rule its occurrence is difficult to determine with certainty. When present it is usually due to weakness on the part of the right ventricle, and is of grave import.

Accentuation of the Second Sound may have its seat at either of the arterial orifices.

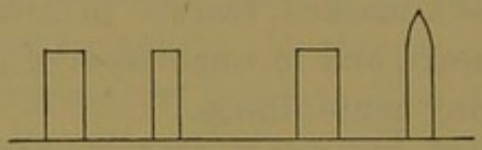


FIG. 17.

Normal first and second sounds. Accentuated second sound.

Accentuation of the pulmonary second sound results from obstruction to and consequent heightening of the blood-pressure in the pulmonary circulation, whether due to valvular lesion at the mitral orifice, or to lesions of the lungs themselves, such as emphysema, etc., which obstruct the capillary circulation.

Accentuation of the aortic second sound depends on increased arterial pressure, and is a concomitant of chronic renal disease, more especially in its cirrhotic forms; it also occurs in atheroma, dilatation, and aneurysm of the aorta.

Modifications in the tone of the Second Sound.—In addition to those already referred to, the second sound has in some cases a flopping character, which indicates a want of tonicity or of tension in the vessel, usually the result of a weak ventricle with the systemic arteries healthy. It is frequently present in febrile conditions.

Alterations in Rhythm—Reduplication of the Second Sound.—This may, like reduplication of the first sound, be regarded as the second sound with a break or notch in it, and is graphically represented as follows :—

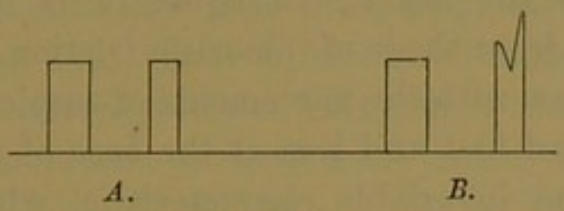


FIG. 18.

Normal first and second sounds. Reduplicated and accentuated second sound.

It may, however, be much more marked than this, the second part being distinctly separated from the first, although immediately following it.

It is caused by non-synchronous closure of the aortic and pulmonary valves, and results from alteration in the relative tensions of the aortic and pulmonary arteries. It occurs most commonly in cases where the tension in the pulmonary artery is greatly increased, and where at the same time there has been a proportional lowering of that in the aorta. It is best heard over the sternum at the level of the pulmonary and aortic orifices, but can often be heard over the entire præcordia.

A peculiar apparent reduplication of the second may be heard at the apex in many cases of mitral stenosis, while the second sound at the base is accentuated but not reduplicated. The second element in this apparent reduplication is probably produced at the mitral orifice, and is in most cases in reality a diastolic murmur.

Irregularity and intermittence accompany similar alterations in the first sound.

Cardiac Murmurs.—Cardiac murmurs or bruits are abnormal sounds present in morbid conditions of the heart. They are divided into exocardial and endocardial.

The commonest exocardial morbid sound is **pericardial**

friction, the result of pericarditis. This sound is synchronous with any or all of the cardiac phases, and is rubbing, grating, rasping, or creaking in character. It is distinguished from the friction of pleurisy by being synchronous with the movements of the heart; consequently its rhythm and frequency differ from those of pleuritic friction, in which they are synchronous with the movements of respiration. Friction is usually heard first and best at the base of the heart. The sound has one invariable characteristic, which is of great diagnostic importance—it invariably conveys the impression of being superficial in origin. Unless under exceptional circumstances the sound is propagated to a very limited extent; when the stethoscope passes off the region of the chest, in immediate relation with the heart, the sound is lost. Exception to this rule is found when a portion of consolidated lung lies in contiguity with the pericardium. When the chest wall is thin, the friction sound can be made longer and rougher by pressing firmly with the stethoscope; it must be remembered, however, that this procedure is liable to cause pain.

In persons with thin chest walls and a dilated right ventricle, the pulsations of which are well marked in the interspaces to the left of the sternum, a rubbing sound resembling friction can occasionally be produced by pressing firmly with the stethoscope in an intercostal space. This must not be confused with friction the result of pericardial inflammation.

Pleuro-pericardial friction is the term used to denote the sound produced by the rubbing of the inflamed pleural surface of the pericardium against the inflamed visceral pleura in contact with it. The friction in this condition is, as a rule, heard only during inspiration, and when it accompanies the cardiac systole; it usually disappears with expiration, but it may continue to be *faintly* audible even then with each cardiac systole. Even in the latter case, however, it becomes much louder during inspiration.

If air and fluid be present in the pericardial sac, splashing and churning sounds, metallic in quality, are produced, but this is rarely met with, and usually results from puncture from without.

When, as a further result of the pericardial inflammation, serum is exuded into the sac, friction disappears owing to the two lymph-covered surfaces being kept asunder. When the fluid is reabsorbed or artificially removed the friction reappears.

Endocardial Murmurs.—The movement of the blood in the heart and vessels, under pathological conditions, may give rise to abnormal acoustic phenomena as distinguished from the normal sounds. These abnormal sounds are known as murmurs. Murmurs result primarily from vibrations in the blood, which vibrations being conducted through the heart wall and parietes are audible. When the vibrations are perceptible to the hand placed on the chest wall they are recognised as *thrills*. If fluid when circulating in a tube pass through a narrow into a wider portion of the tube beyond, eddies or fluid veins are set up, and the vibrations thus caused in the fluid result in a murmur. The same conditions are found in the circulatory system. When the blood passes through a small opening into a wide chamber beyond, a fluid vein is set up, causing vibrations and hence a murmur. When the blood passes through a portion of the circulation where the lumen is somewhat narrowed at one point, eddies are originated, which result in vibrations and a murmur. It must always be remembered that a certain pressure is necessary in the blood stream, before an audible murmur can result. It is frequently stated that an alteration in the characters of the blood has an important influence in the causation of a murmur. This, however, is more than doubtful. The influence of the blood is in all probability only indirect, that is, only through its influence on the heart wall.

When an endocardial murmur is present, it is of the first

importance to determine at which valve orifice it is produced, and the lesion on which it depends. In order to arrive at a definite conclusion it is necessary to determine—(1) The time of the murmur in the cardiac cycle; (2) its point of maximum intensity; (3) its sound character; (4) its direction of propagation.

Time of Murmurs.—For clinical purposes we may regard a cardiac revolution as consisting of an auricular systole, a ventricular systole, and a ventricular diastole; and all endocardial murmurs occur during one or other of these.

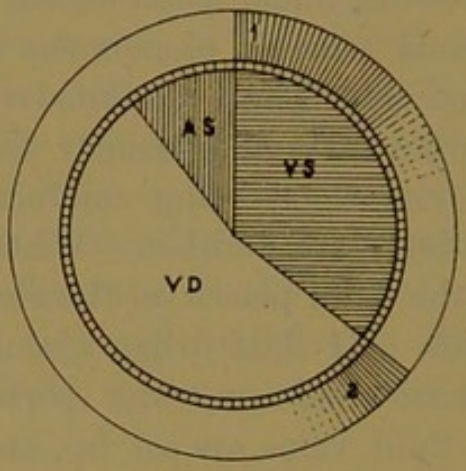


FIG. 19.—Diagrammatic representation of the cardiac cycle (after Gairdner). The inner circle shows the movements of the heart: AS=auricular systole, VS=ventricular systole, and VD=ventricular diastole. The outer circle shows the normal sounds of the heart: 1=first sound, and 2=second sound. The narrow circle between them is divided into hundredths of seconds in order to have the opportunity of giving an accurate representation of the actual duration of each phase.

Murmurs may be produced at the auriculo-ventricular orifices, mitral or tricuspid, at any one of those times, but at the arterial orifices, aortic and pulmonary, murmurs can only arise during ventricular systole and ventricular diastole. Murmurs are *timed* either by placing the hand over the apex-beat, or the finger on the common carotid at the same time as the ear is applied to the stethoscope. The latter is the preferable method. The radial pulse must not be taken, as it is appreciably later in time than the first sound.

Murmurs occurring during the systole of the auricles are, as will be seen below, termed presystolic, because they precede the ventricular systole, and precede therefore the first sound.

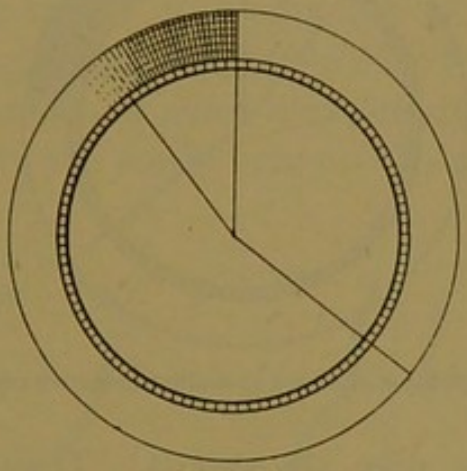


FIG. 20.—Presystolic murmur. In this and the five similar diagrams a murmur is distinguished from a sound by the double shading.

Murmurs occurring during the systole of the ventricles are termed systolic, and accompany or replace the first sound.

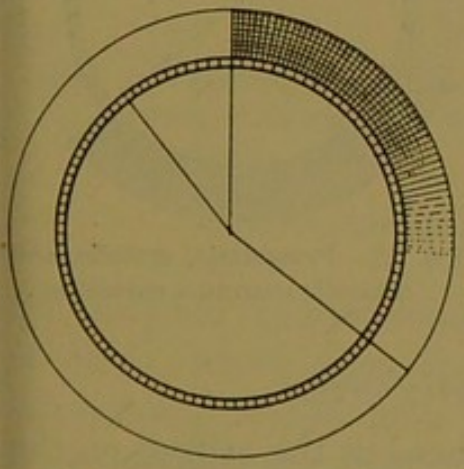


FIG. 21.—Systolic murmur.

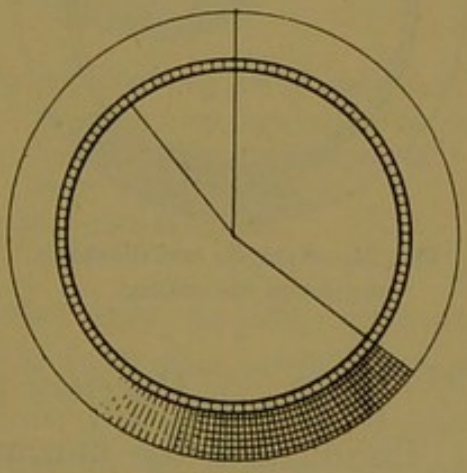


FIG. 22.—Diastolic murmur.

Murmurs occurring during the diastole of the ventricles are termed diastolic. They accompany, replace, or follow the second sound.

Murmurs occurring during different phases are often found

in association in the same case. Presystolic and systolic, systolic and diastolic, or even presystolic, systolic, and

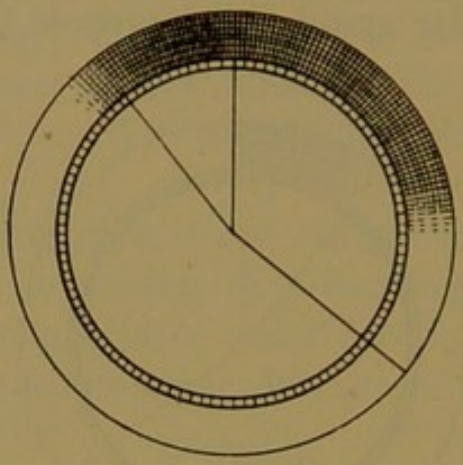


Fig. 23.—Presystolic and systolic murmurs coexisting.

diastolic murmurs may all coexist. They are represented graphically in the accompanying figures.

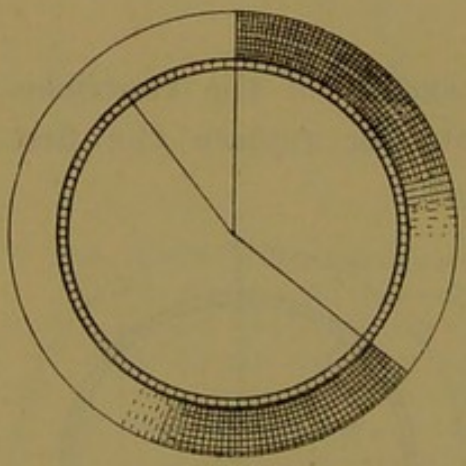


Fig. 24.—Systolic and diastolic murmurs coexisting.

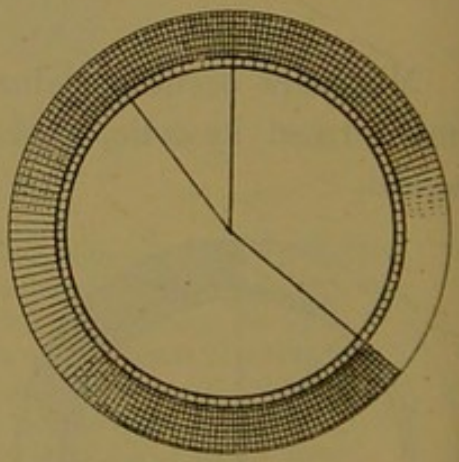


Fig. 25.—Presystolic, systolic, and diastolic murmurs coexisting.

Murmurs at the Aortic Orifice

Taking first the murmurs produced at the orifices on the left side of the heart, we begin with those at the aortic orifice.

Aortic systolic murmur is synchronous with the systole of the left ventricle, and therefore coincides in time with the apex-beat and the carotid pulse. It may be caused—(1) By partial adhesion of the cusps to one another; (2) by

thickening and roughening of the cusps, or calcareous deposit at their junction with the endocardium; (3) not infrequently by a calcareous mass on the endocardium between the base of the anterior cusp of the mitral and the base of the aortic cusps, the region known to anatomists as the intervalvular space of Sibson; (4) by a roughened cusp the result of injury; and (5) by aneurysm, or even a slight degree of dilatation of the aorta above the aortic valves. In the first four cases

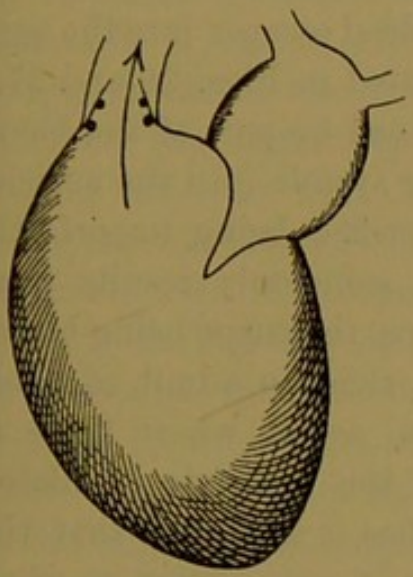


FIG. 26.—Showing aortic systolic murmur from stenosis. Mitral valve closed.

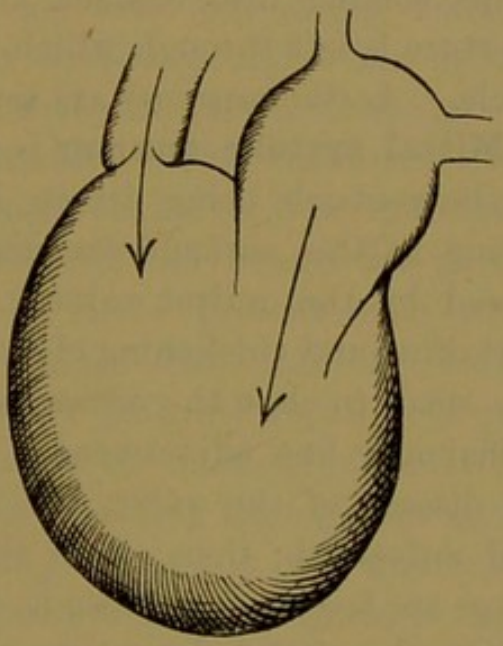


FIG. 27.—Showing aortic diastolic murmur from incompetence. Ventricle filling from auricle at same time.

The arrows indicate the direction of the blood stream.

the murmur is the result of obstruction to the current of blood driven from the ventricle into the aorta during its systole; in the fifth it is due to the dilatation of the aorta producing a condition analogous to obstruction at the orifice of the artery, and which may be referred to as relative stenosis. In the one case the blood passes through an abnormally small orifice into a normal part beyond, while in the other it passes through a normal orifice into an abnormally dilated part beyond. The latter condition is thus equivalent, from a physical standpoint, to the former.

Aortic diastolic murmur occurs during diastole of the ventricle. It is caused by the blood pouring from the aorta into the ventricle, as a result of the valves not closing the orifice as they normally do. The lesion of the valves which most commonly produces this murmur is shrinking and thickening, but perforations or rupture, and vegetations or calcareous nodules on them, also give rise to it. Large vegetations or calcareous nodules prevent the edges of the cusps coming into contact and overlapping, and thus an aperture is left through which the blood escapes into the ventricle. Aortic murmurs are represented in Figs. 26 and 27.

Mitral systolic murmur is produced by part of the blood in the ventricle being driven during systole into the auricle, owing to the auriculo-ventricular orifice being imperfectly closed by the mitral valve. This commonly results from shrinking and thickening of the cusps, the cusps being either too small to close the orifice, or too rigid to admit of their sufficiently fine adjustment. It also occurs where there is no disease of the valve, but when the ventricle is dilated and enfeebled; then either the orifice is dilated so that the cusps are too small to close it, or the muscle, owing to weak action, does not sufficiently contract the orifice to enable the valve to close it.

Mitral diastolic murmurs occur during the diastole of the ventricle, that is, when the blood is flowing from the auricle into the ventricle. They are caused by narrowing of the orifice through which the blood passes. The most common lesion producing the condition is adhesion of the anterior and posterior mitral cusps to each other along their lateral edges. By this adhesion the channel for the blood is necessarily much contracted. The valve is in addition thickened and more rigid than normal, and the aperture in it may be exceedingly small. These murmurs are subdivided into—

(*a*) Diastolic.

(*b*) Presystolic (or auriculo-systolic of Professor Gairdner).

(*a*) **The diastolic mitral** is unfortunately also called post-diastolic. This term is wrong, and here the murmur will be referred to by the former term. The murmur as a rule immediately follows the second sound; it may, however, accompany the second sound as well as follow it, but it does not occupy the whole time of ventricular diastole. The fact that the murmur follows the second sound led to its being called post-diastolic, but from what has been said it will be understood that it occurs not after but during diastole, although it occurs after the second sound; the second sound, it will be remembered, occupies only a short space of time at the beginning of diastole. The murmur occurs during the time that the blood ought to flow noiselessly from the auricle into the ventricle, and before the auricle contracts. It is comparatively rarely present even when there is marked narrowing of the blood channel: the reason of this is not quite evident, but it probably depends, in part at least, on the degree of vigour in the suction power of the ventricle. Normally the blood is passing through the mitral orifice during the long diastole, noiselessly and under low pressure. If, however, the pressure in the auricle be high and the suction power of the ventricle vigorous, the blood may pass through the stenosed mitral valve under sufficient pressure to produce a murmur during the long diastolic pause.

(*b*) **The presystolic mitral murmur** occurs, as its name implies, before the systole of the ventricle, and at that part of the cardiac cycle when the auricle contracts. It has therefore been called by Professor Gairdner *auriculo-systolic*; here, however, the term presystolic will be used. Its production at the particular moment is attributed to the greater energy with which the blood is flowing into the ventricle, owing to the contraction of the auricle.

Systolic and diastolic mitral murmurs are represented in the following diagrams.

Having considered the murmurs which may be present

on the left side of the heart, at the aortic and auriculo-ventricular orifices, it is only necessary to refer briefly to

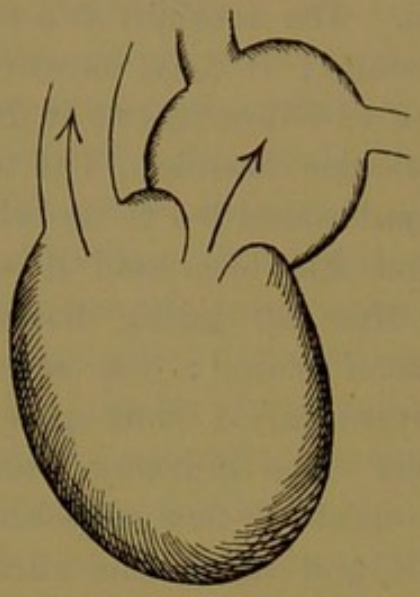


FIG. 28.—Showing mitral systolic or regurgitant murmur from failure of valve to close orifice. The blood current ought to be into the aorta only.

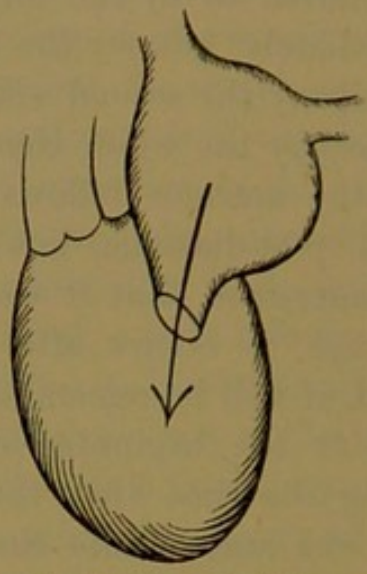


FIG. 29.—Showing diastolic or presystolic mitral murmur due to adhesion of mitral cusps.

The arrows indicate the direction of the blood stream.

those at the corresponding orifices on the right side. These are—

Pulmonary murmurs of organic origin are exceedingly rare; they are systolic and diastolic in time, and are due to lesions similar to those described as causing murmurs at the orifice of the aorta. When they do occur they are not infrequently congenital, and are associated with other lesions, such as patent ductus arteriosus, open foramen ovale, or imperfect closure of the ventricular septum. A pulmonary diastolic murmur sometimes results from persistent high pressure in the pulmonary artery leading to dilatation, secondary to a mitral lesion. The dilatation of the artery causes valvular insufficiency, and a regurgitant murmur results. What are known as *functional murmurs* in this artery are always systolic in time, but they will be referred to more fully hereafter.

Tricuspid murmurs are theoretically three in number

here, as at the corresponding orifice on the left side, but as a matter of fact diastolic and presystolic tricuspid murmurs are of extreme rarity, while systolic murmur is very common. When the two former are present, they are due to lesions similar to those producing corresponding murmurs at the mitral orifice,—that is, to stenosis. Systolic murmur is due to incomplete closure of the orifice, either from shrinking and thickening of the valves or from dilatation of the orifice: the latter is the more common, and is usually secondary to a mitral lesion.

The position of murmurs in the cardiac cycle may be studied in the diagrams on the previous pages. Fig. 19 represents the normal cycle, the first part of the circle representing auricular systole, the second ventricular systole, the third ventricular diastole, the closure of the semilunar valves being included in this last.

Point of Maximum Intensity and Direction of Propagation of Endocardial Murmurs.—The præcordia is divided, as has already been indicated, for purposes of auscultation, into four areas. These areas are respectively aortic, pulmonary, tricuspid, and mitral. They indicate the sites at which murmurs produced at the respective orifices are heard best. Although they are given here, it is better to follow the directions given in the following pages, and to trace more in detail the areas over which murmurs are heard.

The point where a murmur is heard loudest is known as *the point of maximum intensity*, and the several murmurs have each their distinctive point or seat of maximum intensity. The various murmurs also tend to be propagated in definite directions: this is known as *the direction of propagation*. These two can best be considered together. The general law as regards the propagation of murmurs is that they are propagated best in the direction of the current which produces them. They are also propagated by the sternum, by the walls of the heart, and by solid bodies or pulmonary cavities in proximity to the heart and aorta.

Aortic Systolic or Obstructive Murmur is usually described as having its point of maximum intensity on the second right rib or space, close to the sternum—the "aortic area," as it has been long called. This has led to much confusion and to many errors, and here we propose to depart slightly from the classical position. The murmur when present is always heard over the sternum at the level of the second cartilage, and this is the place where it is best to listen for it. The extent of area over which it is propagated depends mainly on its loudness. When very loud, as it often is, it is distinctly audible over every part of the thorax, both anteriorly and posteriorly; this has to be clearly borne in mind, for unless it is, the fact that it is audible at the apex, for instance, is often mistaken by the student as evidence of the existence of a mitral systolic murmur in addition to the aortic systolic. The murmur, however, is not invariably as loud as has just been indicated, and when this is the case it is conducted or propagated in more definite directions. As has already been said, it is heard over the sternum about the level of the second cartilage. It is propagated—(1) *Upwards* by the blood stream, and is, as a rule, heard as distinctly over the entire manubrium as it is at the level of the second rib. By the same means it is conducted into the carotids and heard in them; but, if the murmur be faint over the sternum, it may be inaudible over them. (2) *Downwards* (*a*) by the

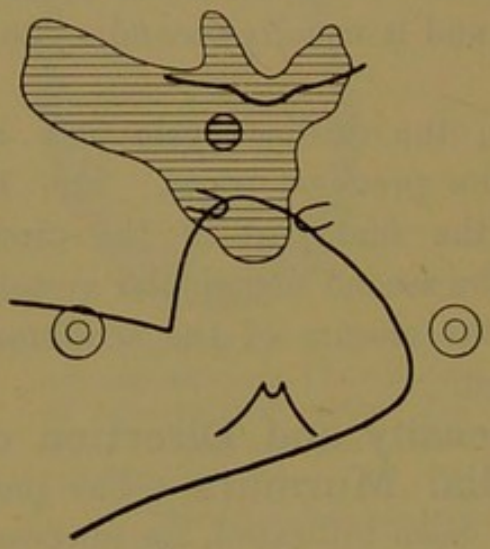


FIG. 30.—Area of audition of aortic systolic murmur.—Gibson.

In this figure and the succeeding, vertical lines represent presystolic, horizontal systolic and diagonal diastolic murmurs. The nipples, the upper margin of the sternum, the second rib, the costal angle and the area of cardiac and liver dulness are represented.

sternum, the bone acting as a good conductor of sound, but, unless the murmur be a very loud one, it diminishes markedly in intensity from about the level of the second space; and (*b*) by the heart itself, which acts as a conductor of the sound, so that it may be audible over the præcordia to the left of the sternum, as far out as the apex.

Aortic Diastolic Murmur or the Murmur of Aortic Incompetence.—The point of maximum intensity of this

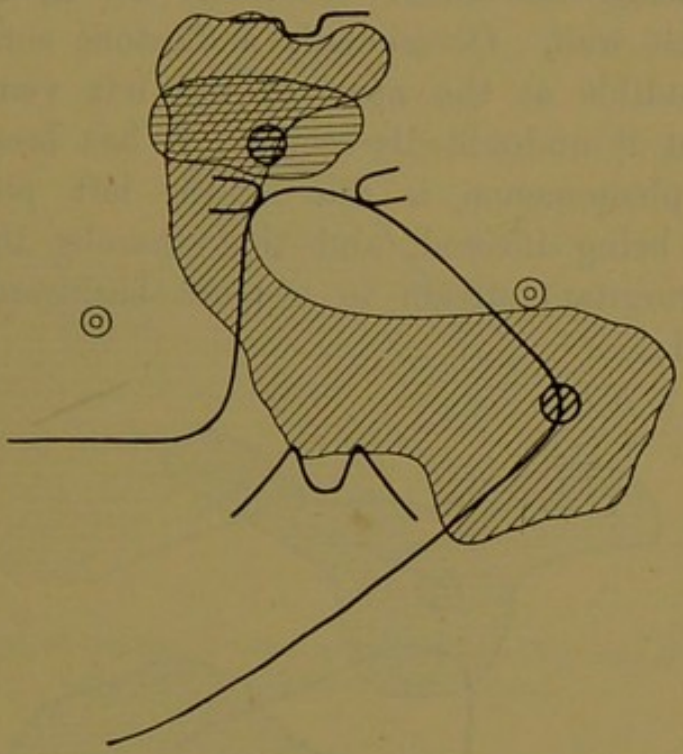


Fig. 31.—Area of audition of aortic diastolic murmur.—Gibson.

murmur is over the sternum, the precise point varying in individual cases. The most common points, however, are —(*a*) The sternum at the level of the second cartilage or second space; and (*b*) the lower part of the sternum. The murmur is as a rule, however, heard all over the sternum, but especially from the level of the second cartilage downwards, while it is usually not nearly as loud over the manubrium: this it will be noted is the reverse of what happens in aortic systolic murmur. When it is heard over the lower part of the sternum, it frequently attains its

maximum loudness at or near the base of the xiphoid. In addition to being propagated down the sternum, the murmur may be propagated by the walls of the heart, and heard over the præcordia to the left of the sternum. It may, however, be louder over the apex of the left ventricle than at any point between the apex and the edge of the sternum. This is explained by the regurgitant stream carrying the sound with it, and, impinging on the wall of the ventricle, communicating its sound vibrations to it, and thence to the chest wall. Occasionally a diastolic aortic murmur is only audible at the apex of the left ventricle: this is rare, but it undoubtedly occurs. It has been suggested that the phenomenon is due to the left postero-lateral cusp only being affected, and that thereby the direction of the regurgitant stream to a more backward course is determined.

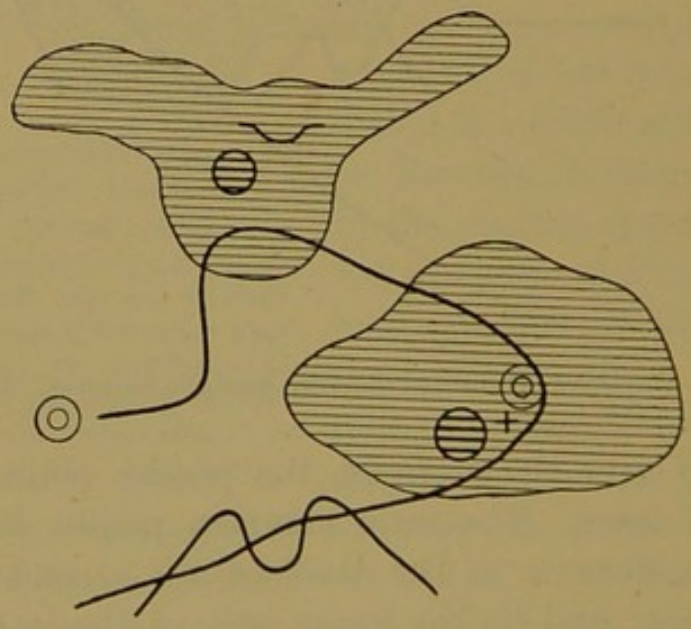


FIG. 32.—Area of audition of aortic and mitral systolic murmurs. —Gibson.

Mitral Systolic Murmur or Mitral Regurgitant Murmur.—This murmur has its point of maximum intensity at the apex of the heart. The area of its audition may be the size of a crown-piece, or even smaller; when, however, it is

moderately loud, it tends to be propagated outwards and upwards towards the axilla, and may even be traced as far as the angle of the left scapula. The extent to which it is propagated in this direction depends not only on its loudness, but also on the degree of enlargement of the ventricle, and probably on the transverse capacity of the thorax; the larger the heart and the smaller the transverse capacity of the thorax, the more nearly does the heart approach the chest wall in the infra-axillary region, and

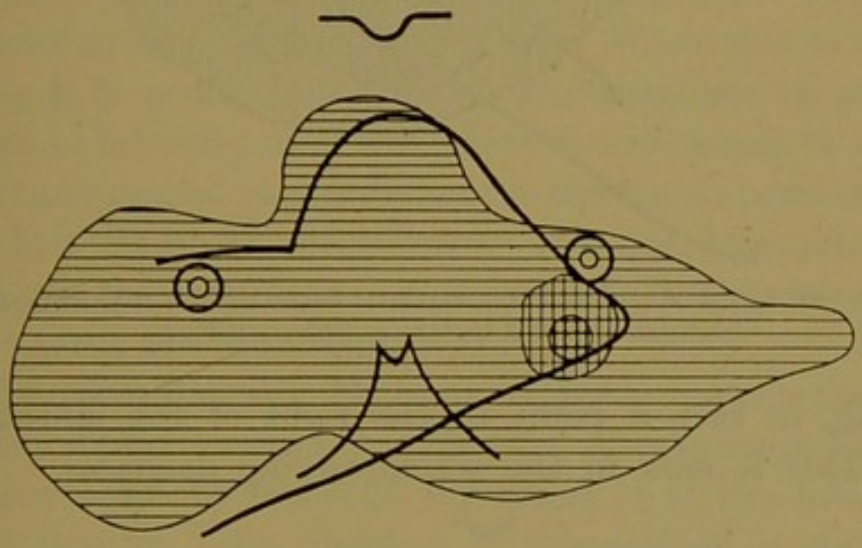


FIG. 33.—Area of audition of mitral systolic greater than area of audition of mitral diastolic murmur.—Gibson.

consequently the greater the area over which the murmur is audible.

The extent of the præcordial area of audition of the mitral systolic murmur depends upon the loudness of the murmur. A loud mitral systolic murmur may be audible all over the præcordial region.

Mitral Diastolic and Mitral Presystolic Murmur.—It is usually taught that both these murmurs have their point of maximum intensity a little above and internal to the apex, and are strictly localised, and that on passing from the region of the apex the murmur rapidly becomes inaudible. This, however, is a statement to be accepted with consider-

able reservation, for in some cases the mitral presystolic and diastolic murmurs are audible over a much wider area,

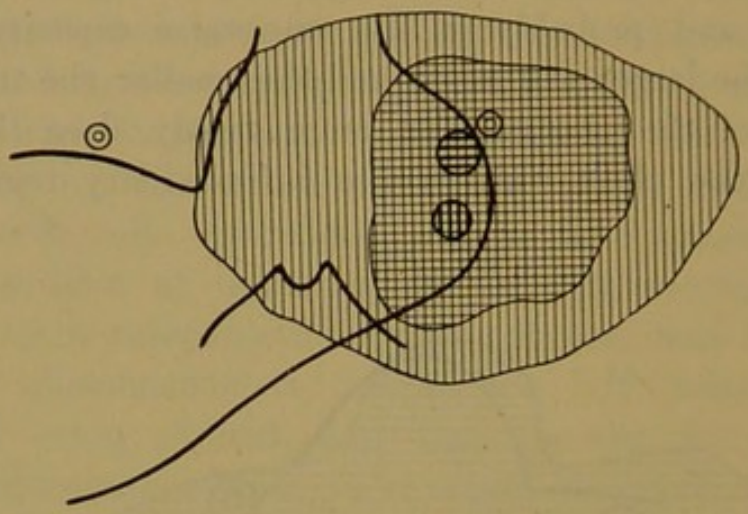


Fig. 34.—Area of audition of presystolic mitral greater than area of audition of mitral systolic murmur.—Gibson.

reaching, in fact, a point to the right of the mid-sternal line. This is seen in the accompanying diagrams.

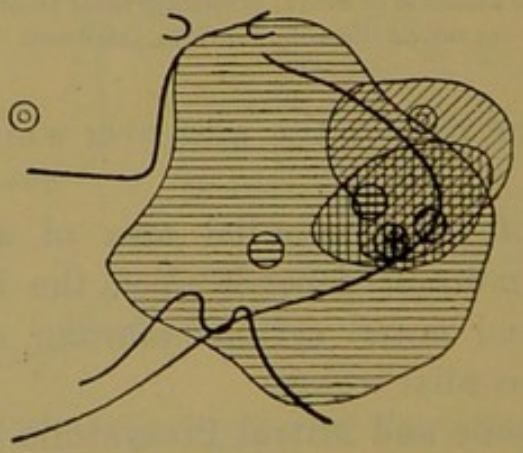


Fig. 35.—Area of audition of presystolic and diastolic mitral murmurs and systolic mitral and tricuspid.—Gibson.

Pulmonary Systolic Murmur.—This murmur has its point of maximum intensity over the pulmonary artery in the second left intercostal space close to the edge of the sternum.

It is, as a rule, a localised murmur, and is rarely if ever heard beyond 1 in. from the sternal edge, or over the sternum adjoining the space, or over the cartilage above or below it. It is frequently present, more especially in anæmia and debility, and it does not, as a rule, denote organic disease at the orifice of the vessel. It will be referred to more fully when functional or anæmic murmurs are considered. Pulmonary systolic murmur is very rarely due to organic lesions of the valves, but when it is, it is commonly congenital and associated with other lesions, as has been already mentioned at p. 60. When due to organic disease it is much louder, and, while attaining its point of maximum intensity over the orifice of the vessel, it may be widely propagated and audible over the whole præcordia and the whole length of the sternum: it is not, however, propagated into the carotids, and this serves to distinguish it from a loud aortic systolic murmur.

Pulmonary Diastolic Murmur.—This murmur is analogous to aortic diastolic murmur. Its point of maximum intensity is either over the orifice of the vessel or at the lower part of the sternum. Its direction of propagation is down the sternum and over the right ventricle to the left of the sternum. It is very rare, and when present may readily be mistaken for an aortic diastolic murmur. If it were louder over the right ventricle to the left of the sternum than at the apex of the left, it would aid in its differentiation.

Tricuspid Systolic Murmur.—At one time this was regarded as a very rare endocardial murmur. It is no longer so regarded, and there can be no doubt that it is really one of the commonest. Its point of maximum intensity is generally stated to be at the base of the xiphoid, but this is not invariably the case.

The murmur may be heard only in the spaces from the third to the sixth left costal cartilage, and it may be of equal intensity in all of them. When most typical, how-

ever, it is heard over all that part of the thorax which covers the right heart, that is to say, it is heard from the septum of the ventricles inwards to the sternum over a considerable area, the left boundary of which is formed by a line drawn from the third left cartilage to a point not far from the apex, the inferior boundary being formed by the lower border of the right ventricle. It is also audible over the sternum adjoining this area, and to the right of the sternum in the third, fourth, and fifth spaces, for from 1 inch to an inch and a half from the right edge of the sternum. When the murmur is thus heard over the whole right heart it commonly attains its maximum intensity over the lower third or two-thirds of the sternum. The position of the tricuspid systolic murmur is thus what might be expected from the relations of the right heart to the thoracic wall. It is heard to the right of the sternum, because the right auricle extends to the right of the sternum; the regurgitating stream carries the sound with it into this chamber, and is propagated through its wall to the thoracic wall. Some other points bearing on this subject will be referred to under anæmic murmurs.

Tricuspid Diastolic and Presystolic Murmur. — Both these murmurs are extremely rare. Their point of maximum intensity ought to be on the sternum over the tricuspid orifice, and they ought to be propagated towards the apex of the right ventricle.

Summary of Preceding

	Point of Maximum Intensity.	*Area of Audition and Direction of Propagation.*
1. Aortic Murmurs.		
A. Systolic . .	Base of sternum.	Over whole sternum; into carotid artery; over whole præcordia; and sometimes over whole thorax, anteriorly and posteriorly.

	Point of Maximum Intensity.	*Area of Audition and Direction of Propagation.*
B. Diastolic .	Sternum at level of second cartilage, or lower part of sternum.	Over whole sternum, but especially over lower three-fourths; at apex; over whole præcordia to left of sternum.
2. Pulmonary Murmurs.		
A. Systolic . .	Second left space, close to sternum.	Not propagated in most cases, but may be heard over whole præcordia.
B. Diastolic .	Second left space.	Down sternum and over right ventricle to left of sternum. (Very rare.)
3. Mitral Murmurs.		
A. Systolic . .	Apex of left ventricle.	Over area round nipple; outwards towards axilla; round to angle of scapula; sometimes inwards towards sternum and upwards; rarely all over præcordia.
B. Diastolic .	Do.	
C. Presystolic .	Do.	
4. Tricuspid Murmurs.		
A. Systolic . .	Over lower third of sternum, or in adjoining spaces to left of sternum.	Lower two-thirds of sternum; over right ventricle to left of sternum; over right auricle to right of sternum; over manubrium.
B. Diastolic and Presystolic	Lower half of sternum.	Right ventricle to left of sternum.

The Tone, Quality, or Character of Murmurs.—The general law regarding the tone of murmurs is that direct

or obstructive murmurs — that is, murmurs produced by obstruction at any of the orifices—are rough in character, while indirect or regurgitant murmurs are soft and blowing in character. This cannot, however, be regarded as of much diagnostic significance, although in many cases the difference is well marked. Some murmurs are distinctly musical in character, depending probably on some incidental element in the lesion, the nature of which it is usually impossible to do more than guess.

There is, however, one murmur which it is necessary to refer to more specially, namely, the *mitral presystolic*; it is a rough murmur, and runs up to the first part of the systole, that is, to the first sound: it may be compared to the sound produced by pronouncing *wrup*. It is frequently confused with a systolic murmur, which occurs very commonly in mitral stenosis, and which ends with an accent, the accentuated part being probably produced by the somewhat delayed flap of the rigid valve. This special modification of the systolic murmur is quite diagnostic of mitral stenosis, but it has nevertheless to be distinguished from the presystolic murmur.

The sound character of a murmur is of importance when a murmur is well heard over a wide area of the præcordia, and the observer is called upon to decide if a single murmur or if two murmurs, having the same time, be present. A difference in the sound character will then be of the greatest importance in forming a decision.

The Intensity or Loudness of Murmurs.—This varies within wide limits. The loudest murmurs are usually aortic in origin. It is a point, however, the clinical significance of which is apt to be misunderstood. It necessarily depends upon two factors—(1) The nature or extent of the lesion, and (2) the vigour with which the blood is driven through the affected orifice. In systolic murmurs, especially at the aortic orifice, great loudness is not of the same serious import as a proportionally loud diastolic murmur at the same

orifice. In some cases this is equally true of mitral murmurs. Loud systolic murmurs, however, always imply that the ventricle is acting forcibly, and is therefore well nourished. The converse is equally true, and thus it is that when the cardiac muscle is enfeebled or degenerated, murmurs become less distinct, and may even disappear, reappearing when the ventricle regains power, under treatment. *The reappearance or intensification of murmurs* is thus in many cases of good omen. The murmur which of all others is most prone to disappear and reappear is the presystolic mitral. This depends not only on the strength of the left auricle, but also, we think, on the varying degree of distention of the right ventricle, for when the right ventricle is more distended than usual the left apex is pushed backwards by it, and thus away from the chest wall, and the murmur is not then conducted to the thoracic wall.

Multiple Murmurs and their Differentiation.—Several murmurs are frequently present in the same patient, and it will be of assistance to indicate some of the combinations which commonly occur, and the method by which they may be differentiated from one another.

In auscultating the heart, the system which is generally in use is to begin by listening at the apex. We, however, have found it better to begin by listening at the base, over the manubrium, and at the level of the second cartilage.

A systolic murmur in the former position, and a diastolic in the latter, indicate that the murmurs are probably aortic in origin. When no murmur is present it may be concluded that the aortic orifice and its valves are normal. The advantage of beginning at the base is thus apparent, for it can be at once decided whether or not one of the most commonly affected orifices is the seat of lesion. From the base we next listen over the lower third of the sternum. If murmurs have been heard at the base, they will probably, but not necessarily, be heard here also; but we shall suppose that

there was no murmur heard at the base, but that a systolic one becomes audible over the lower part of the sternum; when this is the case the murmur is a tricuspid systolic one. It may be followed to the left of the sternum, over the right ventricle, but is lost before we reach the apex. If, however, as most frequently happens, there is a mitral systolic murmur in addition to the tricuspid, we find that as we near the apex the systolic murmur becomes louder, and that it may even be followed outwards beyond the apex towards the axilla. The annexed figure represents a case of this kind. If there is no tricuspid systolic murmur, but only a mitral systolic, there is often no murmur over the sternum or to the left of it, until we near the apex, and the murmur is louder an inch or so to the left of the apex than it is at the same distance to the right of it.

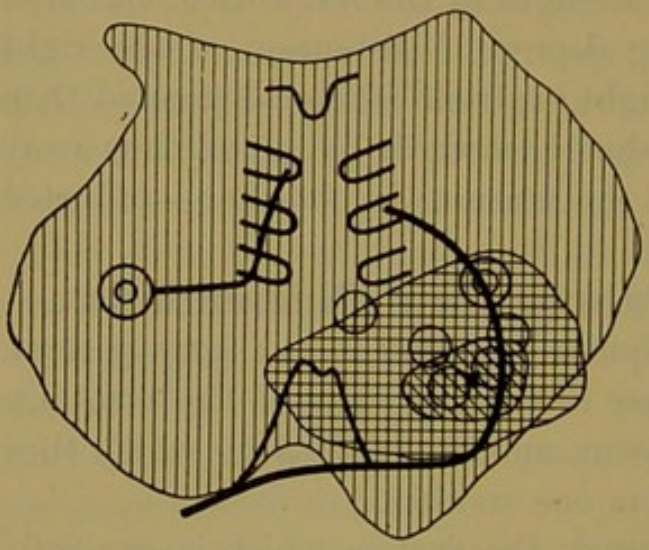


FIG. 36.—Area of audition of presystolic, systolic and diastolic mitral, and presystolic and diastolic tricuspid murmurs.—Gibson.

We shall take another common condition, in which the differential diagnosis is often very difficult, namely, a case in which there is a systolic murmur heard at the manubrium, all down the sternum, and to the left of the sternum as far as the apex, and even beyond it. Here there is no doubt of one point, namely, that there is a systolic aortic murmur, but the murmur is also audible in the tricuspid region and in the mitral area, and the question which has to be decided is whether or not there is a separate tricuspid and a separate mitral murmur. It is quite impossible to decide whether there is a tricuspid murmur; we may be able by the presence of venous pulsation to affirm that there is tricuspid reflux,

but the two murmurs, when present, cannot be separated by auscultation. The further question is—Is there a separate mitral systolic murmur? There are several rules which help us to decide this question. If the systolic murmur audible over the sternum becomes fainter as the stethoscope is moved to the left over the right ventricle, and again becomes louder or distinctly alters in tone at the apex and beyond it, we may conclude that there is an independent mitral systolic in addition to the aortic. Another aid which will be found of service is, that if the murmur is louder at the apex than it is over the left edge of the heart in the space above the apex, or the rib above that, the probability is that there is a mitral as well as the aortic murmur. The reason of this last is apparent, for if the murmur at the apex be the aortic systolic propagated downwards by the left ventricle, there is no reason why it ought not to be as audible at any point along the left edge of the heart as at the apex. In some cases, however, and especially in those in which the aortic murmur is very loud, it is quite impossible by auscultation to determine whether there is a separate mitral. It may, however, be with accuracy assumed that there is mitral reflux if there be evidence of pulmonary congestion and heightened tension in the pulmonary artery.

A diastolic murmur of aortic origin, audible at the apex, is readily distinguished from mitral diastolic, as the former is a longer murmur than the latter, and occupies the whole time of ventricular diastole, including the time occupied by the second sound, which it may entirely mask and render inaudible.

The three murmurs which have their origin at the mitral orifice may all be present in the same case, but this is not so common as the union of presystolic and systolic.

Further Points in the Diagnosis of some Cardiac Conditions.—In many cases of mitral stenosis, neither a diastolic nor a presystolic murmur is present, and yet the diagnosis may be made with practically absolute certainty. The

presence of a systolic murmur ending with an accent has been already referred to as indicative of this lesion. In some cases, however, we have not even this present as a guide, and then the phenomena on which the diagnosis depends, are—(1) Great irregularity in the force and rhythm of the first sound and of the cardiac impulse; (2) a similar irregularity in the force and rhythm of the pulse, and a great disproportion between the number of cardiac impulses, as felt over the præcordia and the radial pulsations, the former being often nearly twice as numerous as the latter; (3) marked reduplication of the second sound; (4) the presence of a systolic murmur at the apex, which may, however, only be heard occasionally, or may not be heard at all when the symptoms are at their worst; and (5) the distention of the right side of the heart as evidenced by the diffuse heaving over it, the presence of a soft tricuspid murmur, and the distention of the veins at the root of the neck.

It is as well to warn the student that it is often very difficult, and sometimes quite hopeless, to satisfactorily analyse the sounds in mitral stenosis. This results from the multiplicity of sound-producing factors in such cases. Among these may be mentioned a possible, although a questioned, non-synchronous action of the two ventricles; at least it is held by some observers that there are contractions of the right ventricle in which the left ventricle does not seem to participate. There is an occasional loud sound, and a strong systole which can be felt in the carotid pulse, followed by a series of short and irregular sounds, none of which have a corresponding pulsation in the carotid, and which may be caused, some by reduplication of the second sound, others by abortive ventricular contractions, and still others possibly by auricular contractions; but to which cause each sound is to be relegated is, we fear, beyond the discriminative faculty of the human ear. Notwithstanding these difficulties, which are mainly matters of abstract interest, the diagnosis can, as we have said, be made from the very difficulties of

the analysis of the auscultatory phenomena. As the heart regains its equilibrium, and distressing symptoms disappear, the analysis of the sounds becomes, however, practicable.

In the fatty and dilated heart percussion shows the heart enlarged, but not necessarily to any very great extent. The auscultatory evidence is mainly or purely negative. The first sound at the apex is either inaudible or extremely feeble, while the second sound continues more or less distinct: beyond the apex the first sound is quite inaudible. It may, however, be audible over the right ventricle, especially over the sternum, but the sound here is certainly caused by the right and not the left ventricle. In many cases the cardiac action is irregular, and there may be frequent intermissions. Further evidence of the weakness of the ventricle is found in congestion of the lungs from the engorgement of the pulmonary circulation; and perhaps of the systemic venous system also, œdema of the lower limbs being frequently present.

There is sometimes a question of diagnosis, between mitral stenosis and fatty dilated heart with mitral regurgitation, owing to the irregularity which may be present in the latter as well as in the former. The irregularity, in cases of equal gravity as estimated by the symptoms, is, however, very much more marked in mitral stenosis.

Secondary Results of Valvular Lesions.—The results of valvular lesions can only be understood by having a very clear mental picture of the circulation, and by being able to call it up without effort.

The general law which may be laid down is, that *hypertrophy and dilatation* occur in the chamber *immediately behind the affected valve*, and that, as the case advances, the effect tells farther back, step by step. Let us illustrate this. Take first aortic stenosis: the result of this lesion is that the left ventricle, which is the chamber immediately behind the affected valve, first dilates and then hypertrophies.

So long as the heart muscle is well nourished the hypertrophy is able to overcome the increased resistance, and there is a state of *equilibrium* or of *compensation*, but, if the nutrition of the muscle be not sufficient for the extra strain, the muscle relaxes and the ventricle becomes dilated. The dilatation tends to increase because the obstruction continues and the condition of impaired nutrition is prone to recur. The impaired nutrition may be the result of extra strain thrown on the heart, which is at the time nourished to its utmost limits; for it has always to be borne in mind that there are limits to increase of nutrition, however wide these limits may be. Whenever this impairment of nutrition or this extra strain (and they may be held as analogous as regards results) supervenes, dilatation results, and thus it is that rest becomes such an important measure of treatment when failure occurs. As a result of repeated loss of equilibrium between the obstruction and the force behind it, the ventricle becomes more and more dilated, and as a consequence the mitral orifice is enlarged and the valves no longer close it: this leads to mitral regurgitation. Mitral regurgitation leads to dilatation and hypertrophy of the left auricle. This after a time leads to engorgement of the pulmonary circulation, and to increased tension in the pulmonary artery, indicated by accentuation of its second sound. Engorgement of the pulmonary circulation leads also to the physical signs of bronchitis from the bronchial vessels sharing in the engorgement, owing to their connection with the pulmonary veins. The engorgement also leads to the effusion of fluid into the smaller bronchi and alveoli, producing crepitations; or into the pleural cavity, giving rise to hydrothorax. An early result of the increased tension in the pulmonary artery is that the right ventricle dilates, then hypertrophies, and after a time dilates again. The dilatation increases, as the dilatation of the left ventricle increased, from the obstruction to the outflow of its blood, and following this there is enlargement of the tricuspid orifice with failure

of the valves to close it, with, as a result, dilatation and hypertrophy of the right auricle. The final stage is engorgement of the whole venous system, and, as a consequence, effusion of fluid from the capillaries into the tissues and serous cavities, and the establishment of general dropsy or anasarca.

If, in addition to stenosis, there is also regurgitation at the aortic orifice, or if regurgitation occur alone, there is a further dilating force, for there is during diastole not only the normal quantity of blood flowing into the ventricle from the auricle, but in addition a quantity flowing backwards into it from the aorta. The ventricle has to accommodate itself to this increased quantity of blood, and it can only do so by dilating. Here, too, however, a certain degree of dilatation and of hypertrophy maintain the circulation in equilibrium; but extra strain or impaired nutrition are apt to occur, and dilatation increases with its consequent evils, and there is what is known as *failure of compensation.*

From the foregoing it will be easy to trace the changes which follow in the course of time on any given valvular lesion, and they need not be individually detailed.

The general result of all valvular lesions is, that the quantity of blood in the parts in front of the lesion tends to diminish, while the quantity behind it tends to accumulate, —in other words, the pressure in the arterial system falls, while that in the venous system rises.

It cannot be too strongly impressed upon the student that, while his first task is to master the auscultatory evidence of various valvular lesions, his further and even more important duty is to be able to *distinguish between compensation and failure of compensation*, and to estimate the degree of failure and the power of regaining what may have been lost. He must on no account assume that the presence of cardiac murmurs necessarily demands therapeutic measures, or implies that his patient's death-warrant is to be promptly sealed. On the other hand, many cases in which there are no murmurs

require the utmost care and skill to be directed to the treatment of the heart.

Hæmic Murmurs.—The murmurs present in anæmia are known as *functional or hæmic murmurs.* The first term is used to imply that they are not due to organic structural changes of the valves, and the second that they are due to the condition of the blood. They are usually classed as being produced in the veins, the arteries, and the heart.

1. **Venous Murmurs.**—If the stethoscope be placed over the right jugular vein between the origins of the sterno-cleido-mastoid muscle, a loud continuous murmur of a humming or blowing character is heard in most cases of anæmia and chlorosis. It received the name of *bruit de diable* from its resemblance to the sound made by a French toy of that name. In some cases it is distinctly squeaking in character, more especially if the cardiac action be rapid from the excitement due to examination, or to any little special exertion immediately preceding examination. Although the murmur is continuous it commonly presents distinct periods of greater loudness, which vary in time in different cases; in some the periods of greater intensity being synchronous with the cardiac movements, in others with the respiration. It is heard best if the patient's head be turned to the left, and the chin be at the same time raised. The physical conditions leading to its production have not been satisfactorily defined. The two factors upon which most stress has been laid in the attempt to explain it are, first, the abnormal physical characters of the chlorotic blood, and, secondly, the production of a fluid vein. For the production of the second of these it is necessary that the blood should pass from a narrow into a wider part of the vein. In anæmia it is assumed that, owing to diminution of the total volume of the blood, the veins contract, but that the lower part of the jugular vein cannot so contract owing to its attachments to the adjoining fascia, and that in this way the relatively wider part is obtained and the murmur is pro-

duced. The explanation is unsatisfactory because there is no evidence that in chlorosis there is a diminution in the total volume of the blood, and the statement based upon it, namely, that the veins contract to adapt themselves to their lessened contents, can hardly be accepted without further proof than is at present forthcoming. On the other hand, from the facility with which this murmur can be produced in chlorotic patients by placing the head in the position indicated above, it would seem that interference with the lumen of the vein from traction upon it, coupled with the alteration in the physical characters of the blood, may be the more important factors in its production. An alteration in the character of the blood is usually ascribed as a factor in the production of the murmur, but experimental observations would seem to negative this view, though it must be admitted that the fact that the murmur cannot be produced in healthy individuals by the position of the head, which so easily produces it in anæmia, is difficult of explanation on any other ground. This murmur is sometimes present over the upper part of the sternum, and its presence here is difficult to explain. A murmur of a similar character may be heard over the torcular Herophili and over the orbit, the stethoscope being placed over the occipital protuberance or the closed lid. The systolic accentuation when present can only be accounted for on the assumption of a backward current during ventricular or auricular systole, and there is reason to accept this, from the fact of the venous fulness and pulsation which is present in these cases when the patient is in the recumbent position.

2. **Arterial Murmurs.**—An aortic systolic murmur, having its origin at the orifice of that vessel, is usually described as being present, but we consider, as stated under the following section, that this is, as a rule at least, a mistake. If it is ever present, its mode of production is not satisfactorily explained.

3. **Cardiac Murmurs.**—These are the most important, as

they are often mistaken for murmurs due to grave anatomical changes in the valves.

They are always systolic in time, but their seat of origin, and the area over which they are audible, vary in individual instances; they will therefore be considered seriatim.

(*a*) *Pulmonary murmur.*—This murmur is audible in the second left intercostal space, for an inch to an inch and a half from the sternal edge. It is strictly localised in this area, not being heard over the ribs or the sternum adjoining it. It may be soft or somewhat rough in character. It is produced in the pulmonary artery, probably from some interference with the lumen of the vessel near its origin. The precise way in which the lumen is affected is discussed elsewhere.[1] The murmur is often only audible when the patient is lying down, and usually disappears during a moderately deep inspiration. It is frequently present in cases of debility when there is no marked evidence of anæmia. It is followed by an accentuated pulmonary second sound.

(*b*) *Tricuspid murmur.*—The development of this murmur can under favourable circumstances be watched. In such cases we have found that it is audible first in the third and fourth left spaces near the edge of the sternum. When fully developed it is heard over the whole right heart,—that is, in the third, fourth, and fifth left spaces, extending farther to the left the lower the interspace, but not reaching farther out than a point about one inch to the inside of the apex of the left ventricle; over the sternum adjoining these spaces; and, in some cases, in the second, third, and fourth right spaces for an inch or one inch and a half from the edge of the sternum. This, as is seen in the frontispiece, is the part of the chest overlying the right heart. If the murmur be heard in the above positions, when the patient is in the upright or sitting position, it may often be noted that, shortly after the recumbent posture is assumed, the murmur

[1] "Investigations into some Morbid Cardiac Conditions," by William Russell, M.D. Edinburgh, 1886.

may in addition be heard over the manubrium sterni. The presence of the murmur in this latter situation has long been regarded as evidence that there was an aortic systolic murmur present. This, however, is a mistake,—the true explanation being, that in the recumbent position the distention of the right chambers becomes greater, and, owing to this distention and a greater reflux through the tricuspid orifice, the murmur is heard over a wider area. That increased distention occurs is shown by pulsation of the right ventricle and by increased fulness of the veins in the neck.

The murmur is due to reflux through the tricuspid orifice from the dilated and enfeebled condition of the right ventricle; and when it is present the pulmonary second sound is sometimes not accentuated.

(*c*) *Mitral murmur.*—This is the least common of the murmurs present in the conditions under consideration. It is, however, not infrequent, and is a soft blowing murmur, audible at the apex and propagated slightly outwards. It is systolic in time, and due to regurgitation through the dilated orifice.

All these murmurs disappear as the patient recovers, and as the heart regains tone and strength.

Examination of the Blood Vessels

(*a*) **Arch of Aorta.**—It is frequently necessary to investigate the condition of the arch of the aorta, as it, more than any other part of the vessel, is liable to aneurysm. It is investigated by inspection, palpation, percussion, and auscultation.

Inspection may reveal pulsation above the base of the heart on either side of the sternum; more frequently, however, to the right than to the left of it. Pulsation may also be present in the episternal notch. In cases of large aneurysm the sternum itself may move with the systolic expansion of the sac. In the most severe cases there is

great bulging of the chest wall covering the aneurysm, the bones become eroded, and there is thinning of all the structures overlying it, so that the sac ultimately may be covered only by integument.

By **palpation** the pulsation is felt, and its distensile character is, in marked cases, very distinct. In some cases a systolic thrill is present. The shock of the closure of the semilunar valves may also be intensified. When the aneurysm is large and superficial, it must be palpated with great care. Caution is still more necessary when percussion is attempted.

Percussion is necessary in all save the most evident cases. By means of it we determine whether the percussion sound is modified over the manubrium. In aneurysm and in other mediastinal tumours the percussion sound in this situation may be dull, the dulness extending to the right or left of the sternum.

On **auscultation** there may be a systolic murmur, but in a considerable proportion of cases there is no murmur. There is usually marked accentuation of the aortic second sound.

In addition to examining the aorta anteriorly it is necessary to *examine it posteriorly*: this is done in the interscapular region. It will be remembered that the aorta reaches the spine at the level of the third dorsal vertebra, so that a considerable aneurysm, involving this part of the vessel, may give evidence of its presence by dulness on either side of the vertebral column. When the bodies of the vertebræ are eroded by pressure there is tenderness on pressure, and there may even be curvature.

The typical signs of the presence of aneurysm are thus—pulsation, thrill, dulness on percussion, possibly systolic murmur, and an accentuated second sound. There are, however, other evidences of its presence which have to be examined for. They are known collectively as **pressure signs.** Thus there may be evidence of—(1) Pressure on one bronchus, as indi-

cated by snoring rhonchi over the seat of pressure, or by various modifications of the respiratory sounds over the lung; (2) pressure on the large venous trunks, leading to fulness of the veins on one or both sides of the face, neck, and upper extremity, and even to œdema of these parts; (3) pressure on the trachea, giving rise by its irritation to a peculiar brassy and hollow cough, or interfering with the entrance of air by direct pressure, and so producing an inspiratory stridor; (4) pressure on the œsophagus, producing difficulty in swallowing; (5) pressure on the recurrent laryngeal nerve on the left side where it turns up under the aortic arch, leading to paralysis of the left vocal cord; (6) pressure on the sympathetic, causing inequality in the pupils; (7) pressure on the vagus or the phrenic; (8) pressure on bone, as already referred to, causing caries and absorption. Finally, *the pulses on the two sides have to be compared.* In aneurysm there is not infrequently a marked inequality, owing to the involvement of the origin of one of the large trunks arising from the arch in such a way that its lumen is diminished, and the circulation through it consequently impeded.

An important symptom of aortic aneurysm is known as "**tracheal tugging.**" It may be made out by inspection when the patient bends the head back, so putting the trachea on the stretch. The trachea will then be visibly dragged down with each cardiac contraction, and if the finger be placed between the thyroid and cricoid cartilages and gentle upward pressure exerted, distinct tugging will be felt with each cardiac revolution. The symptom is of great diagnostic value in aneurysm of the transverse portion of the aorta.

While all these evidences of aneurysm have to be borne in mind, too much stress must not be laid on any one of them, for here, more than in many conditions, the presence of some symptoms, and the absence of others, have to be carefully weighed.

Atheroma of the arch of the aorta, with moderate dilata-

tion, associated with hypertrophy of the heart and calcareous deposit at the bases of the aortic cusps, may very readily be, and often is, mistaken for aneurysm. The differential diagnosis is probably often impossible, but the correspondence in the physical signs in the two conditions must be borne in mind. The points in which they correspond are the presence of pulsation in the episternal notch, increased area of dulness, systolic murmur, and accentuated aortic second sound. Pressure symptoms are not, however, often present.

(*b*) **Descending Thoracic Aorta.**—This part also of the vessel may be the seat of aneurysm. If it is large its position may be made out by percussion, and there may be a murmur. If erosion of the vertebræ has supervened there is tenderness on pressure, while in severe cases there may be spinal curvature and paraplegia from implication of the cord. Minor degrees are hardly discoverable by physical examination.

(*c*) **Innominate Artery.**—This vessel is often involved in aneurysm of the aortic arch; it may, however, be affected apart from it. The bulging, unless the sac be very large, is to the right of the sternum, and reaches to the level of the sterno-clavicular articulation. The orifices of the subclavian and carotid arteries, into which it divides, are very liable to be implicated, and to lead to a difference in the radial and carotid pulses on the two sides. The other signs correspond with those of aneurysm of the arch, but the pressure phenomena are not so varied.

(*d*) **Abdominal Aorta.**—Aneurysm of this part of the vessel will be found under the section on the abdomen.

(*e*) **Carotid Arteries.** — Normally the pulse in these vessels is not visible. In double aortic lesion — that is, when there are both stenosis and regurgitation—the pulsation of the arteries, especially the right one, is visible right up to the ear. This is the only valvular lesion in which this phenomenon is present; it may, however, be present in

exophthalmic goître without lesion at the aortic valves. We have already mentioned that systolic aortic murmur is propagated into and can be heard over the carotids. In addition to this, however, in cases of aortic regurgitation, if the stethoscope be pretty firmly applied to the carotid, *a diastolic murmur* can be heard. This murmur is not, of course, the diastolic murmur propagated from the orifice of the aorta, for murmurs are not propagated in a direction opposite to the direction of the stream producing them, but it is caused by the backward-flowing stream in the vessel passing the part narrowed by the pressure of the stethoscope. This diastolic murmur can often be beautifully heard in the femoral arteries by carefully graduating the pressure with the stethoscope.

(*f*) **Radial Arteries—Examination of the Radial Pulse.**—From what has already been pointed out, it is clear that a study of the condition of the arterial pulse is fraught with indications of a far-reaching character; and in order to acquire skill, not only in the diagnosis of circulatory disorders, but in the investigation of all affections which have even a remote influence upon the circulatory system, a knowledge of the changes which it undergoes is in the highest degree necessary. Such knowledge can only be attained by diligent practice and patient study, since the modifications of the arterial pulse are so numerous and so various as to require much careful observation for their detection, while the student must undergo considerable training before he can possess the requisite faculty of appreciating the differences between the pulse of health and disease.

In the following pages the methods of ascertaining the state of the pulse will in the first place be considered, after which the changes which are to be found in different conditions will be described.

Method of observing the Pulse.—In this place attention must be called to the locality where the pulse may

be most conveniently studied, and the means by which its investigation is to be carried out will afterwards be described.

The examination of the arterial pulse is usually confined to the radial arteries, and the part of the vessels subjected to investigation is that which lies immediately above the wrist, between the flexor tendons on the inner, and the prominent ridge of the radius on the outer side. In this situation the arteries are only covered by the skin and subcutaneous tissues; they can therefore be felt quite easily, and may often be seen as well. At this point, moreover, the vessel is in contact with the radius, and, in consequence of this fact, its examination is rendered at once more simple and more thorough, inasmuch as the bony floor upon which it lies gives a fixed base against which it may be firmly pressed.

The pulse should, if possible, be examined while the patient is in a sitting or lying posture, and the arm which is employed for the purpose must rest upon some fixed object, or be supported by the hand of the observer which is not made use of in palpating the artery. The observer should place himself in front of or to the right of the patient, whose right arm is to be in the semiprone position, with the back of the hand turned to him; he must then pass his right hand across the radial surface of the patient's wrist, and lay the tips of his middle and ring fingers upon the vessel. If the patient's left radial artery is to be examined, the observer should stand to the left and employ his left hand in a similar manner; while if both radial arteries are to be examined simultaneously, he should, as far as possible, stand in front of the patient and use his right hand for the left artery and his left for the right. Attention to these details may seem unnecessary, but the adoption of this method will be found to render the examination of the pulse at the same time more easy and more precise. Laying the tips of the fingers at first lightly upon the artery, the pressure should be varied,

in order to bring out the different points, which will immediately be described.

The only instrument to which reference need be made as of any use in a study of the pulse is *the sphygmograph*, and, as it is nowadays so well known, a description would be as superfluous in its case as in the case of the stethoscope. There are many modifications of the sphygmograph, but the only essential difference between them is that some have the pressure applied, as in Marey's instrument, by means of a spring, while others, such as Sommerbrodt's sphygmograph, have actual weights which bear upon the artery. Of the many varieties which are employed, the only ones which we are able to recommend are Sommerbrodt's, Marey's, and Mahomed's.

It must be borne in mind that the sphygmograph is of limited use in diagnosis. It reveals no new fact which can enable us to discover affections unknown before its invention; it nevertheless brings into prominence certain points in regard to the pulse which would be less definite without its aid, and it is on this account of some clinical interest. On the other hand, there are some aspects of the pulse which can be much more accurately gauged by means of the finger than by the help of the sphygmograph. The instrument can therefore only be regarded as a supplement to the finger of the observer.

In employing the sphygmograph there are a few points which must be attended to. The patient must, in the first place, assume a comfortable attitude, whether sitting or lying, while the arm, the artery of which is to be examined, must be put in an easy position, almost fully supinated, and resting upon a steady support. The button of the instrument is then to be accurately placed upon the most prominent part of the artery, and, after the point yielding the largest movement of the lever has been ascertained, the sphygmograph is to be lashed to the wrist. The pressure, lastly, has to be adjusted, and it will be found that there is one definite amount which gives the fullest amplitude of

movement, an increase or decrease of the pressure yielding a less perfect tracing. Such are the points which must be borne in mind. The use of the sphygmograph can only be acquired by dint of assiduous practice. In the following

FIG. 37.—Tracing taken with Marey's sphygmograph from the radial artery in a case of mitral incompetence; pressure 2 oz.

pages its indications will be mentioned along with the descriptions given of the different elements of the pulse, and their variation as ascertained by means of the finger.

It is necessary to recognise the fact that tracings taken from the same artery at the same time, but by means of

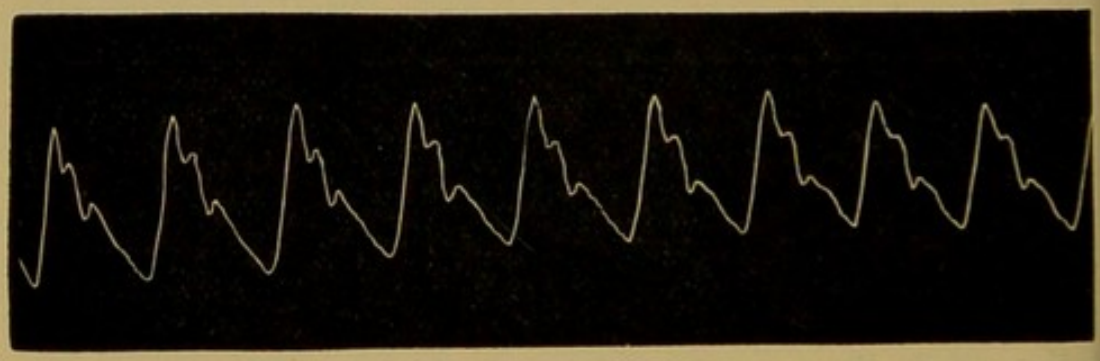


FIG. 38.—Tracing taken with Sommerbrodt's sphygmograph from the radial artery in a case of mitral incompetence; pressure 2 oz.

different forms of sphygmograph, may present very varied appearances. The two tracings above, for example, were obtained at the same time from the right radial artery of a patient suffering from mitral incompetence with hypertrophy of the left ventricle. The pressure in both cases was the same, but in the case of the tracing taken with Marey's

instrument it was due to a spring, while in the case of that obtained by means of Sommerbrodt's instrument it was applied by direct weight. It will be observed that the lever in the former tracing describes the arc of a circle from swinging parallel to the moving paper; while in the latter the lever gives a perfectly vertical line, because the paper travels at right angles to it. This gives the upstroke an absolutely true relation to the rest of the tracing. In tracings taken with Marey's and Mahomed's instruments the upstroke has always a backward inclination on account of the circular movement of the lever.

Phenomena to be studied.—In the observation of the characters of the pulse, it is convenient to separate out its different elements, and estimate each of them in turn. Several of these are doubtless only different phases of the same conditions, but it conduces to the ease of its investigation when the phenomena which the pulse presents are severally analysed. Attention should therefore be devoted in turn to the following points:—

1. The condition of the arterial wall.
2. The character of the pulsation as regards—
   (*a*) Rate.
   (*b*) Rhythm in time and in extent.
3. The character of each pulse wave as regards—
   (*a*) Volume.
   (*b*) Force.
   (*c*) Duration.
   (*d*) The presence or absence of secondary waves.
4. The sustained pressure within the vessel.
5. The character of the pulsation in different arteries at the same time.

Normal Characters of the Radial Pulse.—The vessel should be soft and yielding, moderately filled with blood, and easily compressible. The rate of pulsation may vary greatly within certain limits, but the rhythm should be per-

fectly regular as regards time and extent. Each pulsation should be of moderate and uniform volume and force, while neither too long nor too short in duration, while the sustained pressure within the vessel should be moderate.

Fig. 39.—Tracing from pulse of healthy man ; pressure 2½ oz.

A sphygmographic tracing of the pulse in health, as in Fig. 39, presents a sudden and uninterrupted line of ascent, and a gradual line of descent, marked by two distinct undulations. The line of ascent (*a-b* in Fig. 40) is almost vertical when taken with sphygmographs writing with a lever at right angles to the paper, and it inclines somewhat backwards when obtained with such a sphygmograph as Marey's. This line of ascent is commonly called the *percussion wave*, and it is caused by a swift wave of distention produced by the sudden entrance of the blood from the left ventricle into the arterial system. It terminates in a sharp apex. The line of descent (*b-g* in Fig. 40) is caused by the more gradual contraction of the vessel after the cessation of cardiac systole. As above mentioned, it is interrupted by two distinct elevations. The first or earlier

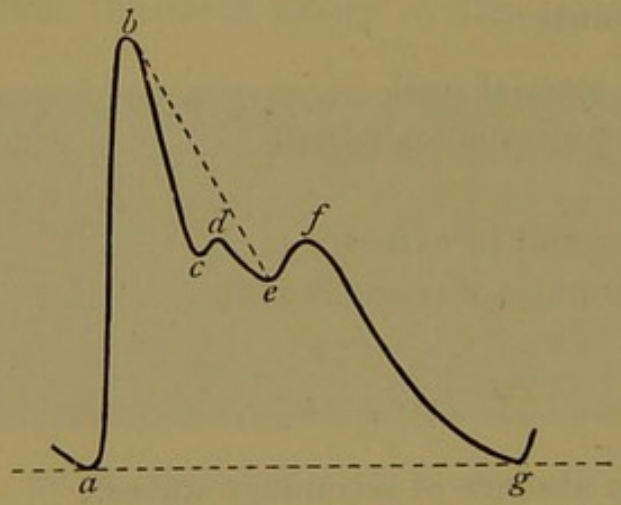


Fig. 40.—Diagram of pulse tracing in health.

of these (*d* in Fig. 40) is commonly known as the *predicrotic or tidal wave*, and it is believed to be caused by the blood stream coursing through the artery. The second or lower elevation (*f* in Fig. 40) is generally termed the *dicrotic or recoil wave*, and it is attributed to the reflection of an impulse from the closed aortic valves. These waves vary, as will be seen below, with the blood pressure, and it may be stated that their position depends on the distance between the heart and the artery from which the tracing has been obtained; the nearer the artery is to the heart, the higher upon the line of descent are these elevations.

1. **The Condition of the Arterial Walls.**—The walls of the radial artery in health are soft and yielding, but they give at the same time a feeling of elasticity to the fingers which are applied to them. The state of these walls gives a reliable guide to the condition of the arterial system. The chief departure from this healthy state is hardness and rigidity of the vessel, caused by thickening or atheromatous degeneration. The artery may be so hard and resistant as to roll from side to side under the finger. Such a condition is common in advanced years as the result of degenerative changes, in the endarteritis of syphilis and in chronic kidney disease. In order to discover if there is any tortuosity of the vessel, the observer should pass his fingers along its course up the arm.

Care must be taken to distinguish this condition from the state of the circulation known as high tension, caused by increased blood pressure. They are frequently associated together, and the means of differentiating the two conditions will be mentioned.

Tracings taken by the sphygmograph from patients whose arteries are atheromatous show a low upstroke, with a blunted apex, and very little tendency to any oscillations during the descent of the lever. The limited range of movement during the ascent of the lever, and the blunt appearance of the summit, are due to the rigidity of the walls,

which also causes the absence of the usual waves seen on the descending line of the tracing.

2. **The Character of the Pulsation** must be observed with regard to its rate and rhythm.

(*a*) **The rate of the pulse,** or the number of pulsations in a given interval of time, varies with changes in cardiac activity, blood supply, and arterial tone.

In an adult man the number of pulsations usually varies between 60 and 70 in a minute, but there are personal idiosyncrasies which in conditions of apparent health may lead to the rate of pulsation being as low as 20 or as high as 100 per minute. An increase in the rate constitutes a

FIG. 41.—Tracing taken from a case of advanced atheroma of the arteries by means of Marey's sphygmograph ; pressure 3 oz.

frequent pulse (*pulsus frequens*), and a diminution in the rate is termed an *infrequent* pulse (*pulsus rarus*).

The rate varies in health with sex, age, size, position, external temperature, hour of day, condition of digestion, occupation, and many other less important circumstances. Speaking generally, it may be said that the pulse is more frequent in the following conditions :—In the female sex ; in infancy and childhood ; in small persons ; in the upright attitude ; in high temperatures ; during the later periods of the day ; after eating and drinking ; and when engaged in any mental or muscular exertion.

The rate of the pulsation is increased by the febrile state, and the increase is in almost all diseases directly proportional to the rise of temperature. In enteric fever and cerebral inflammations this direct ratio is usually absent. The pulse

is increased in frequency in certain nervous diseases, especially exophthalmic goître, and nervous palpitation. It is also more frequent in almost all the organic diseases of the heart, the only common exception being aortic stenosis.

The pulse is rendered less frequent in the critical stages of fevers, especially when there is a tendency to collapse; in certain disorders of the blood, such as jaundice; in high tension pulses,—as, for example, in renal affections; in inflammations and degenerations of the muscular structure of the heart; and in some affections of the membranes of the brain.

(*b*) **The rhythm of the pulse** is absolutely regular in

Fig. 42.—Tracing taken with Marey's sphygmograph from the radial artery in a case of mitral stenosis; pressure 2½ oz.

health, both as regards the time and extent of the beat, but there are personal idiosyncrasies which lead to irregularity without any apparent cause.

The rhythm depends entirely upon the nervous influences governing the motor impulses giving rise to the cardiac systole, and these nervous influences may be disturbed by alterations in blood pressure, such as want of compensation. In mitral stenosis the irregularity of the pulse both in the time and extent of the beat is sometimes a marked feature of the disease even at an early stage, as in Fig. 42.

Irregularity of the pulse is frequently found in cardiac degeneration and in nervous diseases, and it is quite a common symptom in elderly persons without any apparent disease.

Irregularity of the pulse frequently assumes more or less periodicity in its occurrence,—that is to say, the irregularity recurs at regular intervals.

The omission of pulse waves constitutes the symptom known as *intermission* (*pulsus deficiens*), while the intercalation of additional pulsations is known as *intercurrence* (*pulsus intercidens*). These conditions are most commonly associated with lesions of the muscular or valvular apparatus of the heart. In certain cases the pulse waves occur in groups separated by intervals of time. When in pairs the pulse is termed *pulsus bigeminus*, when in groups of three, *pulsus trigeminus*. When a large alternates with a small pulse wave, the pulse is called *pulsus alternans*. In all such conditions the size of the pulsations is unequal. The sphygmograph reveals the characters of these varieties of pulsation in a striking manner.

It should be observed that in many cases some of the contractions of the heart are not sufficiently energetic to drive a wave of pulsation to the periphery of the arterial system. Such pulsations are said to be *abortive*, and they are a frequent cause of intermission.

3. **The character of each individual** pulse wave demands careful study, as it yields valuable evidence in regard to the heart and blood vessels.

(*a*) **The size** varies considerably according to the individual peculiarities of the person whose pulse is under examination, and it depends upon the condition of the vasomotor system, the activity of the heart, and the quantity of blood poured into the arterial system at each cardiac systole.

A large or bounding pulse (*pulsus magnus*) is observed in conditions of relaxation of the arterioles, as in fever, and in some cases of cardiac hypertrophy without constriction of the smaller arteries. It is usually very marked in aortic incompetence, in which, on account of regurgitation from the aorta into the left ventricle, there is a tendency to

emptiness of the arterial system. Fig. 43 gives a sphygmographic tracing from such a case.

A small or thready pulse (*pulsus parvus*) is found in cases of inanition, cardiac weakness, and obstruction in the arterial system,—as, for instance, in aneurysm. It is common in diseases of the mitral orifice, and is very marked in cases of extreme aortic stenosis. The accompanying illustration, Fig. 44, is a tracing from such a case.

A small pulse is frequently associated with high "arterial tonus," a large pulse with relaxed vessels.

In health it is found that the volume of the pulse increases with inspiration, and diminishes during expiration.

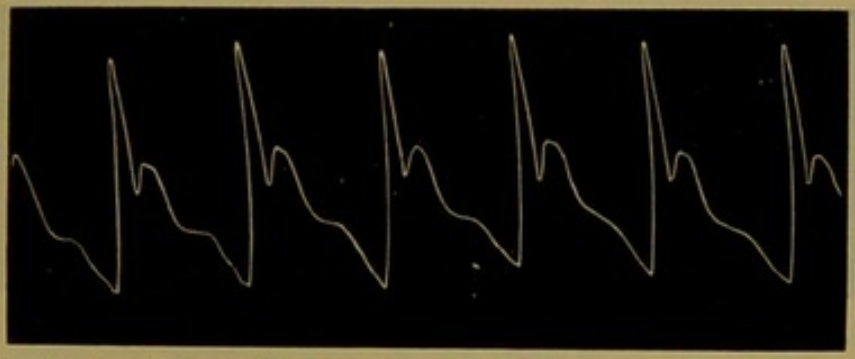


FIG. 43.—Tracing taken with Marey's sphygmograph from the radial artery in a case of aortic incompetence; pressure 3 oz.

A clinical feature is occasionally noted in which the converse occurs, *i.e.* a lessened volume during inspiration, and an increased size during expiration. This condition of the pulse is known as *pulsus paradoxus*. It has been most commonly noted in cases of stenosis of the air passages, and pericardial adhesions; these produce it by increasing the aspiratory force of inspiration.

The size or volume of the pulse wave is to be carefully distinguished from the fulness of the vessel between the beats.

(*b*) **The force** of each wave, the pulse pressure, gives the observer an estimate of the maximum blood pressure within the arterial system. It depends in the main upon the energy of the cardiac systole, but to some extent also upon the tone of the arterioles. It is to be estimated by plac-

ing the fingers upon the artery, and noting the pressure necessarily exerted by the proximal finger to obliterate the wave from the distal finger.

In all conditions of increased cardiac activity the force of the pulse is increased, while in debility of the heart it is diminished.

(*c*) **The duration** of each pulsation depends upon the relation existing between the dilatation of the artery by the blood current, and its contraction during the passage of the blood into the capillaries. The duration is longer when the blood has considerable opposition to overcome in passing out of the arteries, and shorter under converse circumstances.

FIG. 44.—Tracing taken with Marey's sphygmograph from the radial artery in a case of aortic stenosis ; pressure 2½ oz.

It is also longer when the systole of the heart is prolonged, and shorter when the cardiac contraction is brief.

As tested by the finger, the quick is readily distinguishable from the slow pulse by the duration of the impact felt by the observer. The sphygmographic tracing of the two shows a quicker ascent, sharper apex, and swifter descent in the former than in the latter.

A slow or sluggish pulse (*pulsus tardus*) is almost invariably associated with high arterial pressure; but this is not an absolute rule, as in aortic stenosis, for example, where there is much obstruction, the pulse may be extremely sluggish, although the arterial pressure is low. A slow pulse is found in all affections where there is contraction of the smaller arteries, such as angina pectoris, renal disease, and arterial sclerosis.

A quick or active pulse (*pulsus celer*) is found, as a rule, along with low arterial pressure, and it depends upon a relaxed condition of the arterioles for the most part. It is extremely common therefore in febrile affections. A very marked variety of the quick pulse is to be observed in cases of free aortic regurgitation, where the celerity is associated with an empty condition of the artery, and a large pulse wave.

In health the individual pulsations should all be equal in volume, force, and duration; but it is found that when there are irregularities in rhythm, there are almost invariably inequalities in size and force.

(*d*) **The presence or absence of secondary waves.**—When a healthy vessel is palpated, only one pulsation can be

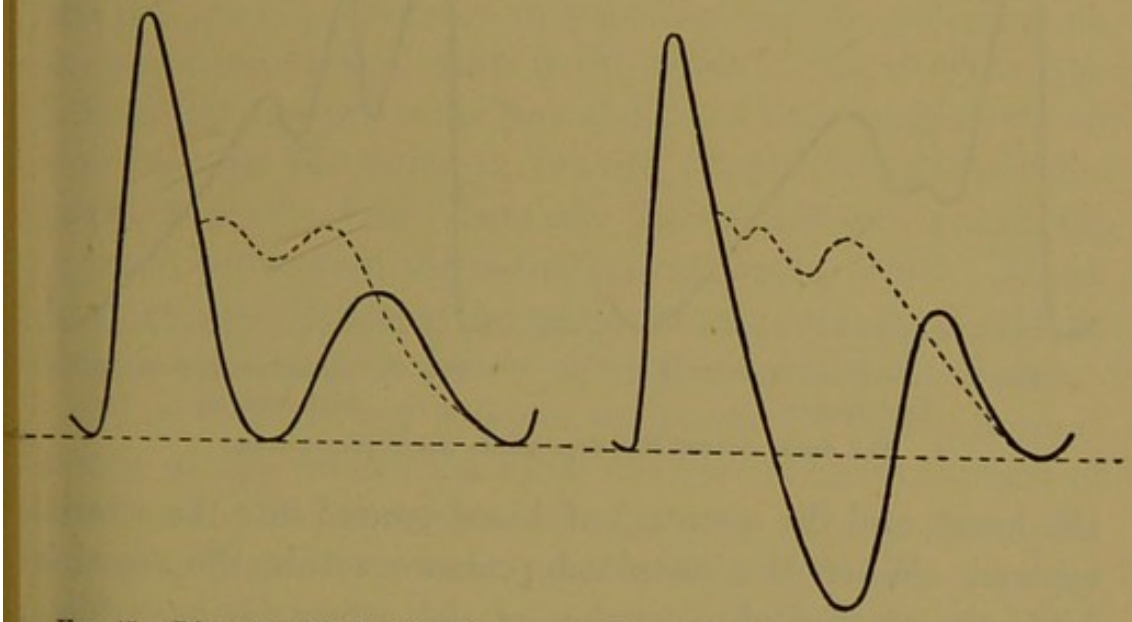


Fig. 45.—Diagram of fully dicrotic pulse.

Fig. 46.—Diagram of hyperdicrotic pulse.

The finely dotted line represents the normal curve.

felt corresponding with each cardiac systole; in certain conditions a second wave can be felt immediately following the first. This secondary wave is an exaggerated dicrotic wave; a tracing of such a pulse reveals an entire absence of the tidal wave, with an exaggeration of the dicrotic notch. Such a pulse is termed dicrotic. When the notch preceding the wave of recoil reaches the base line, as in Fig. 45, the

pulse is termed *fully dicrotic.* When the notch sinks below the base line, as in Fig. 46, the term hyperdicrotic is applied to it.

The condition of dicrotism is intimately associated with low arterial pressure. As a clinical feature it is most frequently found in the later stages of febrile diseases.

4. **The sustained Pressure within the Vessel** may be noted by estimating the pressure necessary to obliterate the vessel between the pulse beats. It gives an estimate of the minimum arterial pressure. It is intimately associated with the condition of the vasomotor system, the activity of

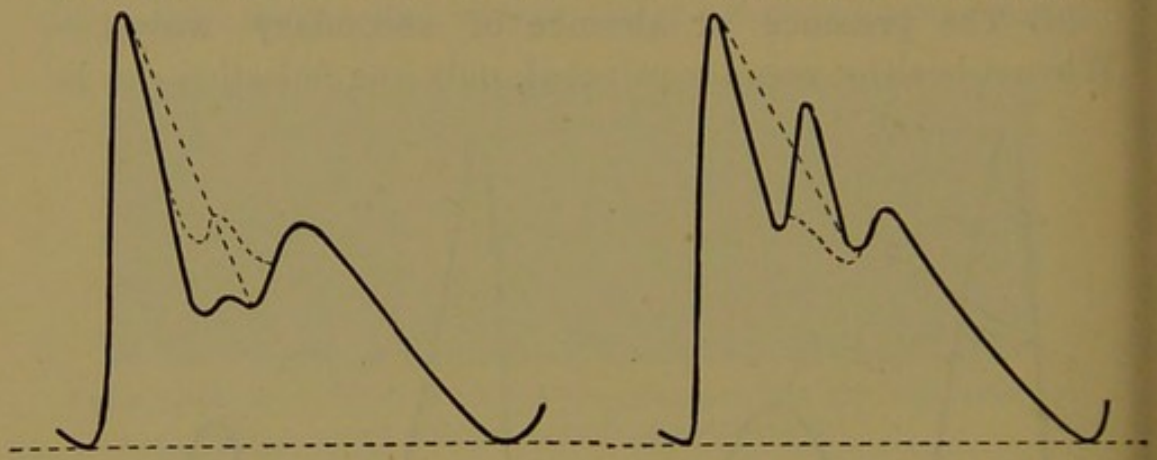


FIG. 47.—Diagram of pulse of low tension.

FIG. 48.—Diagram of pulse of high tension.

The finely dotted line represents the normal curve.

the heart, and the quantity of blood poured into the arterial system. When the sustained pressure within the vessel is high, the amount of expansion of the artery during cardiac systole is, as a rule, small. Sphygmographic tracings give a reliable means of estimating the sustained pressure. If a line be drawn from the apex of the tracing to the lowest point of the dicrotic notch, as in the above figures (Figs. 47, 48), it will be found that the tidal wave is sometimes below it, and at other times reaches or passes it. If it is below it, as in Fig. 47, the pulse is of low or moderate sustained pressure; but if, as in Fig. 48, it reaches as high or higher than the line, it indicates that the sustained pressure is high.

A pulse of high sustained pressure is met in chronic nephritis, atheroma, etc., when there is increased peripheral resistance to the outflow with an active and powerful ventricle. A pulse of low sustained pressure is found typically in the Corrigan's pulse of aortic regurgitation. The pulse of anæmia and wasting disease is also a good example of low sustained pressure.

5. **The Characters of the Pulse in different arteries in the same person** may require attention, in order to determine the presence of certain local interferences with the current through the arterial system. In similar arteries, on different sides of the body, the pulse under ordinary circumstances occurs at the same instant of time, and if there is a difference in time between the pulsation on the two sides, some obstacle is the cause of the delay in the later pulse. In the same way there is a definite interval of time between the pulse in arteries situated at different distances from the heart, between the carotid and radial, for instance, or between the radial and femoral or tibial. If the usual relation between the pulse in such vessels should be altered, some obstacle is here again the cause of the change.

When there is any delay in the pulse of a vessel, there is usually at the same time some change in the characters of the wave. In aneurysm of the arch of the aorta, for example, there may be a considerable difference in time and volume between the pulse of the right and left radial arteries.

The common causes of such local variations are abnormal distribution, or obliteration of vessels, atheroma, aneurysms, thrombosis, embolism, pressure of tumours, and injuries involving the course of the artery.

Capillaries.—A very interesting phenomenon is developed in the arterial capillaries in marked cases of aortic regurgitation. If the nails be examined, an alternate flushing and pallor may be seen. The phenomenon can also be produced by drawing the finger nail across the forehead of

the patient so as to produce a red line, when its alternate flushing and pallor is perceptible. This has been described as the *capillary pulse.*

The distention of the venous capillaries produces the condition known as cyanosis.

The Veins of the Neck.—The veins of the neck present extremely different appearances in stout and in thin people. In the former they can only be seen with difficulty; in the latter they stand out distinctly both in form and colour. It is necessary to pay attention to the condition of the jugular veins in regard to *fulness* and to *movements.*

The fulness of the jugular veins may be increased by such causes as thrombosis of one of the great veins nearer the heart, or pressure upon such a vessel by a tumour. In such a case the venous engorgement is for the most part unilateral. The fulness may, on the other hand, be augmented by such causes as the various dilatations and valvular lesions of the heart and many chronic lung affections. In cases of this kind the venous stasis affects both sides alike, and is but part of a disturbance of the circulation in general.

The movements of the veins depend upon two groups of causes—*respiratory and circulatory conditions.*

Movements caused by Respiration. — In thin persons undulations along the jugular veins, synchronous with the movement of respiration, may often be seen when the respiratory movements are exaggerated. These undulations consist in a gradual filling during forced expiration, and a sudden emptying of the veins at the beginning of inspiration. The explanation is obvious: the fulness during forced expiration is the result of the positive pressure on the veins of the thorax during that phase, which causes a retrograde wave of blood to close the valve above the jugular bulb; the sudden emptying attending inspiration is produced by the negative pressure within the thorax at that time, which draws the blood onwards in the direction of the heart.

Movements caused by Circulatory Conditions. — In

addition to these movements caused by respiratory conditions, there are others solely connected with the circulatory functions. These are grouped together under the term *venous pulse.*

The venous pulse in health consists of slight undulations which are invariably earlier in time than the apex-beat; the venous pulse is therefore, to use the common phrase, pre-systolic. This pulsation is only seen in very thin persons, and it is, as a rule, more visible in the external than in the internal jugular vein. It is produced by the contraction of the walls of the great veins and right auricle, which causes a

FIG. 49.—Tracing from the internal jugular vein in a case of tricuspid incompetence.

current to flow backwards as far as the valves on the veins. The blood current is stopped there, but the closure of the jugular valve communicates a distinct pulsation to the contents of the vein beyond, and every separate shock received by the valve is similarly transmitted to the blood above.

Tracings obtained from the external jugular vein show at times a single wave, caused by auricular systole. There may, however, be two impulses, in which case the second wave is produced by a shock transmitted backwards from the heart, during the closure of the auriculo-ventricular valves. At rare times there may even be a third wave, agreeing in time with the diastole of the heart, and no doubt communicated

to the veins by the shock caused by the closure of the arterial valves.

The venous pulse in diseased conditions, when the right side of the heart is engorged and the tricuspid valve incompetent, is essentially different. During its systole the auricle drives blood not only forwards into the ventricle, but backwards into the vena cava, and an auricular wave is produced. After the auricular systole there is a fall in the venous pressure, while the auricle is filling from the veins and through the incompetent tricuspid valve, the fall in pressure in the vein resulting in a so-called negative wave. This "negative wave" is interrupted as soon as the auricle is filled. If the auricle be filled before the ventricular systole is ended, a positive wave, the ventricular wave, will result in the vein. A sudden elevation in the end of this wave may be caused by the closure of the pulmonary valve momentarily accelerating the flow of blood backward through the tricuspid valve. When the ventricle passes into diastole, the blood flows from the auricle and veins into the ventricle, thus producing a second "negative wave" in the vein. Though this is the usual form of venous pulse in right-sided engorgement, when there is extreme backward pressure it may be modified. The relatively powerful right ventricle may drive the blood backward through the tricuspid valve under such pressure that the relatively weak auricle loses its power of impressing its action on the blood contained in its cavity. There is then no auricular wave in the tracing. A single large ventricular positive wave is followed by a single "negative wave."

CHAPTER VI

EXAMINATION OF THE BLOOD

Clinical examination of the blood is of the highest importance, and will at times give much aid in the elucidation of cases of doubtful diagnosis. A drop of blood for examination may be obtained from either the finger-tip or the lobe of the ear. The skin of the part selected is thoroughly washed with soap and water and carefully dried. The skin is then punctured with a clean lancet, the blade of which can be regulated in length, with a bayonet-pointed surgical needle or with a tenotomy knife. Before the lancet is used it is well to sterilise the blade with corrosive sublimate or alcohol. On no account must pressure be applied to the part to expedite the flow of blood, as by it the composition of the blood has been shown to be markedly altered by the flow of lymph from the surrounding tissues. When difficulty is experienced in obtaining sufficient blood from the finger, as in some cases of profound anæmia, it will frequently be found that the ear gives a better supply.

If comparative blood examinations are being made, they should be undertaken at the same hour of the day, and the hours of meals recorded, as digestion is known to have an influence on the number of leucocytes present.

The examination of the blood when fully carried out consists of—(1) Enumeration of the corpuscles, red and white. (2) Estimation of the colouring matter. (3) Microscopic examination of (*a*) fresh blood, (*b*) fixed and stained films.

(4) Estimation of the specific gravity. (5) Estimation of the alkalinity. (6) Determination of the coagulation time. (7) Examination for microparasites.

Enumeration of the Corpuscles is carried out by diluting the blood to a known extent, and counting the number of corpuscles present in a known quantity of the dilution. The Thoma-Zeiss hæmacytometer is the form of instrument now in general use for the enumeration of the corpuscles.

The instrument is exceedingly simple in design and easy to use. It consists of a graduated capillary tube, expanding into a bulb at the upper end, in which the blood is measured and diluted. In the bulb of the capillary tube is a glass ball for mixing the blood and diluting fluid. The counting chamber consists of a glass slide supporting a square glass cell with a central circular aperture. Within this is fixed a circular disc, upon which are ruled lines dividing the surface into microscopic squares (Fig. 50). When an accurately ground cover-glass is lowered over the chamber there is an interval of 0·1 mm. between the adjoining surfaces of the cover-glass and discs. The area of each square is $\frac{1}{400}$ square mm., the individual lines being $\frac{1}{20}$ mm. apart. The area over each square has thus a value of $\frac{1}{4000}$ c.mm. The small squares are divided into groups of sixteen by more thickly ruled double lines. The capillary tube is graduated by means of a scale beginning with 0·1, and is marked at three points by the figures 0·5, 1, and 101.

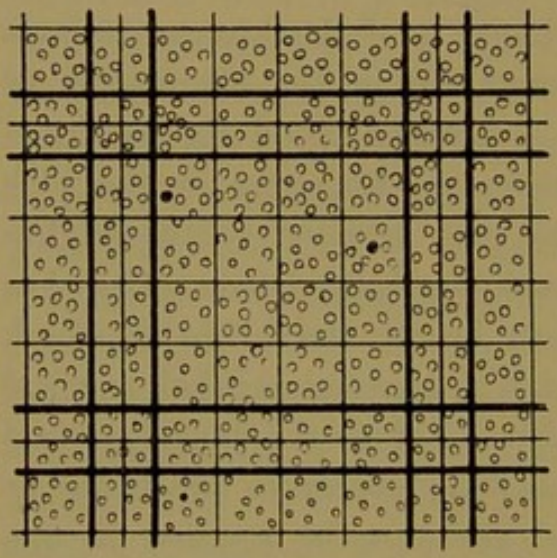


Fig. 50.

In the **enumeration of the red corpuscles** the instrument

is used in the following way. The blood, obtained by the puncture of a finger, as already described, is drawn up the capillary tube until it reaches either the point marked 0·5 or that marked 1, and the end of the instrument, after being wiped, is placed in the diluting fluid, which is drawn up by suction until it reaches the point marked 101. By shaking, the blood and diluting solutions are intimately mixed in the bulb by means of the little ball; a drop is blown out so as to empty the tube of that part of the diluting solution which has not been mixed with blood; another drop is placed in the cell, a cover-glass carefully placed over it, and the corpuscles enumerated with a moderately high power.

The corpuscles should be counted in a series of four sets of sixteen squares, and their average number per square ascertained. Then the estimation of the corpuscles depends upon two factors—(1) The dilution of the blood, which, if it has reached the point marked 0·5, will be 1 : 200, and if to the point marked 1, will be 1 : 100; (2) the cubic space corresponding to each square is $\frac{1}{4000}$ c.mm. The average number of corpuscles in each square must therefore be multiplied by 4000, and by 200 or 100 (according to the dilution), and the result gives the number of corpuscles in a cubic millimetre of blood. When the dilution is 1 : 200, as it usually is, the result may be obtained by multiplying the average number of corpuscles per square by 800,000. This gives the corpuscles per c.mm.

The diluting fluid used in the enumeration of the red corpuscles may be a normal saline solution (0·75 per cent. NaCl in distilled water), or a solution of sulphate of soda of a specific gravity of 1025.

Hayem's fluid, which has certain advantages, consists of—

Mercuric bichloride . .	0·25 grm.
Sodium chloride . . .	0·5 ,,
Sodium sulphate . . .	2·5 ,,
Distilled water . . .	100 c.c.

The addition of an aniline dye, preferably methyl-violet, to the fluid facilitates the recognition of the white corpuscles.

Sherrington recommends—

Ehrlich's purified methylene-blue	0·1 grm.
Sodium chloride . . .	1·2 „
Neutral potassium oxalate . .	1·2 „
Distilled water	300 c.c.

The number of red corpuscles normally present in the blood is estimated at 5,000,000 per c.mm. for man, and 10 per cent. less in women. In the condition known as "oligocythæmia" the number of the corpuscles may become very much diminished, sinking in severe cases of pernicious anæmia below a million per c.mm. The number falls temporarily after severe hæmorrhage. The number is diminished in leukæmia, chlorosis and in cachexias, as in phthisis and the various forms of malignant disease.

Enumeration of the white corpuscles is best carried out with the Thoma-Zeiss hæmacytometer and the special pipette, which gives a dilution of 1 in 20. With this dilution it is necessary to eliminate the chromocytes by rendering them invisible, as otherwise their number tends to obscure the leucocytes and renders the count difficult. The difficulty is overcome by using a 3 to 5 per cent. or even stronger solution of acetic acid tinted with methyl-green. The blood is "laked" by the acetic acid, the red corpuscles are rendered invisible, and the white corpuscles with their stained nuclei are easily recognised and enumerated. In enumerating, the whole cross-ruled space, that is 400 squares, should be counted. The number of corpuscles is calculated on the same principle as in the case of the chromocytes. Each of the 400 squares counted represents $\frac{1}{4000}$ c.mm. of diluted blood. The dilution used was 1 in 20. Let x represent the number of white corpuscles.

Thus $\frac{x}{400} \times 4000 \times 20$ gives the number of leucocytes per

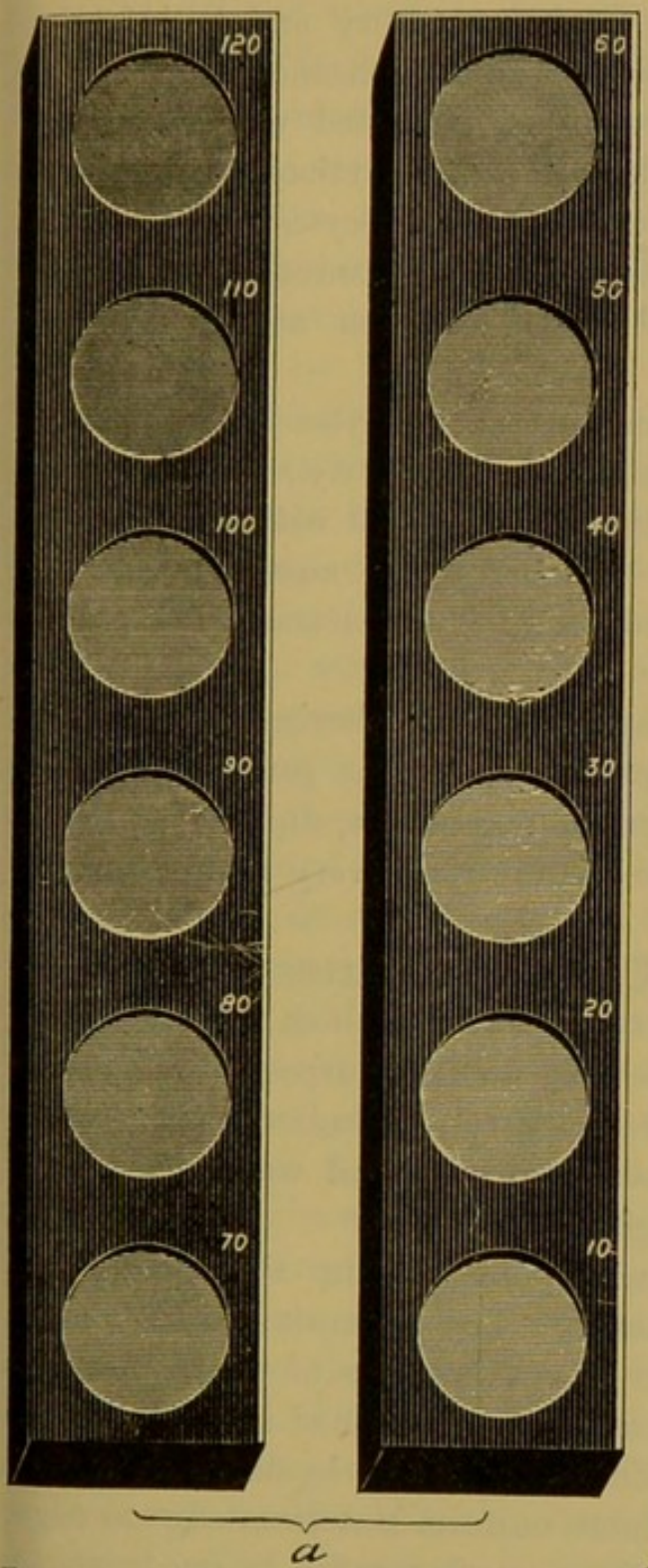


FIG. 51. — Oliver's hæmoglobinometer. *e*, Glass cell for receiving the blood from the pipette; the dilution is effected within the cell itself. *a*, Standard graduations made of tinted glass. The apparatus is shown of the natural size.

c.mm. The same result will be obtained if the total number of corpuscles counted be multiplied by 200.

The number of white corpuscles in 1 c.mm. of blood is usually stated at 7000 to 8000, but it varies greatly even in health. It is rare, however, for the number to rise above 12,000 or fall below 5000 without some abnormal condition being present.

Under normal conditions an increase may take place — a physiological leucocytosis, thus after a diet rich in proteids, during pregnancy and after parturition. Patholog-

ical leucocytosis occurs in inflammatory and in infective conditions, in toxæmia, as the result of hæmorrhage, and in cachectic conditions, especially as associated with malignant disease. In the disease known as leucocythæmia there is an enormous increase in the number of leucocytes present in the blood. Cases are recorded where the count has registered 1,000,000 per c.mm., but the common average in this disease is 400,000.

A diminution in the number of leucocytes—"leucopenia," is found in some infective conditions, *e.g.*, typhoid fever, malarial fever, tuberculosis uncomplicated with suppuration, measles and influenza. In pernicious anæmia, chlorosis, purpura and goître a reduction in the leucocytes to 3000 per c.mm. is common.

A fall in the leucocyte count, following a preceding leucocytosis, without improvement in the general condition, is a grave prognostic sign in pneumonia, diphtheria, septicæmia and allied conditions showing a very high degree of general poisoning.

Estimation of the Colouring Matter of the Blood may be carried out by a colorimetric method. A number of instruments have been devised for this purpose. With most methods it is essential to work in a standard light, with a reflecting surface of a standard tint, and with a means of cutting off extraneous light.

The instrument commonly in use in this country is **Gowers' hæmoglobinometer.** The apparatus consists of a capillary pipette and two glass tubes, one of which contains a standard colour solution, while the other is graduated so that 100° equal 2 c.c. The tubes should be flattened so that their contents present a more uniform tint from edge to edge. By fixing the tubes in their stand parallel to one another, but diagonally across the stand, instead of side by side, their adjacent edges will appear to overlap if looked at from the proper point, and thus the respective tints can be more accurately compared. The method is as follows. Some

drops of distilled water are put into the graduated tube, and to this is added 20 c.mm. of the blood to be examined, the quantity being measured in the pipette, and the two are quickly shaken together. Distilled water is then added drop by drop until the diluted blood is of the same shade as the standard colour solution, and the degree at which it stands is the percentage of hæmoglobin present, *i.e.*, if the diluted blood gives the standard colour at 60, the percentage of hæmoglobin is 60.

Haldane has introduced a modification of the colorimetric method which can be applied to the use of Gowers' hæmoglobinometer, and is exceedingly accurate. The standard colour solution consists of a dilute solution of blood of known oxygen capacity saturated with coal gas. This solution is sealed up in a narrow tube after all the contained air has been displaced with coal gas. When efficiently sealed the solution is permanent. In making the estimation, sufficient water is first added to the graduated test-tube of the hæmoglobinometer to dilute the blood as far as safety will allow. The blood is then measured and added. Before mixing, a piece of narrow rubber tubing, connected with a gas burner, is introduced into the free part of the tube and pushed down to near the level of the liquid, the gas being meanwhile turned on so that the air is displaced from the tube. The gas tube is withdrawn while the gas is still flowing and the top of the graduated tube is quickly closed with the finger. The liquid is then made to run up and down the tube about a dozen times, so that the hæmoglobin becomes thoroughly saturated with CO, and the full pink tint of CO-hæmoglobin appears. Water is then added drop by drop with the pipette until the tint of the graduated tube and the standard are equal. The percentage is read off after half a minute has been allowed for the liquid to run down. Another drop is now added, and if necessary another, till the tints appear unequal. The mean of the readings which give equality is taken as the correct result. Care must be taken that no liquid

is lost during the manipulations. If the graduated tube and liquid be warmed by the hand during the mixing it is apt to spurt; this can be avoided by holding the tube in a cloth. In comparing the tints of the two tubes it is best to hold them up against the light from the sky or from an opal glass globe when artificial light is used. The tubes must be repeatedly transposed from side to side during the comparison, otherwise error may arise. It is claimed by the author that the method is exceedingly accurate; that the standard is permanent, and that, as the pigment is the same in the two tubes, a standard light is not necessary.

The percentage of hæmoglobin usually increases or diminishes with an increase or diminution of the red cells, but this is not always the case. If there be a variation the determination of this variation is known as the "colour index." In a normal individual with 5,000,000 cells and 100 per cent. hæmoglobin the colour index is 1. If the hæmoglobin is diminished the colour index is less than 1. The count of the cells is reduced to a percentage, and, dividing this, into the hæmoglobin percentage. Thus if the corpuscles are normal in number, that is, 100 per cent., and the hæmoglobin is reduced to say 50, the colour index is $\frac{50}{100} = 0{\cdot}5$.

Say that the corpuscles are reduced to 2,000,000, that is, 40 per cent., if the hæmoglobin be 60 per cent. the colour index is $\frac{60}{40} = 1{\cdot}5$.

In chlorosis the reduction of hæmoglobin is great, and the colour index is low; in pernicious anæmia the colour index is high, — an important point as an aid in the recognition of the disease.

An instrument which will be found simple and easily mastered, and which will give accurate results in the estimation of hæmoglobin, is **Oliver's hæmoglobinometer.** It is an adaptation to hæmometric work of the tintometer, an instrument devised for estimating the true colour intensity of substances used in manufacturing processes. The instru-

ment consists of the following parts—(1) An automatic blood measure of a capacity of 5 c.mm., which can readily be filled by capillary attraction; (2) a mixing pipette with a rubber nozzle, which fits over the polished end of the blood measurer; (3) a blood cell of more than sufficient capacity to ensure the complete liberation of the hæmoglobin; (4) standard gradations of colour, circular discs fixed in two slabs, six in each, representing 10° of the scale, from 10 to 120 inclusive, and riders, small squares of tinted glass, for giving the degrees between each standard gradation; (5) a camera tube; (6) standard candles to give a uniform intensity of light. To use the instrument the bore of the blood measure is first dried by passing through it a thread, and the polished point is then brought in contact with the drop of blood; thus the pipette fills automatically. The pipette being filled with an unbroken column of blood, any surplus is wiped away from the polished end with the finger. The mixing pipette being charged with water, its rubber end is adjusted to the polished end of the pipette and the blood washed into the cell by forcing the water through the pipette drop by drop. The cell is then accurately filled with water, the contents being thoroughly mixed. The cover-glass is next adjusted, the presence of a small bubble of air under the glass showing that the cell has not been over-filled. The cell is then placed by the side of the colour gradations, riders being used when necessary to obtain an exact equality in the colour tint. The observation may be made with the daylight standard, but more accurate results will be got by working in a dark room with standard candles placed so as to give a high light, the flame being 3 or 4 in. above the cells. If the observer's eye be strained by observing the colour tints for some time relief will be got by looking at the inside of the lid of the instrument case, which is lined with green morocco, complementary in colour to the blood and the colour standards.

This form of hæmoglobinometer possesses many advantages

over Gowers'. It is more accurate, is easily worked when once mastered, and demands a much smaller quantity of blood, a by no means unimportant point when dealing with anæmic patients.

Microscopic Examination of the Blood.—In the microscopic examination of the blood one of the primary essentials is to obtain clean cover-glasses. To attain this end the cover-slips, which should be the thinnest possible, may be washed in a strong solution of a mineral acid, then in running water, and stored in a stoppered bottle in a mixture of alcohol and ether. Before use they are dried, by being passed through the flame of a spirit lamp or bunsen, or with a fine linen cloth. Another method strongly recommended is to boil the cover-slips in a mixture of concentrated sulphuric acid 6 parts, bichromate of potash 6 parts, and water 100 parts; wash thoroughly in running water, and then store in alcohol in a wide-mouthed stoppered bottle. Before use, the cover-slips are dried by passing them through the flame of a spirit lamp. Whichever method of preparation be used the cover-slips should never be touched with the fingers after preparation. Forceps alone should be used. The part from which the drop of blood is to be taken should be cleansed with soap and water and dried. The part selected is pricked, and the cover-slip is carefully lowered by means of forceps on to the top of the drop of blood, care being taken that too large an amount of blood does not adhere to the glass. If there be too free bleeding from the puncture, the platinum loop used in bacteriological work will be found useful in giving a drop of the desired amount.

(*a*) **Examination of a Fresh Film.**—A drop of blood having been secured the cover-slip is gently lowered on to a clean slide, and observed with the high power. If a warm stage be used, or if the slide and cover be gently warmed before use, the active amœboid movements of the leucocytes will be noted. At the ordinary temperature the observer

will note the formation of rouleaux, the shape and size of the corpuscles, the presence of blood plates, and will gain an idea of the relative number of white corpuscles.

The average size of the healthy red corpuscle or erythrocyte is 7·5 μ, rarely are smaller or larger forms met with in healthy blood. In diseased conditions, however, there are great variations in the size and in the shape of the corpuscles.

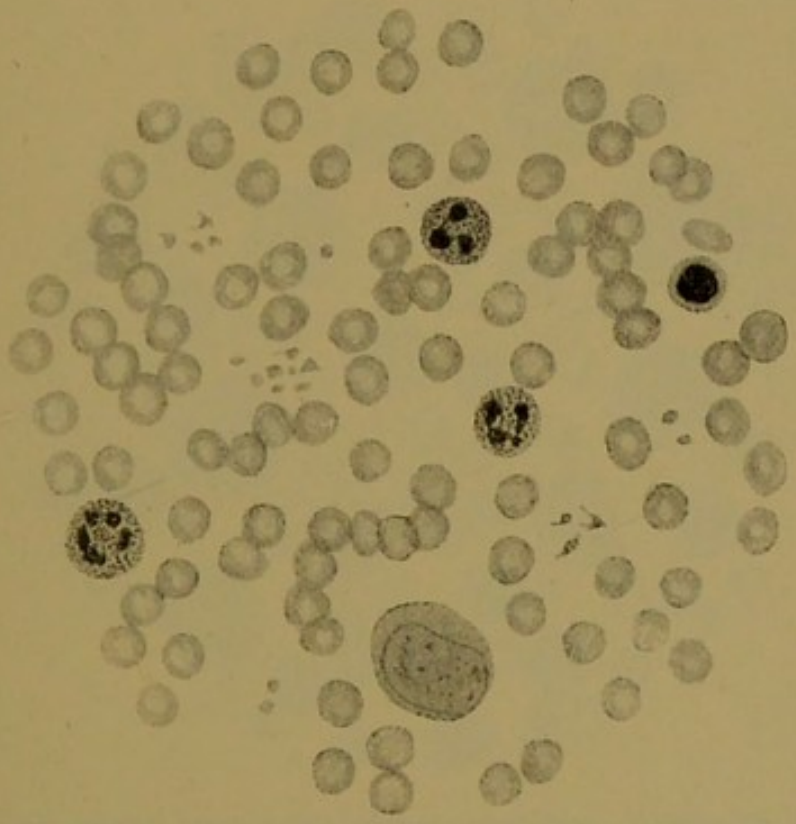


Fig. 52.—Normal blood film. × 500.

This is especially well marked in anæmia, and is known as *poikilocytosis*. Cells of normal size are then seen along with large and with small cells, and there may be marked alteration in shape, some of the cells having become pear-shaped, some kidney-shaped, and some oval. The large cells are known as *megalocytes*, the small cells as *microcytes*.

In working with the fresh blood it sometimes is desirable to fix the corpuscles before examination. This can be done by placing on the point to be punctured a drop of a 2 per

cent. solution of osmic acid. The corpuscles are fixed by the osmic acid as they exude, and can be observed without any alteration in their shape from influences outside the body.

(*b*) **The Fixing and Staining of Films.**—Films may be made on slides, but are more easily manipulated if square cover-slips be used. The cover-slip is taken out of the stock preserved in alcohol, dried by passing through the flame of a spirit lamp or with a linen cloth, and a series is propped

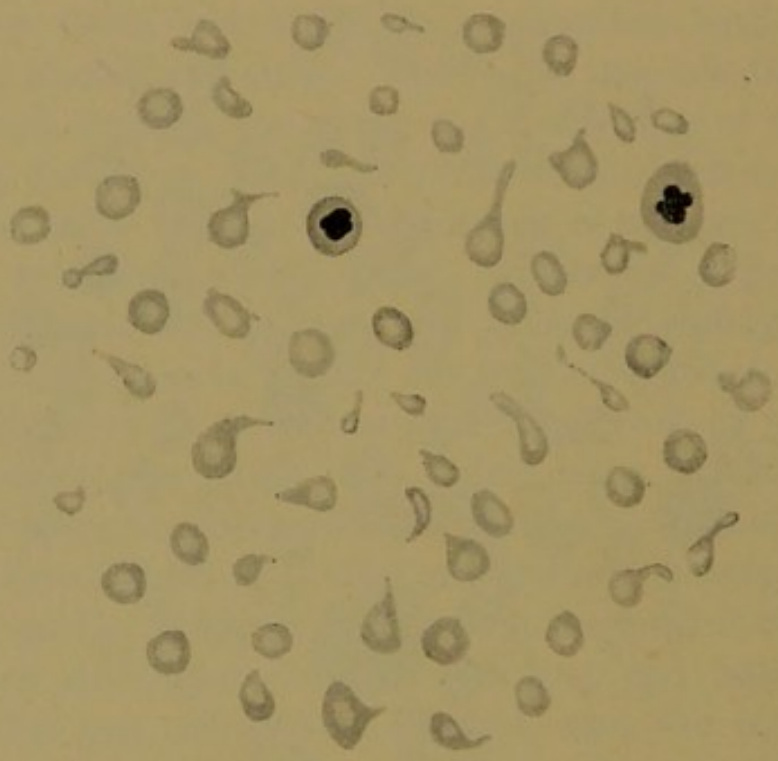


Fig. 53.—Blood– pernicious anæmia. Film shows poikilocytosis and nucleated red corpuscles. × 500.

up in a row, so that they can be easily grasped with the forceps or laid in a row on the edge of a clean sheet of paper. The part is then prepared, pricked, and if a small drop of blood exude a cover-slip grasped in the forceps is lowered upon the apex of the drop. A second cover-slip is then taken with forceps and lowered upon the first, so that the drop of blood is evenly spread out into a fine layer between the two slips. In lowering the second cover-slip the corners of the two should not coincide, but should be placed diago-

nally to one another. The blood being spread out into a thin film, the slips are gently but quickly *slid* apart, and the films are allowed to dry. The film having been made has next to be fixed. This may be done by heat or by reagents.

Fixation by heat is best carried out by placing the cover-slips in a dry oven or hot air steriliser, at a temperature of

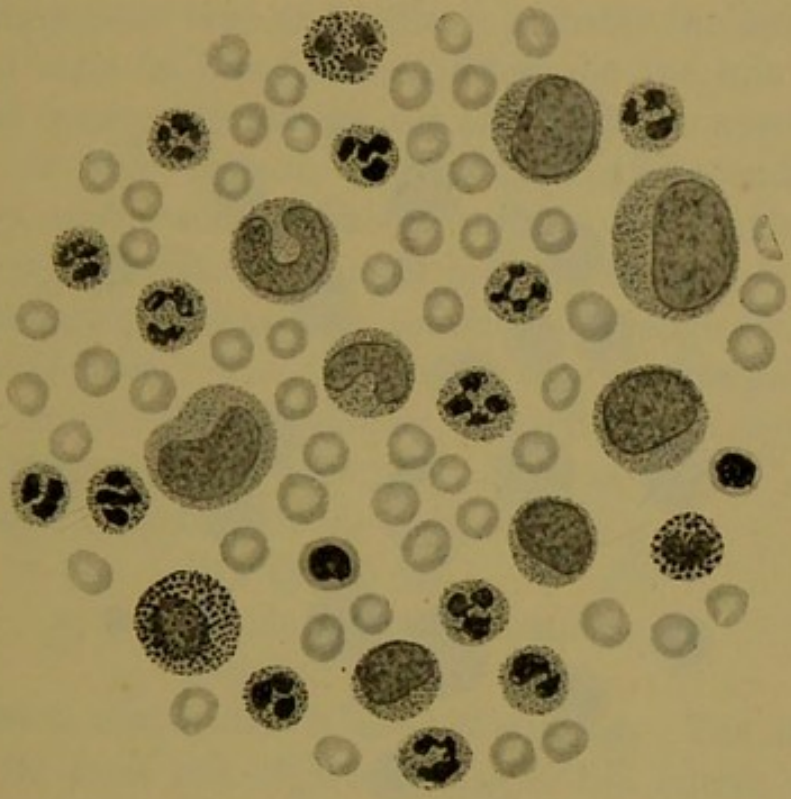


Fig. 54.—Blood—spleno-medullary leucocythæmia. Film shows large uninucleated cells—myelocytes, and an enormously increased proportion of leucocytes. × 500.

110° to 115° C. for fifteen to twenty minutes. In clinical work this method is not always available. Placing the covers in an ordinary paraffin oven for twelve to twenty-four hours will be found an easy and efficient method of fixation.

Fixation by reagents is certainly the more convenient method in clinical work.

The method to be employed is as follows :—

1. Dry film.

2. Fix for three to five minutes, but not longer, in formalin-alcohol solution (Gulland). Formalin commercial, 10 per cent.; absolute alcohol, 90 per cent.

The cover-slip is floated on the solution film downwards, the glass in which the solution is contained being covered to prevent evaporation.

3. Wash in tap water. The film is then ready for staining.

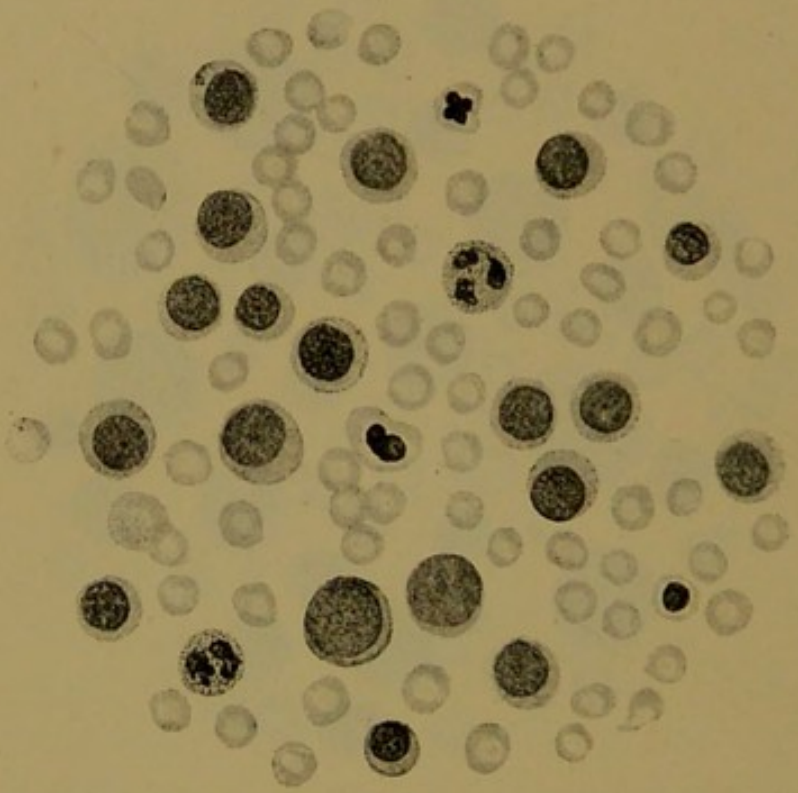


FIG. 55.—Blood—lymphatic leucocythæmia. Film shows great increase in lymphocytes, large and small. × 500.

The sublimate and alcohol methods of Muir and Gulland, while preferable for histological work, are too elaborate for general clinical use.

Staining the Film.—Stains in histological work are divided into two groups, "acid" and "basic." The acid stains, though chemically neutral in reaction, are so called because the staining principle is the acid radical of the compound,—the stain, in fact, from a tintorial point of view, acts as a free acid. The most common "acid" stains in use

are eosin, aurantia and orange G. The "basic" stains are so called because the active staining principle of the compound is the base,—for example, fuchsin, methylene-blue, methyl-violet and safranin. Both acid and basic stains are used in staining blood films, for certain of the cell granules react to acid stains only, and are therefore termed "oxyphil" granules, while others are more readily stained with basic dyes, and are termed "basophil."

(1) **Methylene-blue and eosin** is probably the most useful all-round stain for blood films. Stain for one minute with saturated watery solution of eosin. Wash in water, dry in the air or over bunsen, stain for thirty seconds or as necessary, with saturated watery solution of methylene-blue, wash, dry and mount in Canada balsam dissolved in xylol.

(2) **Ehrlich's triple stain** consists of orange G. fuchsin and methyl-green. The ingredients can be obtained mixed in the required proportions as Ehrlich-Biondi powder or Ehrlich-Heidenhain powder.

The stain is then made up as follows :—

Powder, 15 grs.
Absolute alcohol, 1 c.c.
Distilled water, 6 c.c.

The stain made thus is used undiluted. The films are stained for from one to five minutes, the length of time depending upon the activity of the individual stain and the characters of the film under examination. The film being stained is washed, dried and mounted. The nuclei of the leucocytes are found stained a greenish-blue; oxyphil granules are red and basophil granules are unstained. The so-called neutrophil granules are purple.

(3) **Hæmatoxylin and Eosin** is a useful stain to give a general impression of the character of a film, and is easily worked. It is used in the same way as the methyl-blue and eosin, the eosin used first, and the film then stained with hæmatoxylin, which should be Ehrlich's. The time of staining with the hæmatoxylin varies with the age of the

solution, but should be so arranged that a wash with tap water turns the nuclei of the leucocytes blue. The film should then be dried and mounted in balsam. This procedure gives a good result if it is desired to stain the malaria parasite.

Examination of stained films.—In a satisfactory preparation the corpuscles are found evenly spread out, no rouleaux are present, and the red corpuscles have been fixed after having regained their original shape. For observation of the finer structure of the corpuscles, an oil immersion lens ($\frac{1}{12}$) is necessary, but the student will get satisfactory results in differential counts with a magnification of 500. In normal blood in an eosin stained film the *red corpuscles* have a pure red tint, and the normal biconcavity is visible. In anæmia, in addition to an alteration in the form and size, there are alterations in the staining properties. A diminution in the amount of hæmoglobin in the corpuscles leads to diminished power of staining with eosin, and the corpuscles show varying degrees of a paler pink colour. Some cells may show the normal biconcavity — the pale centre, but others give a uniform, or almost uniform, staining throughout. In hæmatoxylin-eosin stained films, in health the eosin alone is taken up by the red corpuscles, but in anæmia, especially of the pernicious type, the hæmatoxylin may diffuse into the red cells, giving them a bluish tinge. These corpuscles are spoken of as "polychromatophil." In diseased conditions nucleated red corpuscles may appear in the blood, passing from the bone marrow, where they are normally found. Three types are distinguished—(*a*) The *normoblast*, the precursor of the normal red cell, has usually a single well-defined nucleus, which stains deeply and uniformly, and is very frequently placed somewhat eccentrically in the cell. (*b*) The *megaloblast*, a very large cell, with a nucleus which stains less deeply than in the case of the normoblast. (*c*) The *microblast*, a small nucleated cell, is rare, and its exact pathological significance is not at present known.

The white blood Corpuscles.—In stained preparations very important diagnostic information can be obtained by a study of the white blood corpuscles. The different cells may be divided into lymphocytes, eosinophilous cells and basophilous cells.

(1) The *small lymphocyte* is a cell about the size of a normal red corpuscle, with a large round nucleus, which stains fairly homogeneously with nuclear dyes. The zone of protoplasm around the nucleus is very small in amount and difficult to distinguish.

(2) The *large lymphocyte* is twice or three times the size of the red blood corpuscle. It contains a rounded nucleus, which may be indented or horse-shoe shaped, is poor in chromatin and does not stain well with aniline dyes.

(3) The *polymorphonuclear finely granular eosinophilous* or *neutrophil leucocyte* forms a large proportion of the white corpuscles. It is a large cell, having an average diameter of 10 μ. The cell substance contains a large number of fine granules, which are faintly oxyphil. The nucleus is irregular and multipartite, and frequently appears to consist of separate lobes linked together by chromatin threads.

(4) The *coarsely granular oxyphil leucocyte* is characterised by the large size of the granules, which stain homogeneously with acid dyes such as eosin. The nucleus is polymorphic.

(5) The *finely granular basophil leucocyte*—the "*mast-zelle*" of Ehrlich—is a comparatively infrequent form in healthy blood. The nucleus is irregular in shape and poor in chromatin. The protoplasm contains fine granules, which stain violet with methylene-blue.

In examining a film a differential count of the different forms of leucocytes should be made. The count is much simplified by using a movable stage. Begin in the right-hand corner of the preparation, and pass across to the left; then move the specimen forwards so that a fresh field comes into view, and pass back from left to right. In this way the whole preparation can be gone over, and the

different varieties of leucocytes noted. Count five hundred leucocytes, and calculate the percentage of the different varieties.

In healthy adult blood the relative proportion of the different forms of leucocytes is on an average—

Small lymphocytes	20–25 per cent.
Large lymphocytes and transitional forms .	1– 2 ,,
Polymorphonuclear leucocytes . . .	70–75 ,,
Coarsely granular oxyphil leucocytes . .	1– 4 ,,
Finely granular basophil leucocytes . .	0·5 ,,

In disease there is an alteration in the relative proportion of the different forms. Polymorphonuclear or ordinary leucocytosis occurs in inflammatory or infective conditions, toxæmias, post-hæmorrhagic and cachectic conditions, especially where the cachexia is associated with malignant disease.

The lymphocytes are proportionally increased in malignant disease affecting the lymphoid tissue, in glandular enlargement, gastric and intestinal catarrh and in whooping-cough. A coarsely granular oxyphil leucocytosis is found in asthma, certain skin diseases, *e.g.*, pemphigus, urticaria, psoriasis, etc., and parasitic affections such as trichiniasis. In leucocythæmia an enormous increase occurs in the leucocytes present in the blood. In the lymphatic form the lymphocytes predominate—the condition known as lymphæmia. These cells may then reach the enormous proportion of 99 per cent., and either the large or small cells may predominate. The myelogenous form of the disease is characterised by the presence in the blood of a cell found normally only in the bone marrow—the myelocyte. It is a large cell containing a single nucleus, which stains faintly and with fine granules in the protoplasm. In this form of leucocythæmia, known as myelæmia, these cells form usually 30 per cent. of the total leucocytes present. The oxyphil myelocyte is a large cell similar to the neutrophil form, save that the granules are coarse and have an affinity for acid stains.

Blood plates are more or less rounded bodies of about 3 μ in diameter. They are difficult to study in films, on account of the fact that they are apt to adhere to the corpuscles or run into clumps. They are rich in phosphorus. They are best studied by diluting the blood with a mixture of glycerin and 2 per cent. chloride of sodium, in equal parts, saturated with a stain such as dahlia. Their number can then be estimated in relation to the red corpuscles by using $\frac{1}{12}$ immersion lens. Normally their proportional number is 1 to $8\frac{1}{2}$ red corpuscles. The significance of variations is not known.

Estimation of the Specific Gravity of the Blood is best carried out by the benzol-chloroform method. Take a mixture of equal parts of benzol and chloroform in a urine glass. A drop of blood is then taken up with a capillary tube, and blown on to the surface of the mixture. If the globule sinks more chloroform is added, if it floats benzol should be added. After each addition the fluid is fully mixed with a glass rod. When the drop of blood is just held in suspension in the fluid, neither rising nor falling, the proper density has been reached. The specific gravity of the fluid is then ascertained with a urinometer, which must be graduated up to 1060, or by means of the picnometer. The specific gravity of the blood is thus obtained.

The normal specific gravity of the blood is 1057–1060. The specific gravity is found to run parallel with the hæmoglobin percentage, and thus variations will give an idea of the hæmoglobin value. There is a fall in the specific gravity in cardiac disease with dropsy.

Estimation of the Alkalinity of the Blood is a delicate and, by most methods, a somewhat tedious operation. The most accurate is probably Wright's method, for which the reader is referred to the original paper.

Estimation of the Coagulation Time by Wright's Coagulometer.—The instrument consists of a series of tubes, which are kept warm by fixing in a jacket around a hot-water tin. The tubes being warmed to blood heat,

some blood is taken up into each of a series of five tubes, half a minute being allowed between the filling of tubes 1 and 2. The blood must be aspirated a little way up the tubes, to prevent drying at the orifice. After three minutes blow down No. 1, to see if the blood has coagulated; if not, wait. If it is coagulated, proceed along the series till a tube is found where coagulation has not taken place. By this method one obtains—(1) The shortest time required for coagulation; (2) the longest time the blood can remain uncoagulated. The means between these two will give an approximately accurate figure for the coagulation time. In such diseases as hæmophilia the coagulation time is much lengthened.

Microparasites of the Blood.—The spirillum of relapsing fever and the *Filaria sanguinis* can be demonstrated in the fresh blood.

The adult **filaria** have their habitat in the tissues and lymphatics, the embryos alone passing into the circulation. The embryo is a minute, colourless, snake-like organism measuring $\frac{1}{80}$ in. in length, and with a diameter approximately about that of a red blood corpuscle. When fresh it is seen to be constantly in motion, wriggling about and displacing the corpuscles.

The *Filaria nocturna* is, as a rule, only present in the blood during the night, when vast numbers can be observed. The *Filaria diurna* is present by day, and disappears during the night. It has the same anatomical appearance as *Filaria nocturna*. The *Filaria perstans* observes no periodicity. It is smaller than the *Filaria nocturna* and *diurna*.

The **plasmodium malariæ**, the parasite of malaria fever, can be recognised in the fresh state. Take a very small drop of blood upon a *clean* cover-slip, let it fall face downward upon a clean slide. The blood should then spread out into a thin film without the formation of rouleaux. Examine with a $\frac{1}{12}$ immersion lens. If the blood has been obtained shortly before the onset of the characteristic periodic attack

the parasite is seen as a pale, ill-defined disc of protoplasm, occupying a larger or smaller area in a proportion of the red blood corpuscles. Scattered through the pale body are black particles of melanin. The parasite from this stage goes through a process of evolution during the attack. The

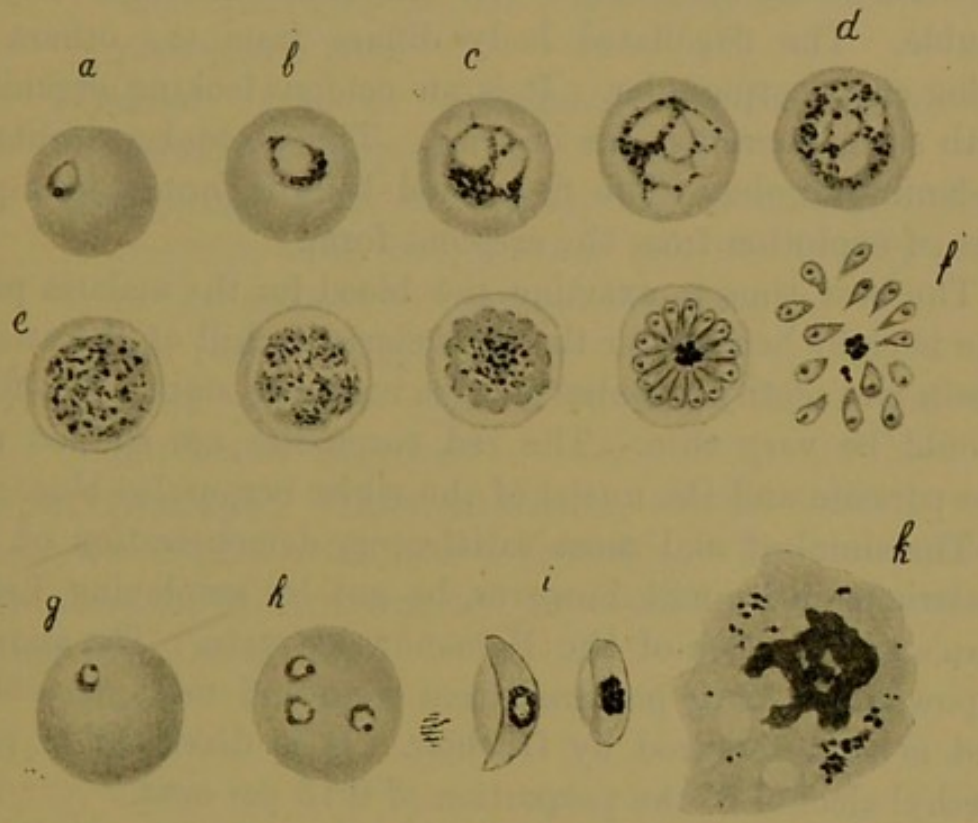


FIG. 56.—Varieties of the asexual forms of the simple tertian malarial parasites found in the peripheral blood.

a. Small non-pigmented intracorpuscular parasite.
b. Small pigmented form.
c–d. Large pigmented form.
e–f. Sporulating forms.
g. Small ringed form.
h. Small ringed form, multiple infection of corpuscle.
i. Crescentic forms.
k. Large mononuclear leucocyte with malarial pigment.

melanin particles collect into groups, which aggregate towards the centre of the protoplasm, which itself becomes segmented; spores are formed. The blood corpuscle then breaks down and the spores are set free. A portion of those find their way into a fresh series of red corpuscles. In the interior of the new corpuscle the parasite shows active amœboid move-

ments. As it develops and increases in size the movements diminish, till it reaches the sporulation stage again. In chronic cases the parasite may in addition be found in the crescent form—a highly refractile body, longer than a blood corpuscle, and about 2 μ in diameter, which contains melanin particles about the centre. At times the crescents may be double. The flagellated body differs from the others by being extra-corpuscular. It is an octopus-looking organism, with six or more flagellæ or arms. The protoplasm contains melanin granules. The flagellated body is formed by a process of evolution from the crescent form.

The best time to examine the blood for the malaria parasite is eight hours after the characteristic chill of the attack. Eosin and methylene-blue may be used as a stain. The films should be very thin. The red corpuscles are stained red, the parasite and the nuclei of the white corpuscles blue.

The simplest and most satisfactory demonstration of the malaria parasite will, however, be got by employing Leishman's modification of the Romanowsky stain. The stain is a powder which is prepared from eosin and methylene-blue, and is manufactured by Grübler. It is dissolved in pure methyl-alcohol in the proportion of 0·15 per cent.

Films having been prepared in the usual way, care being taken that the film is thin and even, are allowed to dry in the air. The cover-slip is then taken up in Cornet's forceps and three or four drops of the stain are allowed to fall upon the film. No attempt is made to check evaporation. The alcohol fixes the film. After half a minute double the quantity of distilled water—that is, six to eight drops—is added and mixed by gently rotating the forceps from side to side. The film is now allowed to stain for five minutes, and then gently washed in distilled water, and a few drops of distilled water allowed to rest on the film for one minute, at the end of which time it is ready and can be examined, either in water or after drying, and mounting in xylol balsam.

The film shows :—

Red corpuscles—pale pink or greenish tint, semi-transparent.

Polymorphonuclear leucocytes — nuclear network stained a deep ruby-red, protoplasm colourless, fine eosinophil granules red ; lymphocytes—nuclei red, protoplasm blue.

Coarsely granular eosinophils — nuclei ruby-red, granules pale pink.

Basophils—granules very densely stained, of a deep purplish-black tint, nucleus red, but usually more or less masked by granules overlying it.

Blood plates—ruby-red.

Malaria parasite — the body stains blue, and its chromatin ruby-red.

Spectroscopic Examination. — Both arterial and venous blood give a distinctive spectrum. The recognition of blood by the spectroscope will be referred to in the section dealing with the urine.

CHAPTER VII

RESPIRATORY SYSTEM

Thorax—Surface Lines and Areas

For convenience in description, the thorax is regarded as presenting several areas, and marked by six vertical lines.

The lines are—(*a*) The mammary line, a vertical line drawn from a point half-way between the inner end of the clavicle and the end of the acromion process to another point half-way between the pubic and iliac spines; (*b*) the parasternal line, drawn midway between the preceding and the mid-sternal line; (*c*) the anterior axillary line, drawn downwards from the anterior fold of the axilla; (*d*) the mid-axillary line, drawn from the centre of the axilla; (*e*) the posterior axillary line, from the posterior fold of the axilla; and (*f*) the scapular line, drawn through the angle of the scapula.

The areas are anteriorly—(*a*) The supraclavicular, above the clavicle; (*b*) the infraclavicular, from the clavicle to the third rib; and (*c*) the mammary, from the third rib downwards.

Posteriorly they are—(*a*) Suprascapular, above the scapula; (*b*) the scapular, which may be divided into a supra- and infra-spinous; (*c*) the interscapular; and (*d*) the infra-scapular.

Laterally they are the axillary and infra-axillary.

They are shown, along with the abdominal regions, in Figs. 57 and 58.

Anatomical Relations of the Lungs

The apices of the lungs rise anteriorly to an inch or an inch and a half above the clavicles; posteriorly they reach

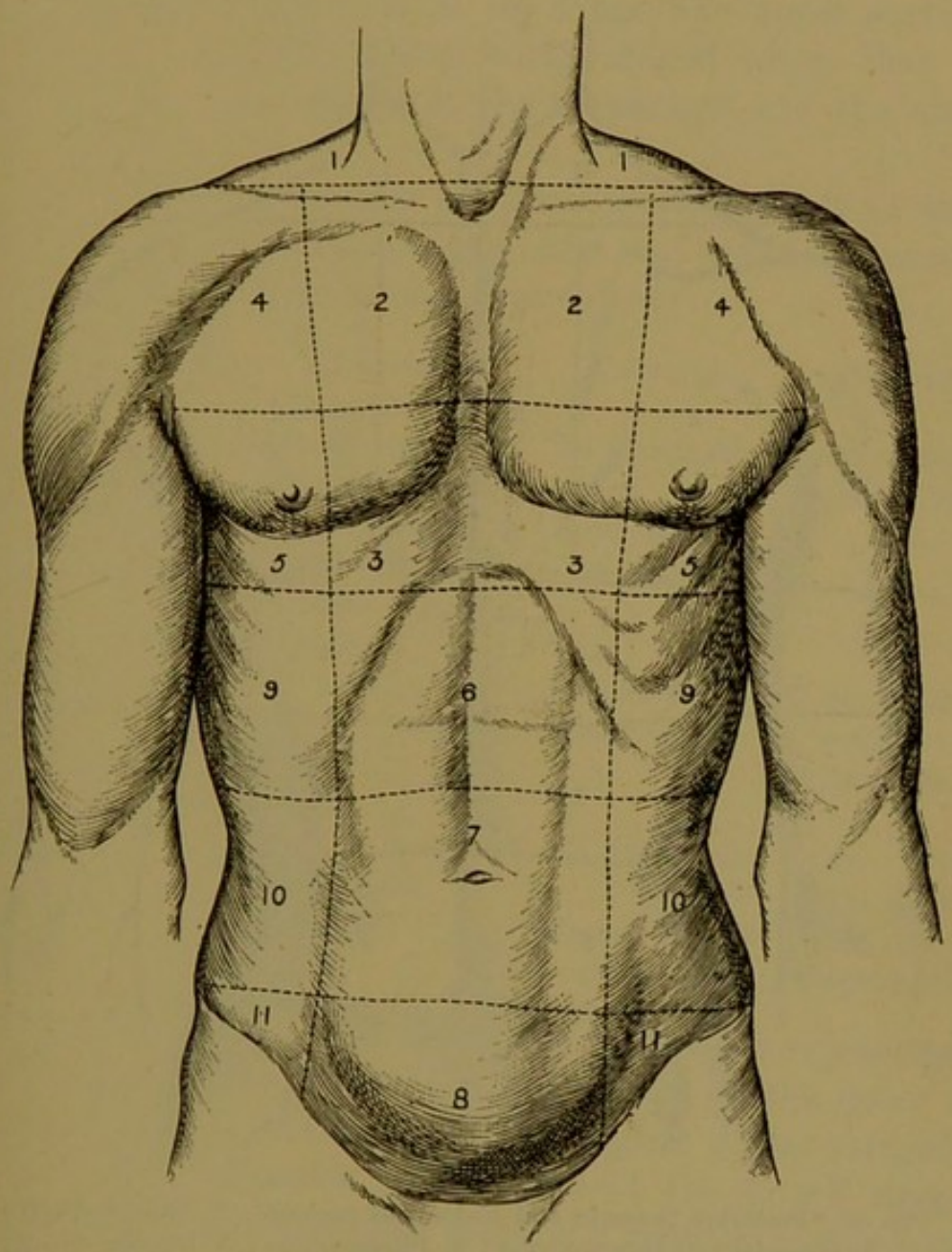


Fig. 57.—Anterior thoracic and abdominal regions. 1. Supraclavicular; 2. Infraclavicular; 3. Mammary; 4. Axillary; 5. Infra-axillary; 6. Epigastric; 7. Umbilical; 8. Hypogastric; 9. Hypochondriac (right and left); 10. Iliac (right and left); 11. Inguinal (right and left).

the level of the seventh cervical vertebra. The base or inferior limit of both lungs is at the level of the sixth rib in

the mammary line, the eighth rib in the axillary line, the ninth rib in the scapular line, and the tenth rib close to the spine. The anterior margins of the lungs are, during inspiration, in contact under the sternum from the second to

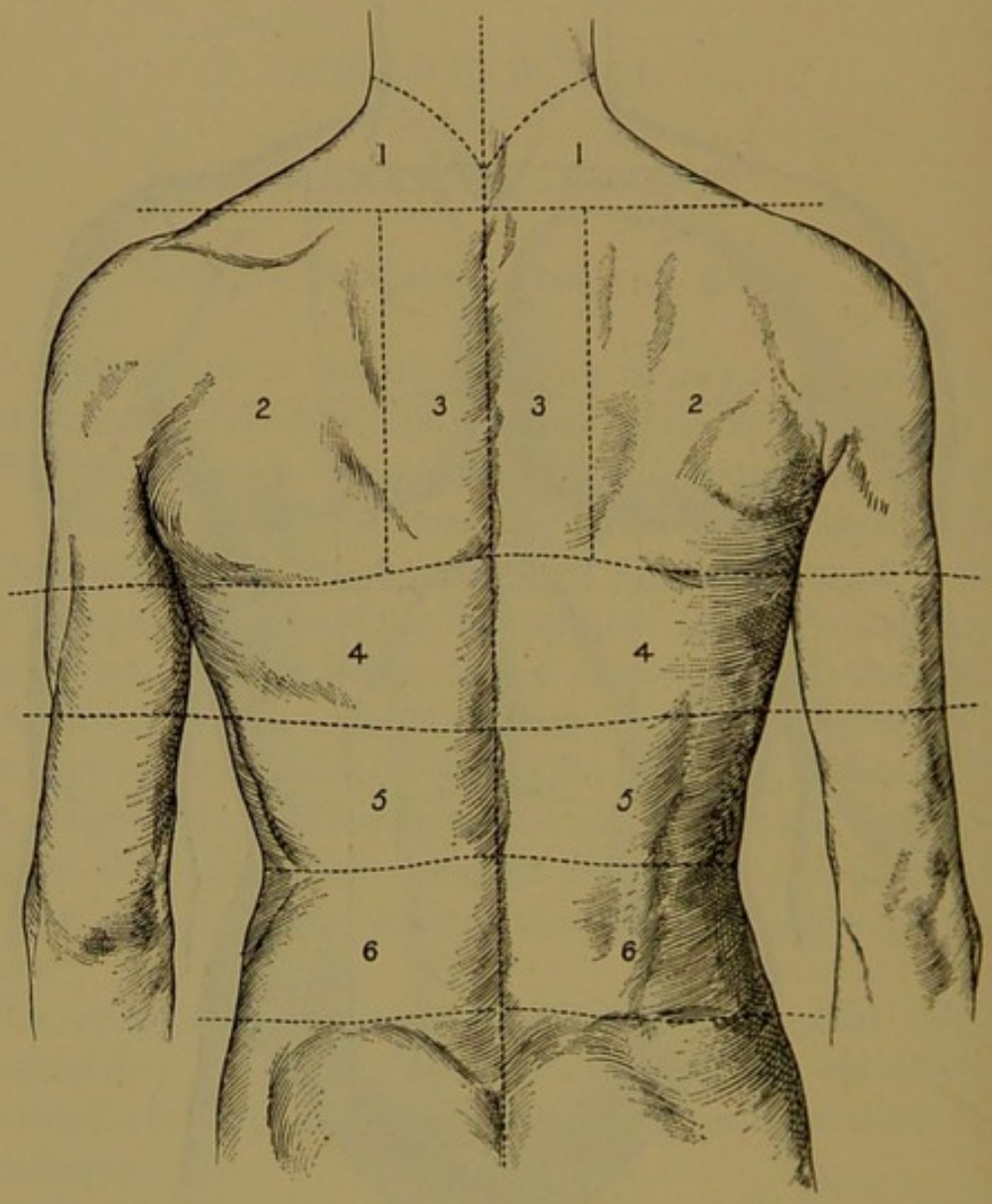


Fig. 58.—Posterior thoracic and abdominal regions. 1. Suprascapular; 2. Scapular; 3. Interscapular; 4. Infrascapular; 5. Inferior dorsal; 6. Lumbar.

the fourth rib. At the level of the fourth rib the edge of the left lung turns outwards and downwards, forming a notch, with its concavity directed to the middle line; the inferior limit of the notch joins the inferior edge of the lung

at the sixth rib about the mammary line. This notch leaves a considerable part of the anterior surface of the pericardium in immediate contact with the internal surface of the chest wall. The bases of the lungs rest on the diaphragm, and are somewhat concave to fit its dome-like upper surface, while their margins become wedge-shaped where they lie between the outer aspect of the diaphragm and the chest wall.

The right lung consists of three lobes, the left of two. The relations of the lobes to the chest wall are of importance clinically, and may be tabulated as follows :—

	Anteriorly (*Mammary line*).	*Laterally.*	*Posteriorly.*
RIGHT LUNG, 3 lobes.			
Upper lobe . .	From apex to fourth or fifth rib.	To fourth rib.	From apex to spine of scapula.
Middle lobe . .	From fourth or fifth rib to inferior margin of lung.	From fourth to sixth rib.	*Nil.*
Lower lobe . .	*Nil.*	From sixth to eighth rib.	From spine of scapula to tenth rib.
LEFT LUNG, 2 lobes.			
Upper lobe . .	From apex to sixth rib.	To fourth rib.	From apex to spine of scapula.
Lower lobe . .	*Nil.*	From fourth rib to base.	From spine of scapula to base.

The relations of the lungs to the surface are represented in the frontispieces I. and II. The summit of the dome of the diaphragm is on a level with the eighth dorsal vertebra. The trachea bifurcates at the level of the fourth dorsal vertebra, the right bronchus being at a slightly higher level than the left.

PHYSICAL EXAMINATION OF THE LUNGS

The physical examination of the lungs comprises four distinct methods, namely—(1) Inspection, including men-

suration; (2) palpation, including vocal fremitus; (3) percussion, including sense of resistance; (4) auscultation, including vocal resonance. We shall take these up seriatim.

Inspection and Mensuration

Preliminary.—Normal respiration is made up of two phases, inspiration and expiration, followed by a pause. Their relative length may be put in the form of an equation, as follows—

Inspiration : Expiration : : 5 : 6

Expansion.—During inspiration the cavity of the chest enlarges, and the lungs expand, while during expiration it diminishes and the lungs contract. The enlargement of the chest consists in an increase in its circumference, as well as an increase in its vertical capacity, the latter being brought about by the descent of the diaphragm.

The increase in the circumference is recognised by the eye, and is technically known as *expansion*. The degree of expansion varies in different individuals.

The descent of the diaphragm during inspiration is recognised by the distention of the upper part of the abdomen, while during expiration the abdomen falls as the diaphragm rises.

In *men*, when the breathing is quiet, it should be noted that the upper part of the thorax moves very slightly, while its lower part and the upper part of the abdomen move distinctly and regularly; this type of breathing is known as *costo-abdominal*. When, however, the breathing becomes accelerated, or if deeper respirations are taken, the upper part of the thorax also moves; this is more especially the case in conditions in which the respirations are deep rather than simply increased in rapidity. In women, on the other hand, the respiratory movement is confined mainly to the thorax, the upper part moving distinctly even

during quiet respiration; this type of breathing is known as *thoracic*.

The frequency of the respiratory movements varies from fourteen to eighteen per minute. Their rate should always be noted.

Inspection may be considered under two heads—

1. Form of the chest.
2. Movement.

The Form of the Chest.—Contour outlines of the chest are made by means of the *cyrtometer*—two long pieces of soft metal united by a leather hinge form a fairly efficient apparatus. The hinge is placed over the spine, the pieces are moulded to the wall of the chest, removed by means of the hinge without altering the shape, and the contour traced on paper.

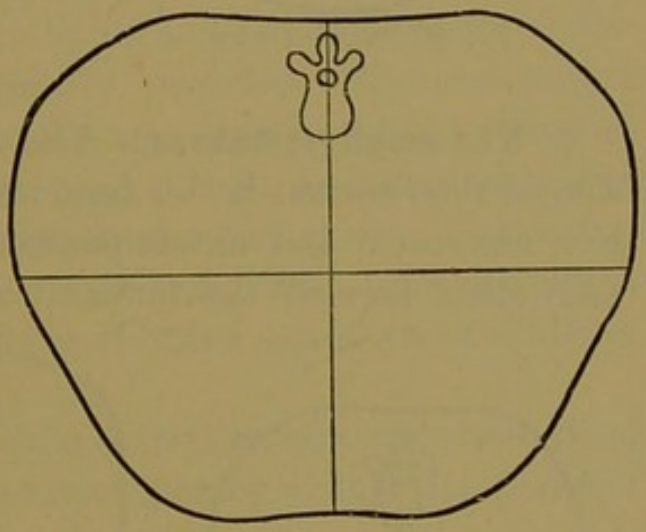


Fig. 59.—Normal chest.—Gee.

There are certain well-marked types of chest in which the departure from the normal (Fig. 59) is so great that they require a special description. They are—(1) The rickety; (2) the pigeon-breast; (3) the emphysematous; (4) the alar.

1. **The rickety.**—In this form, as seen in the diagram, there is a lateral retraction of the thoracic wall, while the anterior surface is squarer than in the next form. It is produced by yielding of the ribs, the result of rickets. It occurs in early life. In addition there is present a row of bead-like projections on each side, at the junction of the cartilages and ribs, which is known as the rachitic rosary.

2. **The pigeon-breast.**—This form is characterised by its

triangular shape, the sides of the chest meeting almost at an angle. The sternum is in this way carried forward, and has been compared to the keel of a boat.

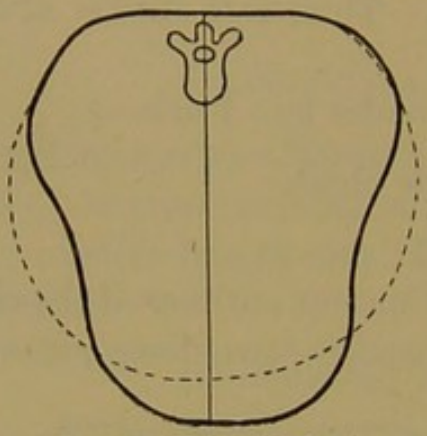


FIG. 60.—Rickety chest.—Gee.

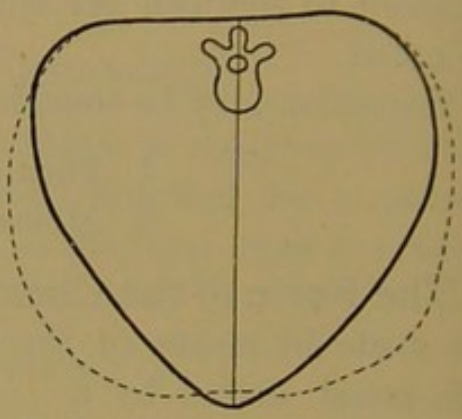


FIG. 61.—Pigeon-breast.—Gee.

3. **The emphysematous.**—The chest becomes more barrel-shaped than normal in this form; that is, it becomes rounder, the transverse and antero-posterior diameters approaching each other in size, the former becoming smaller while the

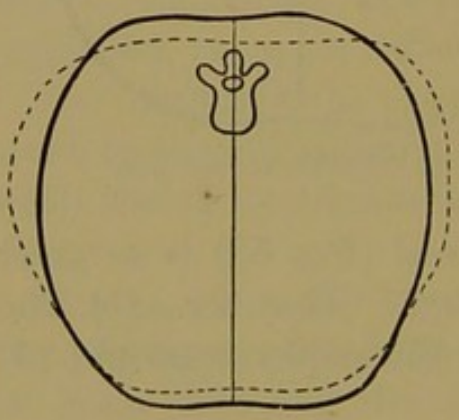


FIG. 62.—Emphysematous chest.—Gee.

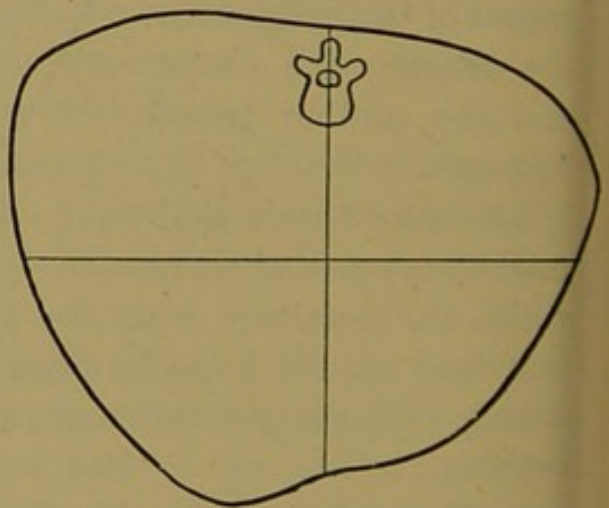


FIG. 63.—Showing retraction of one side of the chest.—Gee.

latter enlarges. The intercostal spaces are, as a rule, sunken. The sternum is arched forwards.

4. **The alar.**—This form is so named from the wing-like projection of the scapulæ. It is associated with a long narrow chest and greatly sloping shoulders. It occurs in persons of a phthisical inheritance.

Local alterations in form.—Contraction of one-half of the thorax may follow on pleurisy or empyema. It is also present in fibroid phthisis and other destructive changes in the lung.

Flattening under the clavicles occurs in cases where the apices of the lungs have been the seat of destructive changes, annulling or modifying their respiratory function. It is therefore associated with diminished respiratory movement. It may be present at both apices or be confined to one.

In addition to the foregoing, which are those most commonly described, it is well to remember that in *angular and lateral curvature of the spine* the shape of the thorax is much altered, and that the limits and relative volume of the two lungs may be materially modified. In pronounced lateral curvature, owing to the altered curve of the ribs, and the altered volume of the lung, it will be found that the percussion sound is different on the two sides. Unsatisfactory physical signs must not in such cases have too much importance accorded to them if there is not other evidence of pulmonary disease.

The size of the chest in its circumference, and its vertical and horizontal diameters, varies greatly within the limits of health.

The following measurements are those usually made :—

The circumference is often the only measurement which is made. It is taken at the level of the nipples. The average is 34 in., but it varies from 28 to 44 in. in the adult male. The chest expands from $1\frac{1}{2}$ to 5 in. during forced inspiration, and it is well to observe this in health, as the diminution in the degree of expansion in various morbid conditions is to be specially noted. It occurs, for instance, to a very marked degree in emphysema and asthma, in both of which the measuring line may not indicate more than from a quarter to half an inch of increased circumference during inspiration.

The semi-circumference of the chest is greater on the

right side than on the left by about half an inch. This measurement is taken from the spines of the vertebræ behind to the middle of the sternum in front; it is well to make a mark at both points before measuring. Distention of one-half occurs in pleuritic effusions when very copious, and also in some cases of pneumo-thorax. Retraction on one side occurs in fibroid induration of one lung, and after some cases of pleurisy or empyema.

In addition to the foregoing the *antero-posterior* and *transverse diameters* are of importance. They are taken by means of an ordinary steel *callipers*. Normally the transverse is greater than the antero-posterior diameter, but in abnormal conditions this may be considerably modified, as has been seen in the preceding pages.

Movement.—While in the normal state a more or less general expansion of the thorax occurs during inspiration, there are conditions in which this is modified or reversed. They may be classed under these heads:—

A. Diminished expansion—general or local.
B. Increased expansion—general or local.
C. Drawing in of the intercostal spaces—retraction.
D. Alterations in rhythm and rate.

Diminished Expansion may be *general* and affect both lungs, as in emphysema, asthma, etc.; it may be *unilateral*, as in pleuritic effusion, pneumonia, etc.; or it may be *local*, as at the apices from phthisis, elsewhere from pleurisy, collapse, etc. It occurs as a result of three main causes,—first, non-entrance or obstruction to the entrance of air into the lungs; second, such a distended state of the pulmonary vesicles as will not admit of much or any more distention; third, paralysis of the muscles which move the ribs.

First. Non-entrance, or obstruction to the entrance of air into the lungs. This condition may be the result of causes —(1) In the upper and larger respiratory passages; (2) in the smaller bronchi; or (3) in the pulmonary vesicles. Under

the first are included membranous laryngitis, paralysis of the vocal cords, laryngeal tumours, the pressure of tumours on the trachea or large bronchi, etc. Under the second, asthma—the obstruction being caused by spasm of the smaller bronchi. Under the third, phthisis, compression of the lung by pleural effusion, congenital or acquired collapse, pneumonic consolidation, and acute pleurisy, pleurodynia, or intercostal neuralgia. In the three last, movement of the painful part is restrained owing to the increase of pain experienced on movement.

Second. An already distended condition of the pulmonary vesicles. This is typically seen in emphysema, the lungs and their containing case being so abnormally distended that even with deep inspiration the thorax may only expand about half an inch.

Third. Paralysis of the muscles of respiration which act on the thorax. This occurs in certain cerebral and spinal diseases or injuries, respiration being maintained by means of the diaphragm.

Increased Expansion.—General increased expansion occurs during violent exercise, and as a result of muscular effort. In the early stages of febrile affections there is, along with acceleration of the breathing, increased general expansile movement.

Local increase may affect one half of the chest, as when the lung on the opposite side is not performing its function. It is present when air or fluid in the opposite pleural cavity compresses the lung, also when there are destructive changes in the one lung, and when the bronchus on one side is compressed or has its lumen obstructed in any way. When the apices of the lungs are the seat of tuberculous infiltration, the respiratory movement of the lower part is increased; and when the lower parts are infiltrated with pneumonic exudation, or compressed by fluid, the upper part shows increased movement.

Drawing in, or Retraction.—(*a*) General depression of

the intercostal spaces, the epigastrium, and the episternal and supraclavicular regions, occurs during inspiration in cases in which there is obstruction to the entrance of air. It occurs from a variety of causes. The obstruction may be in the upper part of the respiratory passage, as in diphtheria; it may be lower down from the pressure of an enlarged thyroid, or of an aneurysmal or other mediastinal tumour on the trachea or bronchi, or it may be due to spasm of the smaller bronchi, as in some cases of asthma or bronchitic asthma.

(*b*) Local depression of the intercostal spaces occurs over areas of congenital or acquired collapse: this is frequently seen in children, and is most marked in the spaces between the asternal ribs anteriorly and laterally.

In all cases the explanation is, that the capacity of the thorax is increased by the movement of its bony and cartilaginous framework, while at the same time the lung tissue does not expand: as a result the intercostal spaces and other yielding portions are driven in, for the atmospheric pressure remains constant while the pressure within the lung is necessarily diminished when the capacity of the thoracic cavity enlarges, and the entrance of air is obstructed.

Alterations in Rhythm and Rate. — Dyspnœa. — This term denotes increased rapidity or increased depth of respiration. It is brought on by physical exertion, but also by various morbid conditions. Elevated temperature is usually accompanied by accelerated respiration, while in many conditions there is not only acceleration, but increase in the depth of the respirations. When dyspnœa is caused by any hindrance to the free ingress or egress of air, it is known respectively as inspiratory or expiratory dyspnœa. *Inspiratory dyspnœa* is present in membranous laryngitis, the paroxysms of whooping-cough, paralysis of the vocal cords, or any condition in which the lumen of the upper part of the respiratory passages is diminished, as by the pressure of

aneurysmal and other tumours. *Expiratory dyspnœa* is present in the more advanced cases of bronchitis and emphysema, in which there are both obstruction in the bronchi and a diminution in the elasticity of the lung. It will be remembered that expiration depends on the elastic recoil of the lungs, and consequently, when this is weakened, expiration must be impaired.

Orthopnœa is a very marked degree of difficulty in breathing, the term being confined to cases where the patient requires to remain in the sitting or standing posture. It is present in the advanced stages of cardiac disease and in asthma, etc. In it the accessory muscles of respiration are called into action. The diseases mentioned above as giving rise to dyspnœa produce orthopnœa in their more advanced phases.

Cheyne-Stokes Respiration. — This is the best-marked type of alteration in the respiratory rhythm. When fully

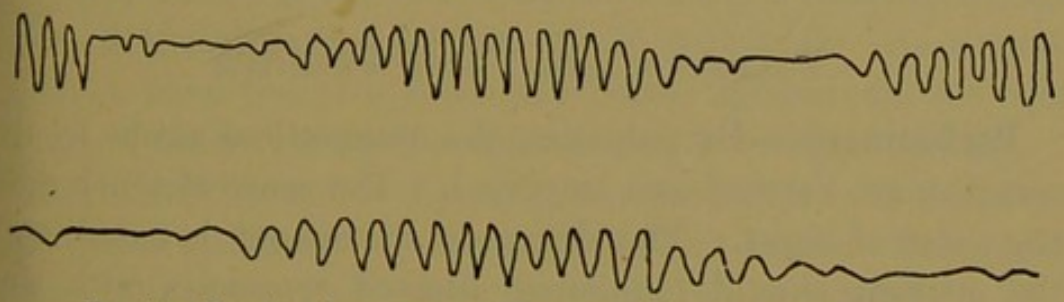


Fig. 64.—Tracings from cases of Cheyne-Stokes breathing.—Gibson.

developed it is characterised by a repeating cycle of phenomena; beginning with a number of superficial respirations it goes on to deep respirations, often amounting to dyspnœa, from which it falls again to superficial respirations resembling those with which it commenced; these are often called the *ascending* and *descending* phases, and following them is a period of complete cessation of the respiratory movements. The whole cycle occupies from a few seconds to two minutes, the pause occupying about one half the whole time. The tracings (Fig. 64) represent graphically the phenomena in this condition. It may occur in all affections involving the

cerebral and medullary centres. It is frequently present in uræmia and many acute diseases.

While the above is the fully and typically developed phenomenon, there are many minor forms of irregularity which are allied to the fully developed form, but require no special description; some of these are not infrequently referred to as *cerebral breathing.*

The Respiratory Action of the Diaphragm.—This has been already referred to at the beginning of the chapter, but it is again necessary to draw attention to the importance of noting the extent to which respiration is performed by the diaphragm in different conditions. In emphysema, for instance, respiration is largely performed by it; while, on the other hand, if it is paralysed, or much impeded in its action by abdominal conditions, as ascites, etc., dyspnœa becomes extreme. No record of the respiratory functions which overlooks this can be regarded as complete.

Palpation and Vocal Fremitus

Preliminary.—By palpation the observations made by inspection are verified and amplified. The sense of touch aids the sense of sight. The degree of movement is noted, and the student should familiarise himself especially with the expansile movement of normal respiration. The hands should be flatly and firmly laid upon the corresponding parts on the two sides. Palpation of the apices anteriorly is more easily performed by standing behind the patient, when that is practicable, and putting the hands across the shoulders; in the same way, for palpation of the apices posteriorly, the hands may be put across the shoulders from the front. The lower part of the thorax, both laterally and posteriorly, should always be palpated as well as the apices, and a note made of the movement in these various situations.

The conditions in which deficient expansion is present

have been already mentioned under inspection, and need not therefore be repeated.

Pain on palpation is sometimes present, and may be even acute. It may be present either in the intercostal muscles, when it may be rheumatic, or it may be confined to the nerves. It is also present before the appearance of the eruption in herpes zoster (shingles). In some cases of pleurisy pain is complained of on palpation, especially if considerable pressure be made on the part.

Vocal Fremitus. — The physical explanation of vocal fremitus has been already considered. The factors which determine its intensity may be classed as follows :—

(*a*) The pitch of the voice : the lower the pitch the more marked the fremitus, and *vice versâ*. This explains the fact that in women it is, as a rule, less marked than in men, the former having the high-pitched voices. Feeble voices are also badly conducted.

(*b*) The size of the tube (the bronchus), and therefore the size of the vibrating column of air within it. As a consequence vocal fremitus is usually better marked on the right than on the left side, the right bronchus being the larger.

(*c*) The distance of the part from the larynx—that is, from the point where the vibrations have their origin : the greater the distance the feebler the fremitus, and *vice versâ*, so it is more marked over the upper part of the anterior aspect of the chest than the lower.

(*d*) The thickness of the chest wall : the thicker the chest the feebler the fremitus. A thick wall, whether due to fat, muscle, or œdema, obstructs the passage of the vibrations to the surface.

From the foregoing it is apparent that too much importance must not be attached to want of success in obtaining fremitus, unless there is corroborative evidence from the other methods of investigation that it is the result of a morbid condition.

The conditions in which vocal fremitus departs from the normal may be classed as follows :—

Diminished in—

Persons with feeble or high-pitched voices (may be quite imperceptible).
Persons with thick chest walls.
Adherent and thickened pleura.
Bronchitis, Asthma, } from obstruction in the bronchial tubes to the passage of the vibrations.

Absent in—

Hydrothorax.
Pleurisy with effusion.
Empyema.
Pneumothorax.
Pressure of a tumour on a primary bronchus.
Anything within the bronchi blocking them, and thus preventing the passage of vibrations to the part supplied by the blocked tubes.
Phthisis, when the bronchi are plugged as in the preceding.
Adhesion with great thickening of the pleura.

Increased in—

Consolidation of the pulmonary parenchyma, as in pneumonia. It is, however, necessary that the principal bronchus going to the consolidated area be not plugged ; if it is, the vibrations are arrested.
Pulmonary cavities, if they have dense walls, are large and near the surface.
Solid tumours in contact with large bronchi, and also with the parietes.

Friction or pleural fremitus is the sensation communicated to the hand in cases of pleurisy where the friction is

unusually coarse. It is a creaking, rubbing, or grating sensation, and is only occasionally present. It is a phenomenon of no special clinical significance.

Bronchial Fremitus.—In bronchitis and asthma, owing to local interferences with the lumen of the bronchi, the passage of air is interrupted, and sounds are produced which are known as rhonchi: they are described later. When, in these conditions, the hand is placed over the larger bronchi in front or behind, the vibrations producing the sounds may be distinctly felt, and bear the name of bronchial fremitus.

Percussion and Sense of Resistance

Preliminary.—The percussion sound of the lungs is usually defined as clear or resonant. Its precise character cannot be described; it can only be appreciated by carefully training the ear to recognise it and its various modifications in different individuals, and at different parts of the thorax. The terms, resonant and clear, are as a rule used as synonymous. It is, indeed, usual to distinguish between greater and less degrees of resonance, and to refer to the percussion sound of the thorax as increased, diminished, or impaired in resonance, or as non-resonant.

The term resonant, as applied to the percussion sound of the normal lung, indicates a somewhat full sound, and conveys the impression of being produced by the vibrations of a structure of considerable size and containing air. The term hyper-resonant is often applied to the lung sound when it is clearer than normal, or when it approaches the tympanitic character; on the other hand, the term "comparative dulness" is used when the resonance is slightly diminished, a diminution which can most easily be detected by a comparison with the sound given by a corresponding portion of the healthy lung on the opposite side; the term dulness is used when there is a marked absence of resonance.

The pulmonary percussion sound depends on the four following factors :—

1. **The thickness of the chest wall.**—The greater the thickness the duller is the percussion sound, and *vice versâ*. The thickness may be due to great muscularity, to the deposition of fat, to œdema, or, as in women, to the mammæ. On the other hand, the chest wall is thin in spare persons, and in exhausting diseases.

2. **The resilience of its bony and cartilaginous framework.**—This is greater in children than in adults, and the percussion sound is clearer in the former than in the latter.

3. **The amount of air within the chest, that is, in the bronchi and pulmonary alveoli.**—When the air cells are filled with solid exudation, as in pneumonia, or when the lung is compressed and the air driven out of it by the pouring out of fluid into the pleural cavity, as in pleurisy and hydrothorax, the percussion sound is dull. The percussion sound is also duller over the heart than where there is only lung underneath the part percussed, for the heart acts as a solid body.

4. **The state of tension of the lung tissue.**—If from any cause the lung tissue—that is, the walls of the alveoli—has its tension diminished, the percussion sound is modified; it is lowered in pitch and may be tympanitic. This modification frequently occurs above the limit of a pleural effusion; in pneumonia, before the lung has become solid; in œdema of the lungs; and in caseous infiltration of the lung tissue, the air not being entirely excluded.

To understand the cause of this tympanitic sound, it is necessary to know that if a lung be percussed after its removal from the body, when it has of course partially collapsed, the sound emitted is tympanitic. It is from this, and from a consideration of the changes present in those morbid conditions in which it may be elicited, that the tympanitic sound is regarded as due to diminution in the tension of the lung tissue, and a diminution in the amount

of air in the alveoli. These conditions are present in the morbid states enumerated above. The tension or tonicity is of course lessened by the inflammatory action in pneumonia and by the pressure of the fluid in pleuritic effusion, while the amount of air in the alveoli is necessarily reduced in all cases where an exudation takes place into them. It must, however, be acknowledged that this explanation is not altogether satisfactory.

Method.—When the anterior aspect of the thorax is to be percussed the patient should be recumbent and unconstrained, or in the sitting posture.

Care must be taken that the finger is laid quite flat on the surface, and the percussion sound on one side is to be compared with that on the other, in corresponding spaces and over corresponding ribs.

The apex is percussed above the clavicle by the mediate method, then the clavicles are percussed immediately, after which the rest of the lung is percussed downwards. In the first instance it is only necessary to percuss the interspaces. If an area be found where the percussion sound is altered, it can be investigated rib by rib and space by space. On no account ought the student to compare an interspace on one side with a rib on the other; and the finger ought never to rest partly on a space and partly on the adjoining ribs. The percussion sound over the ribs differs considerably from that in the interspaces; and further, if the finger is laid vertically over both, there is apt to be a space between the finger and the interspace, which materially modifies the sound, and is very likely to mislead.

It is desirable to lay emphasis on one other point, and that is, the necessity of keeping well out from the edge of the sternum: the mammary line is a good one as far down as the third rib, but from there it is necessary to diverge quite outside the line. This is necessary, owing to the position of the heart on the left side, which of course affects the percussion sound, and when studying the percussion of

the lungs this source of confusion is to be avoided. On the right side it is necessary to keep well out from the sternum, as the presence of the arch of the aorta, the descending vena cava, and, lower down, a distended right auricle may modify the sound. We would lay stress on this, as we have found inattention to these simple matters a very common hindrance to the student. Under percussion of the heart and the vessels at its base, it was pointed out that the outer limit of these structures was indicated by a moderate although definite alteration in the percussion sound as followed from without inwards. The alteration in the sound consists in a heightening of its pitch, caused by the thinness of the anterior edge of both lungs where they overlap the heart and large vessels. This thin edge of lung presents a smaller or shallower area in which vibrations are set up, the result being that the duration of the sound is diminished and the pitch is raised. If the sound is further analysed, it is observed that the sound conveys the idea of being produced by the vibrations of a structure of less volume than that of the thick part of the lung, and from this the term *empty* has been applied to it by Skoda to distinguish it from that heard over the substance of the lung, and termed by the same authority *full*. The term *shallow* would be preferable to empty. In fact, the more general use of the terms shallow and full instead of reference to pitch might be desirable. This diminished duration, or more shallow character of the percussion sound, should also be studied by percussing the right lung from above downwards. At the lower part, from about the level of the fourth rib, the percussion sound is higher in pitch, and shallower than above that limit: this is due to the diminished depth (applying depth in percussion of the thorax to the antero-posterior volume of the lung) of the lung here, and to the presence of the solid liver behind it. The same limit is known as the line of *comparative dulness* of the liver. The accompanying diagram(Fig. 65), representing a vertical transverse section, shows the relations of parts which

lead to the rise in pitch, or shallowing of the percussion sound, before the absolutely dull sound of the liver is obtained when we pass entirely from lung.

At the upper part anteriorly it is often necessary to percuss the whole length of the interspaces, as dulness in this position may be confined either to the inner or the outer part of them, according to the part of the apex affected. In other cases small areas of dulness may be made out scattered over

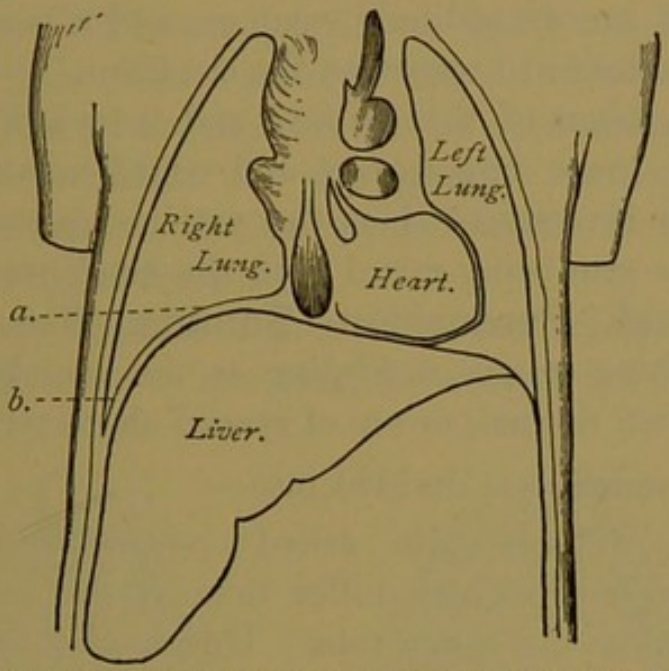


FIG. 65.—Vertical transverse section in mammary line, showing relations of lung and liver (after Symington). Above *a* full lung sound, below *a* shallow and high-pitched lung sound. *b*. Inferior margin of lung, point at which absolute liver dulness commences.

the chest, but this is as a rule difficult of detection, even to a very expert percusser.

When the posterior aspect of the thorax is to be percussed, it is desirable to have the patient in the sitting posture when practicable, and when it does not subject him to too much fatigue. The patient's arms ought to be folded in front. The method to be pursued is the same as that already described for the anterior aspect. We begin above the scapulæ and proceed downwards, comparing the corresponding areas on the two sides. The sound differs much from the sound in front, owing to the difference in the structures over-

lying the lung: from the angle of the scapula downwards, the sound, however, more nearly resembles that produced in front. To make out the extreme inferior limit of the lung, it is necessary to percuss very lightly, and there are few points of greater practical importance than the acquirement of facility in doing this, and of fully appreciating the evidence as to the exact position of the lower margin of the lung here. The reason of this is, that moderate degrees of hydrothorax are exceedingly common, and it is often of the utmost importance to recognise the condition.

The percussion of the axillary and infra-axillary regions is often of great importance, and ought not to be overlooked. To investigate them the patient may sit, or lie on the side, the arm being raised with the hand over the head.

Abnormal Percussion Sounds.—These may be classed in three groups, according as the sounds are duller or clearer than normal, or are of special character.

A. **Dull sounds** are divided into—

1. Slight dulness—also called *comparative dulness*—when it is slightly duller than at the corresponding spot on the other side. This is also spoken of as *impaired or deficient resonance.*
2. Moderate dulness—when the sound is decidedly dull, but not so marked as in the following case.
3. Absolute dulness—when the part gives a sound resembling that of a solid organ.

B. **Abnormally clear sounds** (called also *Hyper-resonant*). These may be divided according to their degree into—

1. Slight or comparative increase of clearness.
2. Moderate increase of clearness.
3. Marked increase. The tympanitic sound.

C. **Percussion sounds of special quality or character** :—

1. Amphoric or metallic.
2. Cracked-pot.

We may now consider these in order.

A. **Dull Sounds.—1. Slight or comparative dulness.**—This is frequently present at one or other apex, more commonly at the right. It is sometimes regarded as physiological, but this is doubtful, for it will be found that, in at least a large proportion of cases, it is associated with and due to adhesion and thickening of the pleura. A corresponding degree of dulness can be made out over other parts of the lung where the same conditions are present. To appreciate this difference percussion must not be strong; indeed, for all the finer degrees of sound it is necessary that the student should acquire the art of percussing with one finger. Slight dulness is also present in commencing tuberculous disease at the apices, and where there is some slight thickening of the pleura.

2. **Moderate dulness** is present in tuberculous disease of the apices when the whole lung tissue has not become in-

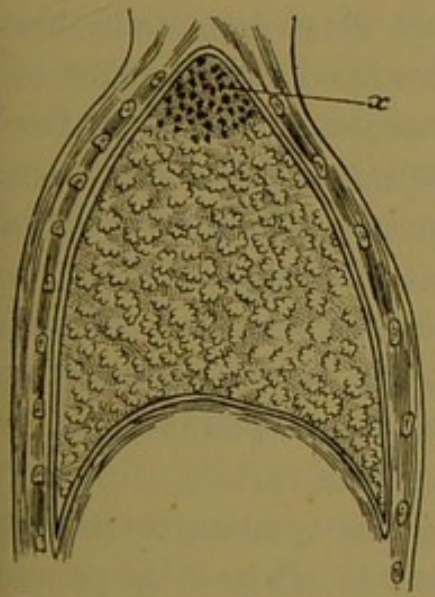


FIG. 66.—Diagram showing at *x* moderate dulness over tuberculous infiltration.

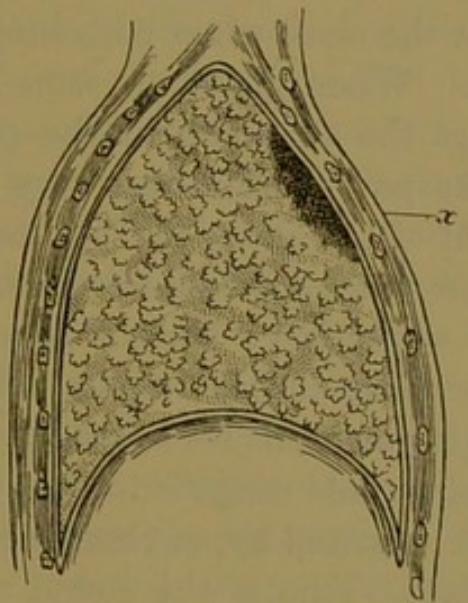


FIG. 67.—Diagram showing at *x* moderate dulness over superficial pneumonic patch.

filtrated. It is also present over patches of catarrhal pneumonia which reach the surface of the lung; and in the patchy acute pneumonia of old people; also in pulmonary congestion in which exudation takes place into the alveoli.

3. **Absolute dulness** is present when air is completely absent from the part percussed: it is therefore present in acute croupous pneumonia in the stage of hepatisation, when the alveoli are filled with coagulated exudation. It may also be present in phthisis from an analogous condition coupled with thickening of the pleura. It is also present over effusion into the pleura, whether the result of pleurisy or a simple hydrothorax; also over tumours, the more common ones being mediastinal tumours extending forwards and laterally, and coming into contact with the thoracic wall. Tumours of the lung itself, when of considerable size and situated at the surface, also give a dull sound.

B. **Abnormally Clear Sounds.**—1. **Slight or comparative increase in clearness.**—Hyper-resonance occurs from the slighter degrees of emphysema, and is not of much practical importance.

2. **Moderate increase or hyper-resonance** is present all over the chest when both lungs are emphysematous throughout. When the emphysema is confined to the edges of the lungs the alteration in the percussion sound is confined to those parts. This can very frequently be noted over the front of the chest, the percussion sound becoming distinctly clearer on percussion from without inwards,—that is, towards the edge of the lungs. It is also present over the area of superficial cardiac dulness, when the heart is covered by the emphysematous border of the left lung. The more markedly clear sounds merge into the tympanitic variety.

3. **Marked hyper-resonance or the tympanitic percussion sound.**—This is the sound which is elicited by percussing the stomach or intestines when they contain air. In the normal condition it is not produced by percussion of the thorax, save inferiorly on the left side, when the stomach is distended, as is specially referred to under the examination of that organ. In several morbid conditions, however, the percussion sound presents this character. They are as follows:—

(*a*) Pneumothorax, *i.e.*, the presence of air in the pleural cavity, leading to retraction of the lung. If the tension of the air within the pleura be great, the percussion sound retains its tympanitic character but rises in pitch, and is commonly referred to as duller. This is also true of the percussion of the stomach and intestines: if their walls be tightly stretched by the amount of contained gas, the tympanitic sound they give rises in pitch.

(*b*) Large and superficial cavities in the lung substance, the result of destructive changes.

(*c*) The early stage of pneumonia before consolidation takes place. This is generally regarded as due to diminished tension in the affected portion of lung.

(*d*) Œdema of the lungs, the physical conditions being much the same as in (*c*).

(*e*) Over the lung, immediately above pleuritic effusion. The explanation of this is similar to that given under (*c*), namely, that the tension of the lung is diminished above the limit of the effusion. The fact of a tympanitic sound being present in this condition is to be remembered, for it is sometimes regarded as proof of the presence of pneumothorax.

(*f*) "The tracheal resonance of Williams" is the term which has been applied to a tympanitic sound present at the apex of the left lung, close to the sternum, when the lung there is consolidated. To understand this it is necessary to note that the percussion sound over the larynx and trachea is tympanitic, the pitch depending on whether the mouth is open or shut. This sound is lost over the bifurcation of the trachea and over the primary bronchi, owing to the interposition of pulmonary tissue; when, however, the intervening lung is consolidated, either from exudation into the vesicles, or from compression, the sound may be elicited owing to the better conduction of the vibrations by the solid tissue.

C. **Percussion Sounds of Special Quality or Character.**—1. **Amphoric or metallic percussion sound.**—This sound is tympanitic in character, but has in addition a metallic

clang or echo. In other words, the sound is prolonged, the prolongation being compared to an echo. It is comparable to the sonorous prolongation of the voice which is observable when we speak in an empty hall; it is also to be noted when a large, empty, or partially filled and covered vessel is percussed. The tympanitic sound, on the other hand, is not prolonged, it ceases immediately after the percussion blow producing it. The amphoric sound is heard best in cases of pneumothorax and in large superficial cavities in the lung; in both it is necessary that the walls of the cavity be smooth, so that the sound waves may be reflected equally from all parts of the wall. In pulmonary cavities the sound comes out best when the mouth is open.

2. **Cracked-pot Percussion Sound** (*Bruit de pot fêlé*).—This sound, as its name implies, resembles that produced by percussing a cracked vessel. It is a peculiar clinking sound which can be simulated by clasping the hands at right angles to each other, and then striking them smartly against the knee. It is produced—

(*a*) Over pulmonary cavities when they communicate directly with a bronchus, and when the chest wall overlying them is emaciated and yields to the percussion stroke. It is almost confined to cavities at the apex. To elicit the sound percussion should be performed during expiration, and the patient's mouth ought to be open. It is caused by the sudden expulsion of a portion of air from the cavity into the bronchus. While this is the most common condition in which the sound is obtained, there are several others in which it is occasionally present, namely—

(*b*) In some cases of pleurisy above the limit of the effusion, due probably to the yielding of the chest wall, and the partially compressed state of the lung favouring the sudden expulsion of air by the percussion stroke.

(*c*) In pneumonia before consolidation has taken place. In both this and the preceding condition it will be remembered that a tympanitic percussion sound is sometimes present.

(*d*) When an opening exists through the parietes into the pleural cavity. It can therefore be readily demonstrated when the pleura has been incised for empyema, and its non-production when the opening is artificially closed can also be demonstrated.

(*e*) In pneumothorax, where there is a free communication between the cavity and a bronchus.

(*f*) It is readily produced in healthy children over the upper part of the lung, especially when crying, owing to the very yielding character of the thoracic wall in infancy.

In all these the physical explanation is the same, and it is only necessary to specially warn students against assuming that a cracked-pot sound necessarily implies the presence of a cavity; and this warning is all the more necessary as there is probably no evidence derived from percussion, unless it be the tympanitic sound, which is more frequently misconstrued.

3. **The Bell or Anvil Sound** is a peculiar ringing bell-like sound, which can be obtained in pneumothorax. To elicit the sound the observer auscultates the chest, while an assistant places one coin upon the chest wall to act as a plessor, using a second coin as pleximeter. The observer auscultating will hear a ringing, musical tinkling sound, which may be compared to the sound of a miniature hammer striking on an anvil. The sound is found in pneumothorax, over the air-containing cavity. The extent of the pneumothorax can be defined by means of the sound, for it is lost when the stethoscope passes the limit of the air-containing space.

The Sense of Resistance.—Before leaving the consideration of percussion it is necessary again to point out the importance of the *sense* of resistance as an aid in percussion of the chest. In the percussion of the chest the power of recognising variations, in the sense of resistance, becomes almost unconsciously very highly developed, and enters to a greater extent than is generally recognised into the deter-

mination of whether a part is dull, and which of two parts is the duller. From what has been said, under the preliminary consideration of percussion, it will be understood that the more marked the dulness, the greater will the sense of resistance be. The physical conditions which produce the dulness, that is, the increased consistency or solidity of the part percussed, lead also to an increased sense of resistance.

Auscultation

One of the greatest difficulties in the auscultation of the lungs is the differences in the classification of the sounds, and the want of agreement amongst teaching physicians as to the characters which distinguish the different groups. This necessarily leads to great confusion in the mind of the student, and surrounds the exposition of the subject with difficulties, while it is, we fear, somewhat hopeless to attempt to meet the views of all teachers of practical medicine. There is not by any means the same degree of confusion in the interpretation of the signs, but even here there is room for greater accuracy, and the attainment of this can only be realised by the more rigid definition of the terms at present in use. In teaching and in learning, the tendency, we think, is too much towards classification without a descriptive and critical examination of the sounds, which would constitute a training in itself. The practice is too much to draw attention to the breathing at a specified spot in an individual case, as bronchial, tubular, broncho-vesicular, or whatever else the individual teacher may choose to call it, without that critical analysis of the sound which would resolve it into the component elements which give it its special features, and on some of which at least its classification must be consciously or unconsciously based. That this must necessarily be the case is easily rendered apparent. Respiration consists of two parts, inspiration and expiration. Over different parts of the respiratory tract the audibility of inspiration and expira-

tion varies in length, loudness, and tone, and the relative length of the one to the other. A knowledge of these variations, as they are heard over different parts in the normal condition, is necessary to the recognition of the alterations which occur in disease, but even in disease all alterations must be confined to inspiration and expiration, and the only modifications they can manifest must fall under loudness, character, pitch, and the relative length of inspiration and expiration to each other. In disease there are, in addition, accessory sounds which have to be considered.

Auscultation in the Normal State.—Normal respiration consists, as has been already said, of inspiration and expiration. During each a sound is produced, and it is on this fact that auscultation of the respiratory organs is practicable.

If auscultation be practised *over the trachea*, or, what is still better, *over the cervical vertebræ*, where the pressure of the stethoscope is less disagreeable to the person auscultated, it will be found that a sound is heard during inspiration and another during expiration. The loudness and tone or character of these sounds depend on whether the individual auscultated is breathing through the nose only, or has the mouth open and is breathing mainly through it, and on whether the breathing is slow or rapid.

If the breathing be performed with the mouth open and is not accelerated, it will be observed—(*a*) That the inspiratory and expiratory sounds are distinct from each other, and separated by a short but distinct break; (*b*) that they are about equal in duration; (*c*) that they have a soft blowing character, which is more pronounced and louder during the expiratory than the inspiratory part.

If now the mouth be closed and respiration carried on through its proper channel, the nose, it will be observed that as regards (*a*) and (*b*) the sounds are the same as when the mouth was open, but that an important change has occurred as regards (*c*). It consists in an alteration in the loudness

and in the character of the sounds—the loudness has become intensified and the sound has become harsher.

In the first case the sound heard was produced by the air passing into and out of the mouth, and by its passing in and out past the vocal cords, where the lumen of the air passage is smaller than above and below them. In the second case the greater loudness and harshness were due to the air passing in and out by the nose. Owing to its construction, a narrow passage is formed opening into a wider one, the pharynx, and this gives the conditions which intensify sound; there are other elements in this, which need not, however, be dwelt upon.

We have also stated that the character of the sounds depends on the rapidity of the breathing. If the breathing be rapid and conducted by the mouth, the sounds are louder and harsher than when breathing is slow. This is due to the greater friction resulting from increased speed, the friction occurring at the vocal cords mainly. In this condition the sounds approximate to those heard when the breathing was nasal.

In these three cases the sounds are heard over the regions specified as they are conducted there by the air in the trachea, or transmitted to the vertebræ from the trachea. They are audible anteriorly until the sternum is reached, and posteriorly as far as the upper dorsal vertebræ. The sound would, of course, be heard over the large bronchi were they within reach of the stethoscope, but they are so overlaid by the parenchyma of the lungs that sounds conveyed into them from the larynx and trachea have to pass through it before reaching the surface, and in doing so they are considerably modified. As will be seen presently, there is a type of breathing which is known as "bronchial," as it resembles what was supposed to be heard over the bronchi. We have, however, seen that the bronchi, even in their primary divisions, are so covered by lung that the sound in them can only be heard after it has been modified by its

passage through lung parenchyma. We take therefore *the sound heard over the trachea and spine* as the type of what is clinically known as *bronchial breathing.*

If now auscultation be practised *over the thorax at any part where the lung comes into contact with the parietes,* it will be observed that the sounds have greatly altered. The first thing that as a rule impresses the student is the difficulty of hearing anything, the sounds being so much feebler than over the trachea. Instead of the more or less harsh or blowing sound during inspiration and expiration, there is now an extremely soft and often a very feeble sound heard during inspiration, which runs into a still feebler sound synchronous with the first third or so of expiration. The relative lengths of the movements of inspiration and expiration may be expressed thus—

Inspiration : Expiration : : 5 : 6,

whereas the relative duration of the sounds is—

Inspiration : Expiration : : 3 : 1.

But while this is the typical relation, the expiratory part is in many individuals quite inaudible. *Vesicular breathing* is the term applied to breath sounds of this type; it has been compared to the rustling of leaves, and is often referred to as breezy in character. There is still a difference of opinion as to whether this sound is produced by the air passing into and out of the air vesicles, or is due simply to the conduction downwards of the tracheal sound, the tissues through which it has to pass modifying its character. We believe the latter to be, in the main, the correct view, and abnormal auscultatory signs are more easily explained by it.

While these are the sounds heard at the upper and lower parts of the respiratory system, there is present in some individuals at certain points what must be regarded as a transition sound. When present it is best heard in the interscapular region, about the level of the spines of the

scapulæ. Its intermediate character is shown—(*a*) By the soft blowing character of the inspiration, or by an inspiration rather harsher than that heard over the body of the lungs; (*b*) by an expiration which is neither as long nor as harsh as that heard higher up; while (*c*) the relative lengths of inspiration and expiration approximate more nearly to those present over the parenchyma than over the trachea. From its intermediate character it has been called *broncho-vesicular or indeterminate* breathing, the former being the better term. Its explanation is that the large bronchi are nearer the chest wall here than at any other part, but that even here the sounds conducted into them from above have to pass through a considerable thickness of lung tissue to reach the chest wall, and therefore the ear; in their passage they are *damped down.*

The term broncho-vesicular would be equally applicable did we hold the opinion that the air vesicles themselves produced sound during their inflation and retraction, for the sound would then be a combination of the bronchial sound conducted from a distance, and the vesicular sound produced at the spot.

The foregoing is simply a statement of fact, the phenomena having the names in general use applied to them. The cause of the unsatisfactory state, not of the nomenclature so much as of the individual application of it, necessitates some explanation, and thereby what appears almost hopeless confusion to the student may be simplified and put somewhat in order.

In the first place, it is found that the term *bronchial* is applied by some to all the varieties of sound described in the preceding pages as audible over the trachea, cervical vertebræ, and interscapular regions. This includes breathing characterised at one end by harsh inspiration and expiration of equal length, to, at the other end, breathing with a soft blowing inspiration, followed by a short expiration which varies in tone. Not only this, but breathing the inspiratory

part of which shows any of these characters, and is followed by an inaudible expiration, is also classed by some as bronchial. To meet the difficulty raised by including such a variety of sound under one term, some use the term *tubular* to denote the type of harsh sound heard over the trachea; but this only meets a small part of the difficulty.

On the other hand, some exclude from the term *bronchial* all sounds which have not both the harsh inspiration and the long harsh expiration heard over the trachea with the mouth shut; or extend it also to a breathing in which the expiration is as long as the inspiration, but is blowing in character instead of harsh.

The confusion which must necessarily result is apparent, and there is little probability of advance in accuracy in the auscultation of the lungs until something is done to clear it up. As a matter of fact we have hardly made any advance on Laennec's work, and this is scarcely to be wondered at, when it is realised that the auscultatory phenomena recorded by one observer convey no sufficiently definite and distinctive impression to the mind of another, to enable him to reproduce in imagination the sounds which were heard.

It is then of the first importance that the student should analyse the sounds to which he is listening. He should, *first*, separate inspiration from expiration; *second*, note their relative lengths, and the presence or absence of a pause between the two phases; *third*, note the character of inspiration and of expiration, whether soft, or harsh, or blowing, or a combination of these; and, *lastly*, the pitch of the sound. By so doing he will be able readily enough to adopt any classification necessary for the present, and it will tend to make him an independent observer in the future.

Having thus considered the phenomena to be noted in auscultation of the respiratory organs in a state of health we have next to turn our attention to the phenomena in diseased conditions.

Auscultation in Abnormal States.—Auscultation

in abnormal states is practically confined to those parts of the chest which cover the lungs, where in fact vesicular breathing, or, as in the interscapular regions, broncho-vesicular breathing is normally present.

A. **Varieties of Vesicular Breathing.**—Vesicular breathing is liable to various modifications, which however are not sufficient to remove them from the rank of vesicular: they are classed as follows:—

Weak vesicular or senile.
Harsh do. or puerile.
Do. do. with prolonged expiration.
Wavy, jerking, or interrupted vesicular.
Absence of breathing.

(*a*) **Weak vesicular or senile.**—In this variety the relation of expiration to inspiration is maintained, but the sounds are faint. It is present in persons whose lungs are voluminous, and who, from the extent of breathing surface they possess, breathe correspondingly slowly and quietly. It is also, however, present over any part of the lung where there are pleural adhesions, pleural thickening, or a thin layer of fluid between the lung and the chest wall.

(*b*) **Harsh vesicular or puerile.**—The normal relation of expiration to inspiration is maintained here also, but each is louder and harsher than over the normal adult lung. It is called puerile, as it is the character which the vesicular breathing normally presents in children. It is present in the adult when the function of a considerable area of lung is in abeyance. It is therefore present over the unaffected side when there is large pleural effusion on the other side. If there be moderate effusion into both pleuræ, as is often the case in cardiac disease with failure of compensation, it is present over the upper part of both lungs. It is also present over the unaffected lung in pneumonia, over the upper parts of both when there is congestion at the bases, and in fact over the unaffected part in all conditions where, as has been

said, the respiratory function of part is in abeyance, or has been permanently destroyed or seriously impaired. It is therefore also present over unaffected parts in phthisis.

(*c*) **Harsh vesicular, with prolonged expiration.**—The expiration in this variety is, as its name implies, longer than in the preceding or puerile variety. The character of inspiration and expiration is the same as in puerile. It is present in many individuals at one or other apex, commonly the right, and is regarded by some as a normal condition. This, however, is exceedingly doubtful, and so far we incline to

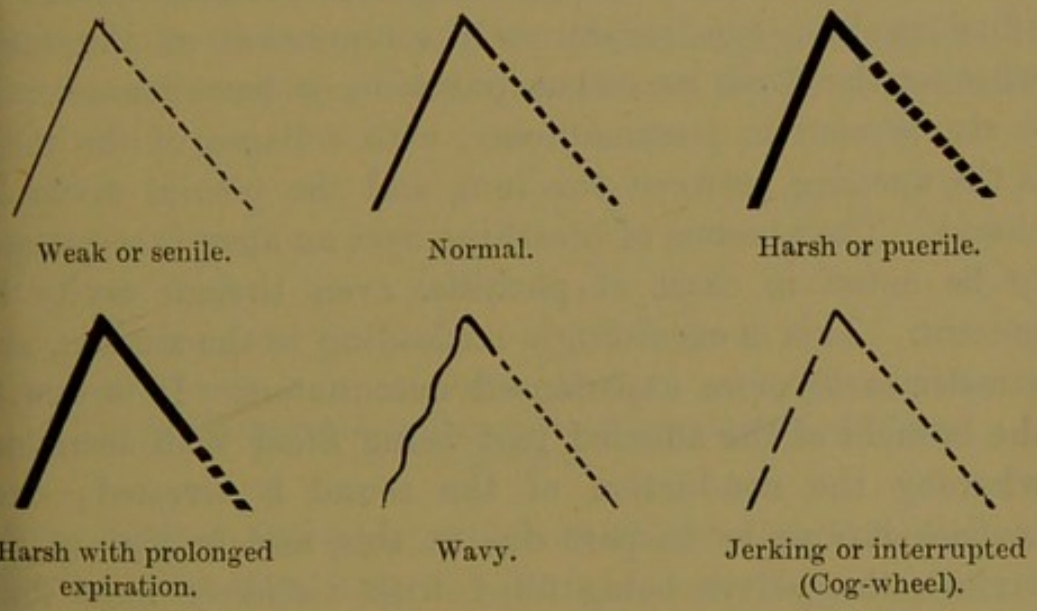


FIG. 68.—Diagrammatic representation of varieties of vesicular breathing. The dotted part of down line represents the inaudible pant of expiration. —After Wyllie.

hold that in many of these cases—we cannot say in all—there is some morbid alteration, not however necessarily indicating active change, but a change the result of an old inflammatory process, and usually fibroid in its nature. The great frequency with which such changes are seen in the post-mortem room tends to support this view.

(*d*) **Wavy, jerking, or interrupted vesicular, or cog-wheel respiration.**—Here the inspiration, instead of being continuous and sustained, is, as its name implies, wavy, jerking, or interrupted. Its character is best understood by

reference to the subjoined diagrams. It is present in nervous patients, especially when they make efforts to breathe deeply at the request of the auscultator. It is, however, also present in early tuberculous infiltration at the apex. In the first it is due to the irregular and undecided manner in which the respiration is performed, while in the second it is probably due to breaks or delays in the passage of the air, and therefore of the sound, passing affected parts.

(*e*) **Absence of vesicular breathing.**—This is noted in conditions in which conduction of the respiratory sounds is no longer possible. It is most marked and typical in effusions into the pleura with compression of the lung, whether the fluid be serous, purulent, or hæmorrhagic. It is also present in pneumothorax, with collapse of the lung, if the opening between the lung and the pleural cavity be closed. The absence of breathing over an apex is sometimes to be noted in cases of phthisis, even though cavity be present. This is exceedingly misleading to the student, and sometimes to more experienced auscultators. It is due to the bronchi of the affected part being filled with secretion, whereby the conduction of the sound is arrested; over cavities it may be in part due to this, and in part to the cavities themselves being filled with viscid or more fluid contents. Coughing, however, usually elicits sounds which indicate the condition of the part.

B. **Bronchial Breathing.**—In morbid conditions, which will be referred to presently, bronchial breathing is present over those parts of the chest where normally the character of the breathing is vesicular.

The characters which separate it from vesicular have been already considered, and the indefiniteness of the term as in general use has been referred to; it is, however, desirable to describe more fully the various varieties of breathing sounds heard over the pulmonary parenchyma which have been included under this head.

The term is applied to breath sounds characterised by an

inspiration and expiration equal in length, separated, however, by a break, and having the bronchial or blowing character heard over the trachea during nasal breathing. This sound is imitated by putting the tongue in a position for pronouncing the "guttural" *ch* and then breathing deeply.

Three types of bronchial breathing are distinguished, differing from each other in the pitch of the sound—

(*a*) **High-pitched Bronchial Breathing,** frequently called "tubular breathing," is the form of breath sound common over the consolidated lung in croupous pneumonia. Inspiration and expiration are about equal in length, or expiration may be rather longer than inspiration; they are separated

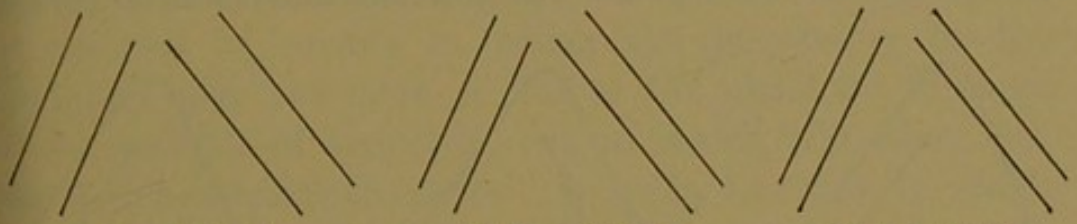


FIG. 69.—Bronchial Breathing—various degrees of pitch.—Modified from Wyllie.

by a distinct interval, and both have the bronchial or blowing character, but the sounds are higher pitched, harsher, and more intense than the sounds heard over the seventh cervical vertebra. The sound produced in the upper air passages passes down the column of air in the trachea and bronchi, and, the lung being consolidated, the sound is conducted through it unaltered, and is heard over the chest, but little altered from its original character. At times the sound is noted to be even more intense than the sound over the trachea. The explanation of this is that the consolidated lung acts as a resonator, it intensifies the sound. High-pitched bronchial breathing is found in conditions such as croupous pneumonia when the consolidation of the lung is intense.

(*b*) **Medium-pitched Bronchial Breathing** is the sound

heard over the seventh cervical vertebra. Inspiration and expiration are about equal in length; they are separated by an interval and have the bronchial blowing character, are medium in pitch, and have a softer character than the high-pitched variety. Such a sound is heard over consolidated lung where the consolidation is not sufficiently intense to produce the high-pitched sound. It is common in tuberculous and other conditions where consolidation exists.

(*c*) **Low-pitched Bronchial Breathing,** commonly spoken of as "cavernous breathing," differs from the other forms by the sound being lower in pitch and having a hollow character. Inspiration and expiration are again about equal in length, separated by a pause, and have the blowing bronchial character, but the sounds are low-pitched and have a hollow

FIG. 70.—Broncho-vesicular breathing.—Modified from Wyllie.

quality, from which the sound has derived the name "cavernous." It is met with where there is cavity formation in the lung, phthisical or bronchiectatic.

The different varieties of bronchial breathing depend primarily upon one factor—the consolidated lung conducting the sound so that it is heard much as at its point of production. The consolidation may be intense, the sound is then high-pitched and may be even intensified. With less complete consolidation the sound is lower in pitch and less intense. If cavity formation be present there is always consolidation around the cavity, the sound then acquires the hollow cavernous character. It must never be forgotten that for the production of bronchial breathing it is necessary that the bronchus communicating with the consolidated portion of lung be unobstructed. If the lumen of the

bronchus be obstructed, as it may by secretion, the sound waves will be cut off, and there will be an absence of breath sounds rather than bronchial breathing. For bronchial breathing it is necessary to have consolidated lung conducting the sound waves from an unobstructed bronchus.

Clinically, bronchial breathing is found in pneumonia, tuberculous infiltration of the lung, fibroid induration, in pleuritic effusion over the compressed part of the lung above the limit of the effusion, collapse and neoplasm of the lung; in all conditions, in fact, where consolidation occurs.

Bronchial breathing, or its cavernous variety, is also present over phthisical cavities or bronchiectatic dilatations.

C. **Broncho-Vesicular, Vesiculo-Bronchial, or Indeterminate Breathing** is a designation which should be confined to sounds in which the inspiratory or the expiratory part has more or less of the character of some of the varieties of bronchial, but in which the other half retains the vesicular character. In cases, however, in which the inspiration may be high or low-pitched bronchial, while the expiration may have nothing to distinguish it from vesicular, the breathing might be described as presenting high or low-pitched inspiration, with expiration harsh or soft, and not prolonged, or inaudible, as the case may be, and thus classification would be avoided.

This class of breathing is heard in some persons in the interscapular region, about the level of the third dorsal vertebra, owing to the proximity of the large bronchi, and the tracheal sound not being so much modified as where the interposing pulmonary tissue is greater. It is also sometimes heard over the manubrium sterni.

In abnormal conditions it indicates partial consolidation, and may be present at the apex in the early stage of tuberculous infiltration.

D. **Amphoric Breathing.**—This sound is not heard in the normal condition over any part of the respiratory system. It can be imitated by blowing into the mouth of any narrow-

mouthed vessel, and is caused by the mode of reflection of the vibrations from the walls of the vessel. It varies in pitch with the dimensions of the vessel.

It is heard over large pulmonary cavities into which a bronchus opens, and in pneumothorax if the opening between the lung and the pleural cavity be patent.

It is often confused with cavernous breathing, and care should be taken to strictly confine it to those cases where its specific character is distinctly marked.

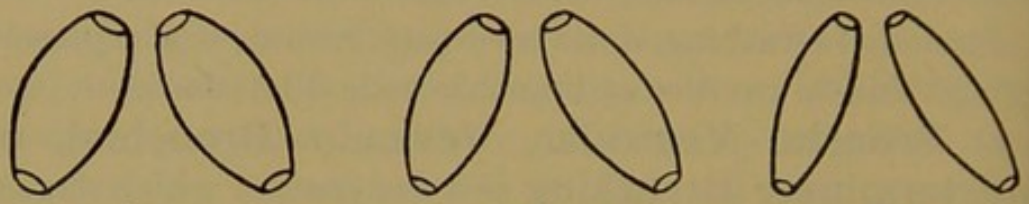


FIG. 71.—Diagrammatic representation of amphoric breathing.

Accompaniments.—In addition to the modifications or alterations in the character of the breathing which have been considered in the preceding pages, there are accessory sounds present in various morbid conditions which are known as accompaniments. They are produced in four different positions—(1) The bronchi; (2) the alveoli; (3) in pulmonary cavities; and (4) in the pleural cavity.

With the exception of the last, the term *râle* is applied to them all. Etymologically the word signifies a noise, but it is applied in a general sense to all accessory sounds in the air passages.

The various accompaniments have distinctive names as follows:—

A. **Rhonchi** are sounds of a whistling, sonorous, cooing, or squeaking character, produced in the bronchial tubes. The low-pitched ones are produced in the larger tubes, and are known as *sonorous rhonchi*, while the high-pitched ones produced in the smaller tubes are *sibilant rhonchi*. In both, the sounds are caused by—(*a*) Interference with the lumen of the tubes by an unequal thickening of the mucous

membrane; or (*b*) what is more common, by the presence of mucus lying in the tubes at different points; and (*c*) by spasmodic contraction of the bronchi. In all these the air, passing the constricted point into a wider part beyond, sets up vibrations which are heard as rhonchi, and may even be felt by the hand applied to the chest wall. They are represented in Fig. 72.

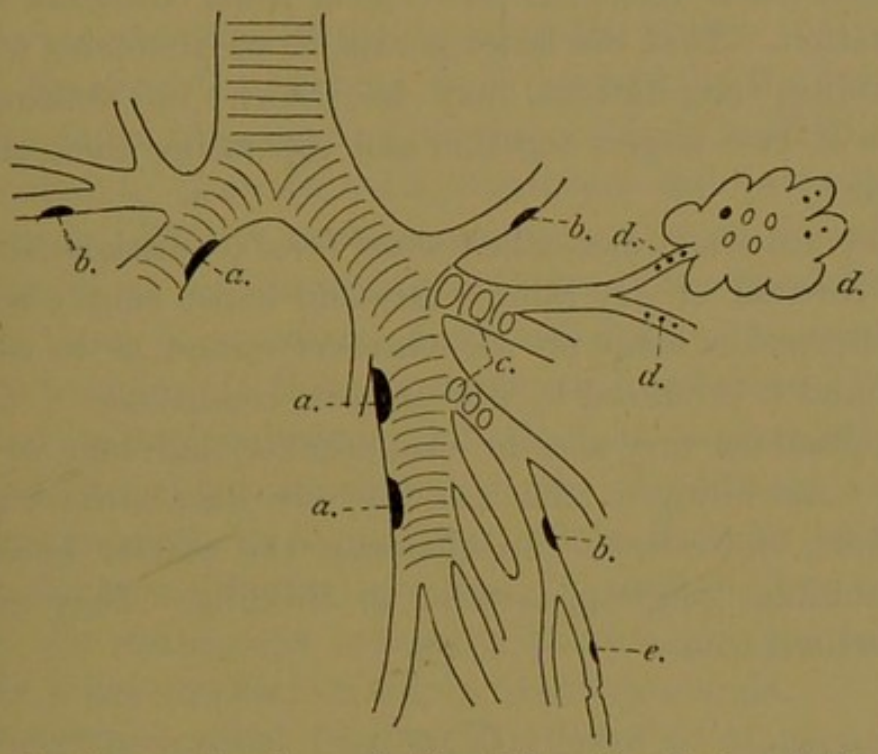


Fig. 72.—Representing the production of rhonchi, and of crepitations in the bronchi and alveoli,—at *a*. rhonchi from mucus in the large bronchi; at *b*. in the small tubes; at *c*. coarse crepitations in large tubes; at *d*. fine crepitations in small tubes and alveoli; at *e*. rhonchi from spasm of small tubes.

They are present either during inspiration or expiration, or both. From their character they are sometimes called *dry râles* to distinguish them from the next class.

Rhonchi are present in bronchitis, whether it be a simple bronchitis, or part of the pulmonic changes in phthisis, or due to congestion of the bronchial vessels from mitral lesion. They are also present in asthma, and in bronchitic asthma.

B. **Crepitations.**—This term is applied to sounds which resemble those produced when salt is thrown into the fire,

the bursting of the bubbles in boiling water, or by rubbing the hair between the fingers close to the ear. They are produced in the bronchi, in the alveoli, and in pulmonary cavities. They denote the presence of air and fluid, or viscid secretion in these positions, and are caused by—(*a*) The bursting of air bubbles as the air is drawn into or expelled from the lung, and (*b*) by the separation of the bronchial and vesicular walls from their contents during inspiration. That the latter is capable of producing a sound resembling crepitations may be shown by pressing the points of two fingers together and separating them close to the ear.

They are sometimes called *moist râles* to distinguish them from rhonchi or dry râles. Dry and moist simply indicate the impression made on the observer's mind as to whether the sound is produced by dry or moist conditions.

Crepitations vary also in size, number, and tone or character. According to their size they are *fine*, *medium* or *subcrepitant*, and *coarse*. In character the coarser kinds may be bubbling, gurgling, metallic or tinkling. They may be represented thus—

Fine crepitations. Medium crepitations. Coarse crepitations.

Fig. 73.—After Wyllie.

They are present mainly during inspiration, but sometimes during expiration also, and the part of respiration during which they are present ought to be noted.

Fine crepitations are heard in the first and last stage of croupous pneumonia; they are usually present in very great numbers, and give the impression of uniformity in size. They are also present in œdema and congestion of the lungs,

in which, however, they may be larger and belong to the subcrepitant variety. The medium or fine variety is present also in bronchitis affecting the smaller tubes. Coarse crepitations of bubbling or gurgling character are formed either in the large bronchi or in pulmonary or bronchiectatic cavities. Metallic or tinkling crepitations are so called from their metallic tone; they are produced in large pulmonary cavities, or in hydro-pneumothorax, their special character being due to the reverberation of the sound in the cavity.

C. **Friction** is a rubbing sound caused by the movement of one inflamed surface on another. Heard over the lungs it is due to pleurisy. Its coarseness and intensity vary widely in different cases; sometimes it is so fine as to be hardly, if at all, distinguishable from fine crepitations, at other times it is a coarse, loud sound, like the creaking of thick leather. If there is doubt as to whether a sound is pleural or intrapulmonary in origin, it can sometimes be settled by making the patient cough, when, if the sound be intrapulmonary, it is modified, while if it be pleural, no appreciable modification results. In addition, firm pressure with the stethoscope intensifies friction in some instances, while it has no effect on intrapulmonary sounds.

Pleuro-pericardial friction is referred to at page 52, and need not be again considered.

Other points will be found referred to at page 172, under pleurisy.

Vocal Resonance.—The signification of this term has been referred to already, and its intensity depends on the general laws enumerated at page 139.

It is liable to variations, which require to be further considered, and are the result of morbid changes in the lung and pleura, these acting either in the direction of intensifying or diminishing its loudness, or modifying its character.

Increase of vocal resonance may be so slight that it can only be described as *slight increase*; when, however, it is more decided, it is known as *bronchophony*, from its resem-

blance to the vocal resonance over the bronchi, as heard in the interscapular areas. It indicates that the vibrations set up in the larynx by speaking are conducted through the lung better than in the normal condition, that in fact changes have occurred in the lung which make it a better conductor of sound vibrations. Consolidation of the lung is therefore the condition in which it is most typically present; it is also present over collapsed lung, over cavities, and over a part where both consolidation and cavity formation exist. When very marked the resonance may be even more intense than over the trachea, this being due to the consolidated lung or the cavity acting as an intensifier of the voice sound.

Pectoriloquy is the term used to denote that intensity of vocal resonance which conveys the impression to the auscultator's ear of the voice being spoken into the mouth of the stethoscope, and which closely resembles the voice as heard on auscultating the larynx or trachea. This peculiarity is most striking when the patient whispers, and is then known as *whispering pectoriloquy*. It is present over pulmonary cavities and consolidated lung when connected with large bronchi.

Diminution or absence of vocal resonance.—Attention has already been drawn to the fact that vocal resonance is very imperfectly, if at all, distinguishable in some persons. The pathological change which, however, most commonly completely annuls it, is effusion into the pleura. It is also absent if the bronchus leading to a part be occluded, and secretion in the bronchi diminishes its intensity. In all these the vibrations are impeded and do not reach the chest wall.

Modifications in the character of the vocal resonance.—The first of these is *ægophony*, so termed from its bleating character. It is heard in some cases of pleuritic effusion, towards the upper limit of the fluid where it forms but a thin layer between the lung and the thoracic wall. Its mode of production is difficult of explanation, but it is most prob-

ably the result of an arrest or non-transmission by the fluid of the fundamental tone of the voice, while the harmonics are not arrested.

The second is *amphoric resonance*, which is fully referred to under Pneumothorax at page 176. It may also be present over intrapulmonary cavities.

The Physical Signs of some of the Principal Lung Affections

Bronchitis.—This is one of the commonest affections of the respiratory organs, and only presents distinctive features on auscultation. Inspection and percussion are not modified. On palpation bronchial fremitus can often be felt. The characteristic auscultatory signs are rhonchi, which vary in time and loudness in individual cases, and also at different times in the same case. They are, as a rule, best heard posteriorly in the interscapular regions and at the base. They are heard over both lungs equally. When the signs of bronchitis are limited to a part, as to one apex, it suggests a suspicion of phthisis.

Emphysema.—At page 132 the barrel shape of the chest in this condition has been referred to.

Expansion is much diminished, and in extreme cases is absent, especially at the upper part, the principal movement being an upward one of the chest as a whole, performed mainly by the action of the sternocleido-mastoid and scaleni muscles. When the patient coughs there may be marked distention of the supraclavicular regions and of some of the intercostal spaces close to the sternum, owing to the distention of the emphysematous lung tissue from the increased pressure during coughing.

The percussion sound is increased in clearness, and may even be somewhat tympanitic. The area of superficial dulness of the heart is diminished, and may be entirely absent, owing to the emphysematous lung interposing between the organ and the

chest wall. The line of superficial dulness of the liver on the right side is lower than normal; in fact, the clear percussion sound of the lung may be present as low as the edge of the ribs. Posteriorly the clear pulmonary sound is also lower than

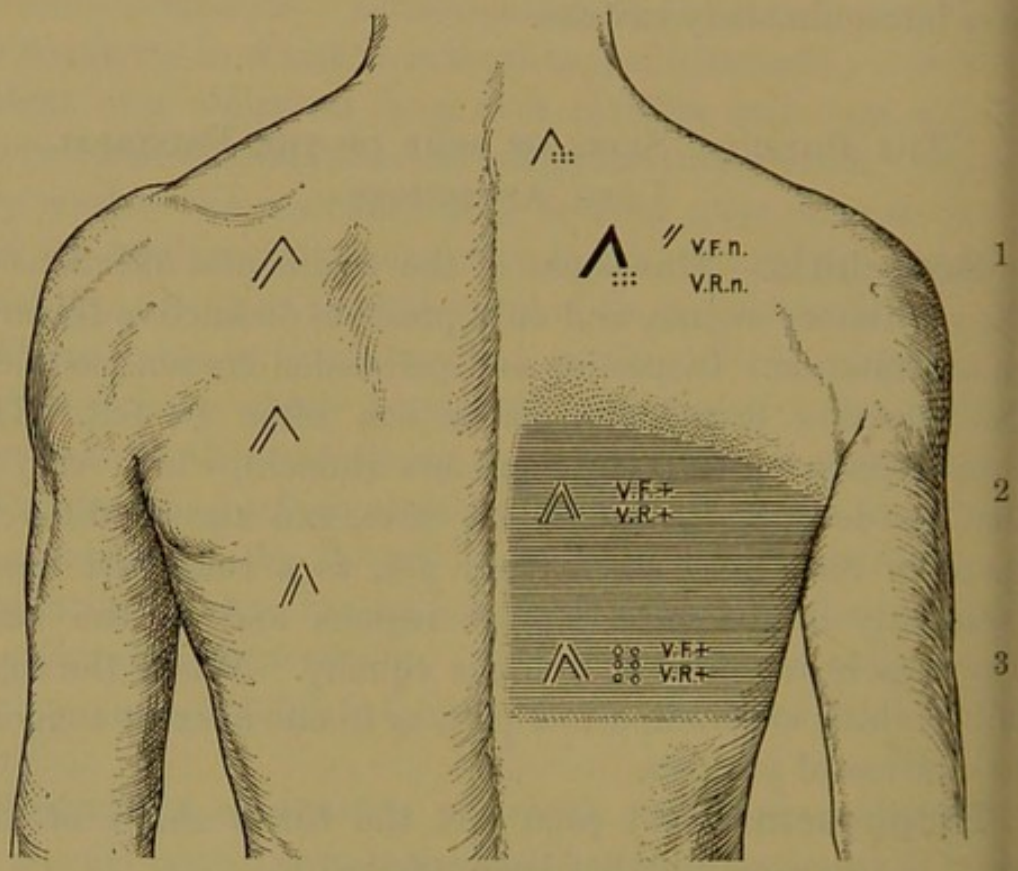


FIG. 74.—To illustrate the graphic method of representing the physical signs of pneumonia.

On the right side—(1) At upper part of lung normal or harsh vesicular breathing with fine crepitations at the end of inspiration and friction—stage of congestion. Stippled area represents area of hyper-resonance above consolidation. (2) Stage of hepatisation—high-pitched bronchial breathing with increased vocal fremitus and resonance. (3) Stage of resolution—high-pitched bronchial breathing with crepitations; increased vocal fremitus and resonance.

On left side broncho-vesicular breathing.

normal. Owing to the increased bulk of the lungs, not only is the thorax distended but the diaphragm is depressed, and consequently the liver is pushed down. The heart is also lowered in position, its axis is more vertical, and its pulsations may be visible in the epigastrium.

The signs present on auscultation have been variously described, and the want of unanimity appears to be due to the fact that the majority of cases of emphysema have, at the same time, bronchitis. When bronchitis is not present the breathing is vesicular in character and fainter than normal, but, on the other hand, if bronchitis be present, the respiratory sounds are harsh, and there are snoring or sibilant rhonchi, especially marked during expiration.

Owing to the obstruction which results in the pulmonary circulation the pulmonary second sound is accentuated, the right heart is dilated, the veins of the neck are distended, and there is more or less lividity.

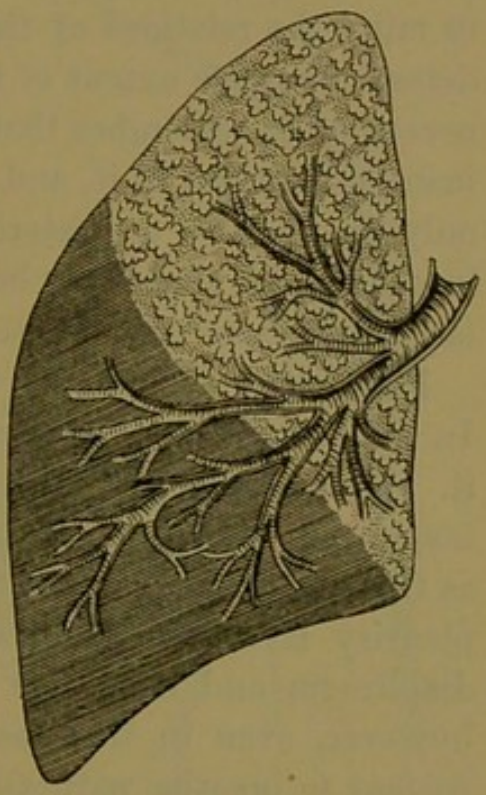


Fig. 75.—Representing pneumonia of the inferior lobe: physical signs characteristic of consolidation.

Pneumonia.—In the first stage (congestion and exudation) there is comparative percussion dulness, the pitch being higher than on the unaffected side; vocal fremitus is not increased; on auscultation there are vast numbers of very fine crepitations *towards the end of inspiration*, and vocal resonance is not increased. A deep breath or a cough makes the crepitations more numerous and more audible.

In the second stage (consolidation) there is absolute dulness on percussion; the vocal fremitus is increased; on auscultation the breathing is bronchial, in the most typical cases having the harsh *ch* sound, and called by some tubular, but in other cases it is more blowing: the sound is often louder than the respiratory sound heard over the trachea or cervical spine, the consolidated lung acting as

a resonator; vocal resonance is much intensified, being bronchophonic.

In the third stage (resolution) the physical signs more resemble those of the first stage, crepitations reappear, and are often present during both inspiration and expiration. In this stage they are known as *redux crepitations.*

As a rule pneumonia affects the whole lobe of one lung, and is strictly confined to it. It is thus necessary to bear in mind the relations of the different lobes to the surface in determining the extent of lung affected. It is also, however, necessary to remember that, while this is the rule, there are many exceptions to it, and that cases are met with in which only the anterior or posterior part of a lobe is affected, while in other cases there may be a comparatively small patch, this last being especially the case in old people.

Pleurisy.—The most typical sign of pleurisy is friction. In the early stage, however, it is not always possible to hear it. This is, in part at least, due to the pain caused by inspiration, which leads the patient to use the affected part as little as possible: this, for example, is effected, when the pleurisy is at the base, by preventing the action of the diaphragm and using the upper part of the lung. It may, however, even in this case be elicited by encouraging the patient to breathe naturally or deeply, or by coughing. In not a few cases the pleurisy begins on the diaphragmatic surface of the lung, when it is of course beyond the reach of auscultation.

Later, when effusion has occurred, there is dulness on percussion, absence of vocal fremitus, absence of breath sounds and of vocal resonance. By means of vocal fremitus it is as a rule possible to define very accurately the upper limit of the fluid. It is more accurate than percussion, because the compressed and airless lung above the fluid may give a dull sound.

The physical signs which are frequently present at the uppermost part of the effusion, or immediately above it, re-

quire to be clearly kept in mind, as they frequently lead to errors in diagnosis. The œgophony which may be present has been already referred to at page 168, and is not a misleading sign. Those which mislead are—(*a*) The presence of a *tympanitic percussion sound* above the dull sound caused

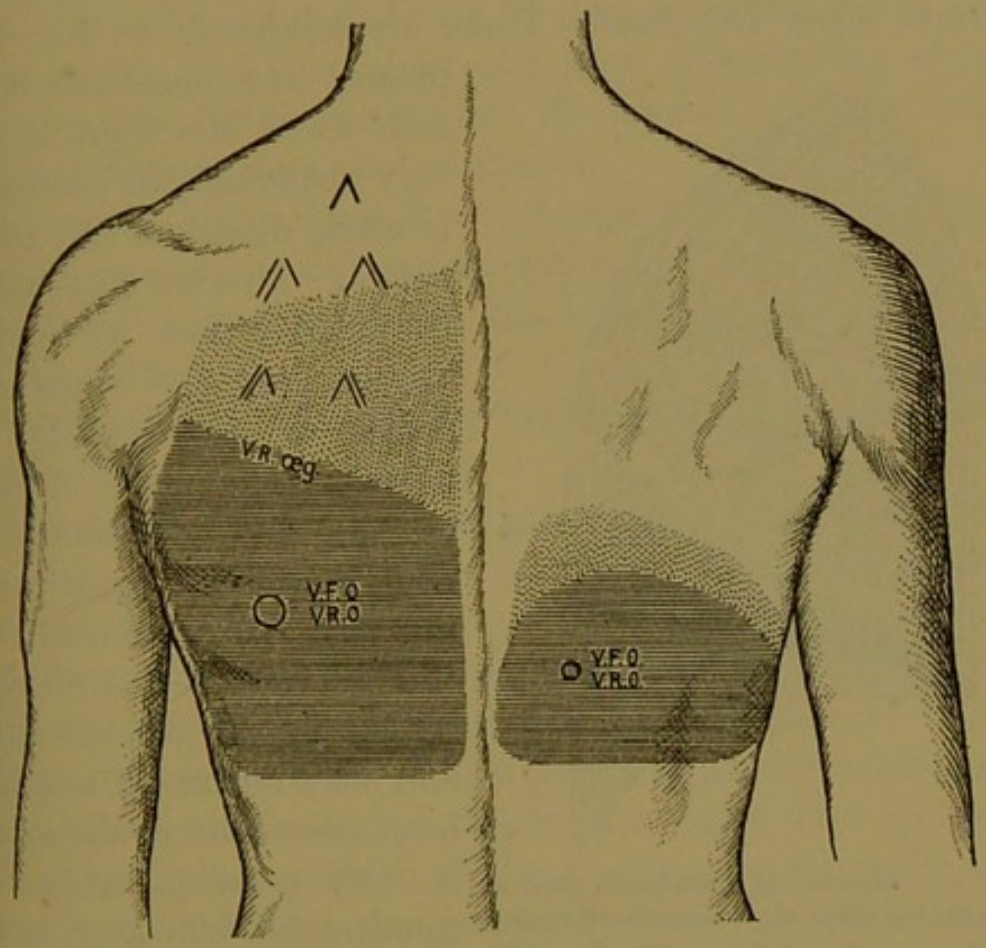


Fig. 76.—To illustrate the graphic method of representing the physical signs of pleurisy with effusion.

On left side, upper margin of dulness rising towards axilla; vocal fremitus and resonance absent; breath sound absent. Above hyper-resonance with broncho-vesicular breathing.

On right side, area of dulness highest in scapular line.

by the fluid. The sound is produced by the compressed but not perfectly airless lung. Its position is often determined by the pre-existence of pleural adhesions. If there are no adhesions the lung is compressed upwards and towards its root, and the tympanitic sound is immediately above the limit of the fluid; but if, for instance, the lung be

adherent anteriorly, it cannot be compressed upwards, but is compressed forwards, and over the upper part of this compressed area, which is not quite airless, and is lying against the anterior aspect of the chest wall, the tympanitic sound may be present and may be mistaken for pneumothorax. (*b*) The presence of *broncho-vesicular breathing and crepitations above the fluid.* These are produced in the compressed and congested lung, and when the fluid is not very abundant the condition is often mistaken for pneumonia. This not only occurs in pleurisy but in hydrothorax, and the signs disappear if the fluid be removed. The upper outline of the area of dulness in effusion into the pleura varies in form. In small effusions the dulness is first demonstrable in the scapular line, and may remain at a higher level there than at the spine. A very common outline in considerable effusions is for the upper edge of the dulness to rise in the axilla and show a lower level anteriorly and posteriorly. At times the upper outline is horizontal.

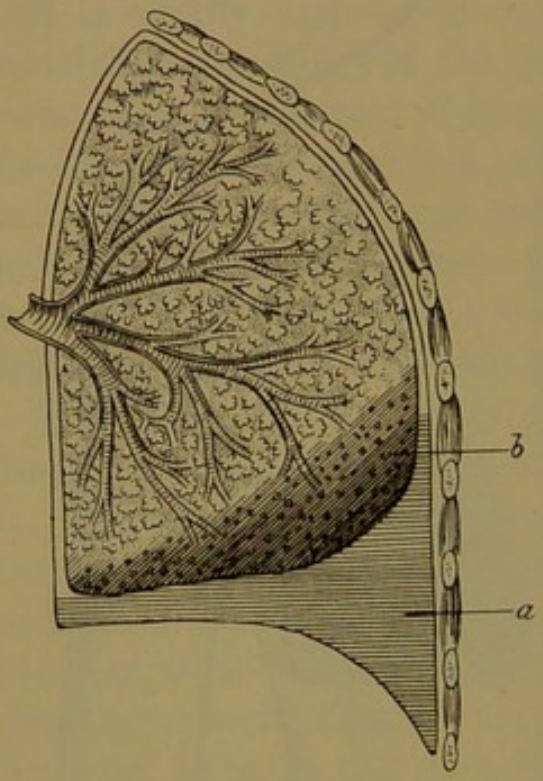


FIG. 77.—Showing at *a* moderate pleural effusion with, at *b*, bronchial breathing and crepitations over the compressed and congested portion of lung.

It is of great importance to learn to recognise the presence of moderate effusion into the pleural cavity, whether it be simply a hydrothorax or the result of pleurisy, and to do this it is necessary to be familiar with the percussion sound over the extreme bases of the lungs posteriorly, for dulness of a few fingers'-breadth in this position may indicate a very considerable effusion. Dulness, and the absence of

vocal fremitus, even should the breath sounds not be quite inaudible, often warrant the diagnosis.

When effusion is very copious the whole lung is compressed, the semi-circumference of the affected side is increased, and the intercostal spaces may be bulged. In addition, if the effusion be on the left side, the heart is displaced to the right, and may be seen pulsating below the right nipple, and the spleen is pushed downwards; if on the right side, the liver is pushed downwards, and the heart may be displaced somewhat to the left.

When the whole lung is not compressed, the breathing over the non-compressed part is puerile. When the greater part of the lung is compressed, the breathing over the other lung is puerile.

Phthisis.—Phthisis usually begins at the apex of one or other lung.

In the early stage it is characterised by diminished expansion and perhaps flattening, comparative dulness on percussion, breathing, which is harsh vesicular with prolonged expiration, interrupted vesicular or broncho-vesicular in character, crepitations, and increased vocal resonance. The crepitations may only be present in the supraclavicular and supraspinous areas or just below the outer end of the clavicle, so the auscultation of these regions ought never to be neglected. When the crepitations are abundant and present during both inspiration and expiration, the type of the breathing may be quite obscured, and when this is so it ought to be mentioned.

In the more advanced stages, when the changes in the lung and pleura are of a very varied kind, the physical signs also vary greatly. There are marked diminution in expansion, considerable flattening, and dulness on percussion, or if a large cavity be present at the apex, the cracked-pot sound may be elicited. Vocal fremitus is usually increased. The breath sounds are bronchial—high, medium, or low-pitched—or amphoric. The accompaniments are coarse

crepitations, often gurgling, metallic, or tinkling in character, and often, along with this, wheezing and squeaking sounds which belong to the type of rhonchi. The vocal resonance is bronchophonic, pectoriloquous, or amphoric.

The sounds vary greatly at different times, owing to the variable state of the alveoli, the cavities, and the bronchi. If the bronchi are filled with secretion no air may enter the affected part and little sound of any kind may be audible.

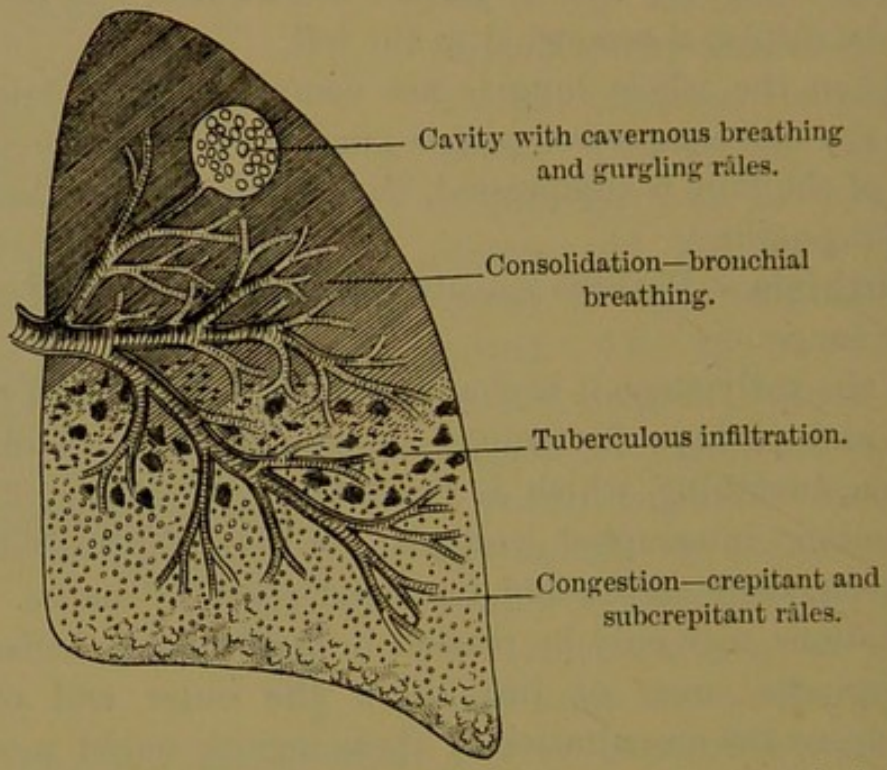


Fig. 78.—Showing phthisis at various stages in one lung, the physical signs depending on the stage.

If cavities are filled with secretion and their communication with the bronchi occluded, the coarse gurgling crepitations which may have been present at one examination disappear, while if the patient be asked to cough the sounds may again be produced.

Pneumothorax.—This term is applied to the presence of air in the pleural cavity, which may be produced by many causes. It most commonly occurs in cases of tuberculous disease of the lungs, the pleura becoming undermined and perforated by the tuberculous affection, so it can only occur

where adhesions have not been formed between its visceral and parietal layers. It may also result from pyæmic abscesses of the lung bursting through the pleura; from gangrene of the lung involving the pleura; and from wounds of the lung, either by puncture through the thoracic wall, or by laceration from the end of a broken rib. It may also be caused by violent respiratory efforts, but when this happens it is almost certainly owing to a weakened condition of the part where rupture occurs. In a considerable number of cases it is impossible to be quite satisfied as to the precise cause, for it supervenes in individuals who are *apparently* free from pulmonary disease, and who have not been subjected to strain or injury.

Inspection may show distinct *bulging* of the affected side, or of part of the side. Bulging of part of the side occurs in cases where partial pleural adhesions exist. Where the pleural cavity is completely obliterated by adhesions air cannot, of course, escape into it, but air may escape into a part which is not obliterated, and when this occurs the bulging is local. In some cases, however, there is no bulging. *Absence of respiratory movement* over the affected part is always noted. There may be slight up-and-down movement, but this must be distinguished from the true inspiratory movement, which is one of expansion.

Percussion.—The percussion sound varies in different cases, the variation depending on the size of the air-containing space, the degree of tension of the air within the space, and the patency of the perforation between the lung and the pleural cavity. The aperture tends to become closed by the approximation of its edges, as a result of the collapse of the lung, and also from the effusion of lymph round and over it. An aperture which has in this way become temporarily closed often reopens during a fit of coughing, from the violence of the expiratory act, and the increased intrapulmonary pressure which accompanies it.

When the air in the pleura is under high pressure, and

the aperture in the lung closed, the percussion sound is dull; when the aperture is patent, or the pressure not great, the percussion sound is clear, resonant, and may be distinctly tympanitic. In a considerable number of cases, however, the percussion sound is not sufficiently distinctive to be of much value. When the condition occurs on the left side, and causes displacement of the heart, we have noted that the tympanitic character was more distinctly marked over the normal cardiac region than elsewhere.

Palpation shows absence of vocal fremitus.

Auscultation.—The auscultatory signs are very variable, but often present phenomena of great interest. The ordinary respiratory sounds are absent; in fact, there may be an entire absence of any sound whatever when the ear is first applied. There may, however, be a sighing musical sound, resembling the sound made by the wind passing through a crevice. This may be present during inspiration or expiration, usually most markedly so during the latter, and it may continue after inspiration or expiration has ceased: the phenomenon is due to air passing through the aperture in the pleura, the aperture being in great part closed, a small chink only remaining open. If the patient be asked to cough, various changes may be noted: a respiratory sound may become audible, and be distinctly amphoric in character. The sound of the cough also may have the amphoric character,—that is, it sounds as if the patient had coughed into a chamber containing air only, the sound, however, being not only hollow in character, but also having a very distinct and pleasing metallic character. Further, a phenomenon which is known as metallic tinkling may be present: this sound exactly resembles the dropping of water on to the surface of fluid contained in a circumscribed space. It may be imitated by dropping water on to the surface of water in a water-bottle, and is often heard in house cisterns. This sound may be present when the patient is breathing quietly, but it may only be present after coughing. The number of

tinkles also varies; a solitary one after coughing may be all that is audible. The phenomenon is only present when there is fluid as well as air in the pleural cavity: it is produced by drops falling from the walls of the cavity on to the fluid, or by the bursting of air bubbles on the surface of the fluid. The explanation is that the act of coughing more or less shakes up the fluid, and splashes it up the sides of the cavity from which it drops down; or the disturbance of the fluid leads to the formation of air bubbles, which burst.

Another of the metallic phenomena which is often present is elicited by listening through the stethoscope while percussion is exercised with two coins. The sound which is then heard is usually referred to as the *bell sound*; as a rule, however, it has much more what Professor Wyllie has happily described as the *anvil sound*, for it has a very striking resemblance to the sound produced by striking with a hammer a smithy anvil, and heard at a distance. If the stethoscope be retained at a fixed point, and the coins be moved about, the limits of the air-containing space may be accurately defined, for as soon as the coins pass to a part of the chest which does not cover the air space the anvil sound is lost. This anvil sound cannot always be elicited, even in cases where there can be no doubt that pneumothorax is present; the precise meaning of this is somewhat obscure,

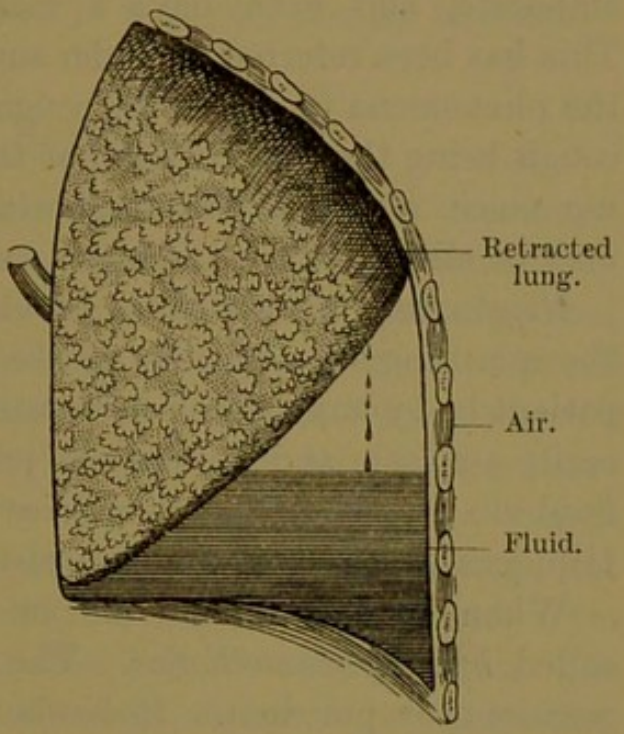


FIG. 79.—Showing the condition of parts in hydro-pneumothorax from a perforation in the pleura. Metallic tinkling is represented by drops falling on to the surface of the fluid.

but it probably depends upon the degree of tension in the air space, and the patency of the aperture in the pleura.

The various phenomena collectively termed metallic owe their special character, as has been already indicated, to the presence of an air-containing space; the sounds, however produced, acquire the metallic character from the mode in which reverberation takes place in the cavity.

Vocal resonance may be absent, but it may be distinctly amphoric, and even have a metallic character or clang. This has been referred to under auscultation when indicating the phenomena produced by coughing, the resonance of the cough being the same in kind as the resonance of the speaking voice, although the amphoric character of the former may be elicited when the latter cannot.

Hippocratic succussion is the term generally applied to the splashing sound which can be produced by shaking the patient or by any sudden movement on his own part. It is really a splash, and can only be produced when both air and fluid are present in the pleural cavity. It bears the name of Hippocrates, as his is the earliest description of it extant.

When both fluid and air are present the condition is called *hydro-pneumothorax*. The fluid may be serous, sero-purulent, or purulent. It tends to become purulent, even if not so from the beginning, but its character can only be definitely settled by an exploratory puncture with a hypodermic syringe. When fluid is present its position can be mapped out by percussion and auscultation, or by a combination of these. The percussion sound over the fluid is dull, there is absence of all respiratory sound, and the anvil sound is lost when the coins reach the part of the chest wall covering the fluid. The position of the fluid is of course at the most dependent part of the cavity, and alters with the position of the patient.

Displacement of organs has been already referred to. When the condition is on the left side, the heart may be felt beating as far to the right as the right nipple. The

mediastinum may also be displaced, a clear percussion sound being present as far as the right edge of the sternum. When the lesion is on the right side the liver may be pushed down, the heart displaced somewhat to the left, and the mediastinum also to the left. Dyspnœa is most distressing when the lesion is on the left side, the action of the heart then being more embarrassed. On whichever side the lesion exists the breathing power of the affected lung is annulled, and the circulation in it is very greatly impeded.

SPUTA

The sputum presents some important naked-eye characters, and in some cases requires to be examined microscopically.

It may consist of materials derived from the mouth, nares, or fauces, when its composition is not as a rule of much importance. The possibility of its origin in these situations has to be remembered, for patients not infrequently complain of spitting blood, a symptom popularly associated with serious lung disease, and therefore alarming, but which on examination may be found to proceed from the fauces or posterior nares. It is sputum from the respiratory tract which alone requires special consideration here.

The **quantity** of the sputum varies much. In persons affected with a slight bronchial catarrh, as evidenced by the presence of a slight cough in the morning, the coughing usually terminates by the expectoration of a little mucus of a greyish colour. In pronounced bronchitis and bronchiectasis, the expectoration may be very abundant, more than 1000 c.c. being coughed up in twenty-four hours.

The **reaction** of the sputum is alkaline. When, however, the sputum is contaminated with the contents of the stomach the reaction becomes acid, from the presence of the acids of the gastric juice.

The **consistency, form, and colour** differ much, and enable a division of the different varieties into—

The mucous.
The muco-purulent.
The purulent.
The serous.
The blood stained.

The **mucous sputum**, as seen at the beginning of an attack of acute bronchitis, is either quite glassy and transparent or greyish, and is viscid in consistency.

The **muco-purulent sputum** consists of a mixture of mucus and pus in varying proportions. The greater the proportion of pus present the less the transparency, till, if much pus be present, it becomes opaque and of a greenish colour. The pus may be evenly distributed through the sputum or may be aggregated into bullet-like lumps or masses known as *nummula*—the nummular sputum seen frequently in phthisis.

The muco-purulent sputum is by far the commonest form of sputum, and is met with during some part of almost every affection of the respiratory tract.

The **purulent sputum** consists of almost pure pus, and has the characteristic greenish appearance of pus. It is homogeneous and sinks in water. It is found in cases of lung cavity and perforating empyema.

The **serous sputum** is found typically in œdema of the lungs. It is copious, very fluid and frothy and of a clear grey colour. It may be slightly tinged with blood. Microscopically it is found to contain very few cell elements.

The **sanguineous sputum** may be any of the varieties mentioned, but is in addition tinged more or less deeply with blood. The blood may merely be in streaks through the sputum, as it frequently is when it comes from the upper air passages, or it may be intimately mingled, as in pneumonia. In croupous pneumonia the intimately mingled

blood, with glairy tough mucus, gives the sputum a uniform brown colour — thence the name the *rusty* sputum. In some severe cases of pneumonia it assumes a darker colour, the *prune-juice* sputum. The sanguineous sputum, again, may consist of almost pure blood, as in hæmoptysis in phthisis, mitral disease, etc. It is then, as a rule, bright red and frothy.

The **odour of the sputum** is usually of a mawkish character. At times it has a rancid, putrid odour, as in the last stages of phthisis. In gangrene of the lung and putrid bronchitis the odour may be intensely fœtid and offensive. In some individuals a very offensive odour is communicated to the sputum from the lacunæ of the tonsils; this may be misleading. Foreign bodies present in the sputum may give it a distinctive character, *e.g.* in the various forms of pneumoconiosis. Breathing an atmosphere constantly laden with carbon, as in the case of coal miners, leads to the production of a sputum varying from grey to black.

Microscopic examination of a thin layer of sputum will, as a rule, show abundant cell elements. Leucocytes are always present, in various stages of degeneration,—red blood corpuscles usually. Epithelial cells are numerous. Squamous cells are derived from the mouth and the surface of the vocal cords: cylindrical epithelial cells, in the form of mucus and goblet cells, from the bronchi. Ciliated epithelial cells are comparatively rare, and when present are usually derived from the nose, though they may come from the trachea: they are, as a rule, however, the result of commencing acute naso-pharyngeal catarrh. When destruction of the lung occurs, as in phthisis, *elastic fibres* may be found in the sputum. They are best demonstrated by boiling the sputum for a few minutes in 10 per cent caustic potash solution. The potash disintegrates all tissue except the elastic fibres. After boiling, the sputum is poured into a conical glass filled with water. On settling, the sediment is examined.

The fibres vary in length and breadth, have a double outline, are dark coloured and slightly curved. To have a decided diagnostic value as evidence of lung disintegration, they should exhibit an alveolar outline. When this is not present they may have been derived from the food. It is always advisable, before collecting the sputum for examination, to have the patient's mouth well washed out to avoid contamination.

Fibrinous casts of the bronchi are found in the sputum in fibrinous bronchitis and sometimes in pneumonia. They are whitish branched structures. They may be visible to the naked eye.

Spirals are small clear bodies like sago grains, very tenacious and easily distinguishable by the naked eye if the sputum be spread out on a thin plate of glass on a black surface. Under the microscope they usually show a central thread with around it a network of fine fibres looped in spirals, and overlaid with epithelium and possibly Charcot-Leyden crystals. They occur in the sputum of asthma, bronchitis, pneumonia, and pulmonary œdema.

Crystals.—Colourless pointed octahedral crystals—Charcot-Leyden crystals—are fairly common in asthma. Hæmatoidin occurs as ruby-red rhombic prisms, either solitary or in groups, or as needles or clusters of needles. They result from effused blood which has remained for some time in or about the air passages. When the crystals are enclosed within cells they point to a previous hæmorrhage, when free and in quantity to the bursting of an abscess into the respiratory tract. Cholesterin crystals in the familiar form of rhombic plates with a notch in one corner are sometimes met with in purulent sputum, especially when it results from the bursting of an abscess into the respiratory tract.

Fatty needles, tyrosin crystals, triple phosphates and oxalate of lime are all occasionally to be observed in the sputum in different respiratory affections.

Parasites.—Animal parasites are at times found in the

sputum. The most important is the echinococcus. Shreds of cyst wall and hooklets may be expectorated, and are easily recognised under the microscope. In the East the *Distomum pulmonale* occurs. Its presence causes attacks of hæmoptysis. The ova can be recognised under the microscope.

Amongst the non-pathogenic vegetable parasites are moulds, yeasts and fission fungi, such as *Sarcina pulmonis*, leptothrix, and various bacilli and micrococci. Of the pathogenic vegetable parasites, actinomyces may be present, but is rare in the sputum. The most important parasites of the sputum are the pathogenic organisms of pulmonary disease—the tubercle bacillus, the pneumonococcus, and from the fauces and upper air passages the *B. diphtheriæ*. These will be discussed under Clinical Bacteriology.

CHAPTER VIII

THE ALIMENTARY SYSTEM, INCLUDING THE ABDOMINAL VISCERA

The Mouth

The Teeth.—The state of the teeth is a matter of considerable practical importance, as a moderate number of good teeth is essential to efficient mastication, and imperfect mastication is one of the commonest faults in persons who suffer from various forms of indigestion.

The Gums present characteristic appearances in some diseases. In lead poisoning they show a blue line at their junction with the teeth. In scurvy they are spongy and bleeding, and in hæmorrhagic purpura they bleed on slight pressure. In anæmia they are pale and bloodless. In poisoning from mercury and other metals, and in syphilis, they may be ulcerated.

The Mucous Membrane of the cheeks and hard palate only exceptionally presents appearances of importance. In the eruptive fevers the rash characteristic of the special fever is usually present on it. In children, and in adults suffering from exhaustive diseases, aphthous patches may be present on the cheeks, palate, and gums. In Addison's disease sharply circumscribed patches of pigmentation may be seen on the mucous membrane of the lips and cheeks.

The Tongue.—The tongue has from time immemorial been scrutinised by the practitioner of medicine, and its appearances have been regarded either as an indication of the

general condition of the patient, or simply as an indication of the condition of the digestive organs, and more especially of the stomach and liver.

Leaving out of account the condition of the voluntary movements, which will be referred to more particularly under the nervous system, the points to note are as follows—(*a*) **Tremulousness.** — This may be the result of temporary nervousness, or due to organic changes in the central nervous system, or it may be part of a general condition, as in alcoholism and the acute febrile disorders. (*b*) **Size and shape.**—Some tongues are large and flabby, with a broad tip; others are firmer, and protruded with a pointed tip. The former often presents an indented edge, produced by pressure against the teeth. (*c*) **Humidity.**—The tongue may be dry, moist, or watery. In health the tongue is moist; it is watery in certain forms of indigestion; and it is dry in arrest of the salivary secretion, and in acute diseases. (*d*) **Condition of its covering.**—The appearances presented by the mucous membrane within the limits of health vary considerably in different individuals. The term *furred* is applied to the tongue when its surface looks as if it consisted of a fine fur. It may be whitish or brownish in colour; when the latter, it is frequently confined to the posterior part of the organ. A whitish fur is often diffused over the whole dorsum. When the papillæ project as red points through the fur, the tongue is known as a *strawberry tongue*, from its supposed resemblance to that fruit. The tongue is *coated* when there is a thick covering over whole or part of it, which may be whitish, yellowish-brown, or even black in colour. In addition, a coated tongue may be moist or dry—the combined dark and dry coating or incrustation only occurring in severe forms of constitutional disturbance. In some cases, after the coating comes off, the tongue is red, bare, and raw-looking; in others, in which there has been no antecedent coating, the tongue has a similar red and raw-looking appearance. (*e*) **Abrasions.**—The tongue further

may be the seat of cracks and fissures, these being the result in some cases of prolonged indigestion, but, when present in acute diseases, the result of the dryness of the organ combined with other causes.

The Pharynx

Few words need be spent upon a method of examination which is practised in the domestic circle as well as by physicians. In order to obtain a view of the pharynx the patient is seated before a window; better still, in such a position that a strong light can be thrown into the mouth by means of a laryngeal reflector. The tongue is then depressed, either with the handle of a spoon or an instrument specially constructed for the purpose, called a tongue depressor. A view is thus obtained of the anterior pillars of the fauces, the soft palate, uvula, and tonsils, while in the background is seen the posterior wall of the pharynx. It must be particularly noted whether the parts seen are normal in colour and size; the mobility of the soft palate should also be tested by making the patient phonate. The pressure of mucus in excessive quantity will at once make itself apparent should this condition exist, while sometimes the pharyngeal secretion shows a great tendency to dry up into more or less viscid crusts as soon as it is pressed out. We need not here refer further to the occasional presence of false membrane (as seen in diphtheria), masses of leptothrix, etc.

In young children who refuse to open the mouth the pharynx can often be examined by having their hands fixed, then compressing the nostrils and introducing the tongue depressor, when want of air compels the patient to separate the teeth or jaws.

As a matter of course, palpation with the finger, or even probing, may be necessary in certain cases of pharyngeal disease, but these methods are rarely required. If the parts be unduly irritable—as sometimes happens in those who

live freely or are of a gouty habit—it may be advisable to blunt the sensibility of the pharynx with cocaine before examination.

The Œsophagus

The presence or position of strictures has to be determined by means of œsophageal bougies, and their mode of use hardly falls to be considered here.

The Abdomen

Regions.—For convenience of description the abdomen is divided, as represented in Fig. 57, by two vertical and two transverse lines. The vertical lines are drawn upwards from the middle of Poupart's ligament, one on each side; while the transverse are drawn one at the level of the lowest part of the thorax, the second at the level of the highest point of the iliac crest. The regions thus marked out are represented in the diagram, and are from above downwards as follows:—

Right Hypochondrium.	Epigastrium.	Left Hypochondrium.
Do. Lumbar	Umbilical.	Do. Lumbar.
Do. Iliac.	Hypogastrium.	Do. Iliac.

The structures occupying these regions are enumerated in the ordinary text-books of anatomy.

Inspection.—By inspection the shape, the degree of distention, and the state of nutrition are observed. If there are local bulgings their position is to be specified. The presence and position of pulsations, and any engorgement of the superficial veins if present, are to be noted. The measurement of the circumference of the abdomen is taken at the level of the umbilicus. A record of the measurements taken at fixed periods is necessary in cases of ascites, etc.

Distention is due either to the accumulation of gas in the intestines, to fluid in the peritoneal cavity, or to tumour.

When due to the first of these, the abdominal wall is distended forwards, the distention being greater in the umbilical region; when to the second, the distention is most marked in the flanks, but if there is much fluid present there is distention forwards as well as laterally. This description is applicable mainly to the appearances in the recumbent position; when the sitting or the erect posture is assumed the lower part of the abdomen bulges, from the fact that the fluid changes with the change of position, and always gravitates to the most dependent part.

The upper part of the abdomen may be bulged by a distended stomach, or by an enlarged liver or spleen, but these will be more fully dealt with under their respective sections.

In the lower part of the abdomen the gravid uterus, uterine or ovarian tumours, and a distended bladder lead to local bulging. Large uterine and ovarian tumours lead to distention of the whole abdomen.

Laterally enlargements of the kidney may produce bulging.

Umbilical hernia, when present, is to be noted; it occurs not infrequently as a result of distention from ascites.

The epigastric veins may be unduly prominent from interference with the portal circulation, or from obstruction to the inferior vena cava.

In women who have borne children, or in whom the abdomen has been greatly distended from any other cause, there are white lines (*striæ albæ*) of scar tissue on the abdomen and upper part of the thighs.

On the other hand, a drawn-in condition or *retraction* of the upper part of the abdomen is seen in meningitis, especially in children.

Epigastric pulsation is met with in a variety of conditions; it may be cardiac, arterial, or venous in origin. Cardiac pulsation results from the visible impact of the right ventricle upon the parietes, as in hypertrophy and dilatation of the right ventricle; or in displacement of the heart down-

wards, as in emphysema of the lungs or intrathoracic tumour. At times the left ventricle may be found pulsating in the epigastric region in marked displacement of the heart to the right. When cardiac the pulsation is synchronous with the ventricular systole.

Arterial pulsation may arise from the visible pulsation of the abdominal aorta, the so-called "dynamic aorta" of nervous subjects, with thin and flaccid abdominal walls, from abdominal aneurysm, or from pulsation communicated from the aorta to an epigastric tumour, such as a carcinoma of the pylorus.

Venous pulsation is sometimes visible in the dilated hepatic veins in tricuspid regurgitation. Arterial and venous pulsations are somewhat delayed in time when compared with the apex pulsation of the heart.

The movements of the abdominal wall.—Normally the abdominal wall expands during inspiration, receding during expiration. A fixed abdominal wall is a valuable indication of pain, as in peritonitis. It may be local or general. In paralysis of the diaphragm the movements are altered, recession taking place during inspiration, some expansion during expiration.

Exaggerated peristaltic movements of the intestine may be visible in chronic intestinal obstruction. When the obstruction is at the ileo-cæcal valve, the distended coils of small intestine may be visible passing across the centre of the abdomen, forming the "ladder pattern," while collapse of the colon, causing flattening in the flanks, accentuates the appearance. In obstruction at the sigmoid flexure the distention is more general. A dilated and distended stomach may give rise to a tumour showing peristaltic movements.

Palpation.—After inspection the abdomen is palpated. To facilitate this proceeding the patient should lie flat on the back with the knees drawn up, and be requested to continue breathing quietly. This last point is of importance, for there is a tendency to hold the breath, and when this is

done the abdominal muscles become tense and resisting; it also takes the patient's mind away from the abdomen, and concentrates it on respiration.

The condition of the abdominal wall as regards the amount of subcutaneous fat, the presence of œdema, the lax or tense state of the muscles, is to be noted.

The characters of local bulgings are then investigated. Those due to affections of the abdominal wall have to be distinguished from those caused by intra-abdominal conditions. When due to the latter, they are usually elastic or hard, and are caused by enlargement of a solid organ, or a tumour, and the surface may be smooth, or nodulated, or umbilicated. The tumour may be fixed, or it may be movable, or it may move with respiration. Movable tumours are not very common, one of the commonest being a loose or floating kidney. Tumours which move with respiration are usually connected with the liver or spleen, for these organs move with the diaphragm during respiration, and tumours connected with them necessarily participate in their movements. This is an important diagnostic point. The movement may be visible, at other times it can only be made out by placing the hand, especially the fingers, flat and firmly on the part; and it is necessary to be sure that the diaphragm is itself moving freely, for patients are prone to impede its action when the abdomen is palpated, and if its action is arrested during examination the observations made will not be reliable.

The presence of fluid, as ascertained by palpation, is fully described under ascites.

The limits and characters of pulsations are also to be noted.

Percussion.—The greater part of the abdomen is normally tympanitic, but if the intestine be filled with solid matter or with fluid at any part, the sound is dull; this is specially noticeable over the great intestine when it is loaded with fæces. The difference in pitch over the stomach, large

intestine, and small intestine respectively, can sometimes be distinguished, but it requires much practice, and is surrounded by many possibilities of error. The percussion sound is also dull over fluid, over the solid organs, as the liver and spleen, over a distended uterus or bladder, and over all abdominal tumours, if not overlaid by intestine. The tympanitic percussion sound over the stomach and intestine varies considerably even under normal conditions. It depends upon two factors—the tension of the contained air and the depth of the column of air. Speaking generally, the higher the tension of the contained air the higher the pitch of the sound. In two air spaces under similar tension a sound of higher pitch will be given by the space containing the lesser volume of air. Thus under normal conditions a sound of higher pitch will be given over the small intestine than over the colon.

We have now to deal more in detail with the physical examination of some special morbid conditions of the abdominal cavity and of the various abdominal organs.

Ascites.—By this term is meant accumulation of fluid in the abdominal cavity.

Inspection.—The abdomen is distended, the degree varying with the quantity of fluid. When the quantity is small the anterior surface is normal or slightly rounded, and there is lateral bulging in the flanks. If the patient be lying inclined to one side, the bulging is greatest on the side towards which he is inclined; if he be in the sitting or standing posture, the bulging is greater at the lower part of the abdomen. When there is much fluid the roundness of the anterior aspect, and also the bulging of the flanks, are greater; the upper part, including the epigastric and both hypochondriac regions, is also distended, even the lower ribs being pushed outwards; in short, the whole abdomen becomes barrel shaped and the abdominal wall exceedingly tense. In addition, the umbilicus is stretched, and often projects beyond the surrounding surface. Umbilical hernia

not unfrequently follows upon ascites, especially if there is much cough, for the right and left rectus abdominis muscles are separated by the abdominal distention, and coughing favours protrusion in this situation, as it is one of the weak points in the abdominal wall. When there is much distention the pressure on the inferior vena cava and the iliac veins leads to congestion of the veins of the lower extremities, and as a consequence to œdema.

Percussion.—When the patient is on his back the percussion sound over the anterior aspect is clear and tympanitic, as the intestines float on the fluid and are in contact with the abdominal wall in this situation. In extreme degrees of distention the clear percussion sound is only present above the umbilicus, owing to the intestines being pushed up by the fluid, while above this the liver dulness reaches a higher point than normal, the thorax being encroached on by the upward pressure of the intestines, liver, and diaphragm. The flanks, the lateral parts, including the hypochondriac, lumbar, and iliac regions, and the hypogastrium, are dull. This is due to the fluid gravitating to the posterior, lateral, and lower parts of the cavity, the percussion sound over fluid always being dull. If the patient be turned on the side the level of dulness on the dependent side rises, that is, it approaches nearer the middle line, while a tympanitic, or at least a clearer, sound is present over the elevated side: this is due to the fluid being free in the abdominal cavity, and changing with the position of the trunk. A similar phenomenon is produced by moving the patient from the recumbent to the sitting posture, the limit of dulness in the hypogastrium then rising higher, that is, towards the umbilicus.

While the foregoing is the result of percussion in the majority of instances, cases are frequently seen in which, notwithstanding the presence of considerable ascites, there is a tympanitic sound on percussion in one or other flank; this is to be explained by gaseous distention of the colon

bringing it into contact with the abdominal wall at the part where the tympanitic sound is obtained.

Palpation.—The abdomen has an elastic feeling. If one hand be placed flat over one lumbar region, while the opposite lumbar region is tapped with the fingers of the other hand, a distinct wave-like impulse is felt when much fluid is present. It is well in carrying out the examination to exclude error from a wave in the abdominal wall, produced by the percussion, by an assistant placing a hand vertically on the abdominal wall in the middle line. If the quantity of fluid be small, the wave may not be obtained from side to side, but it can be elicited by a similar proceeding applied to the lateral aspect by, for instance, placing one hand over the iliac region while the lumbar region of the same side is tapped with one finger of the other hand. The impulse may be caused either by the fluid or by the intestine floating on it, but it can only be produced when fluid is present. A true wave is not obtained in flatulent distention.

Conditions which produce ascites.—Ascites is caused by valvular lesions of the heart, more especially mitral stenosis and incompetence, with secondary tricuspid incompetence and general venous engorgement; by diseases of the lungs interfering with the circulation through them, and producing tricuspid incompetence and general venous congestion; and by kidney disease, more especially the chronic parenchymatous form with cardiac failure. In addition, there are conditions which act directly on the portal circulation and obstruct it. These are obstruction—(*a*) *Within* the liver, from cirrhosis or tumours; (*b*) *without* the liver, from the pressure of a tumour or from phlebitis of the trunk of the vein. In all these, with the exception of nephritis, the pouring out of the fluid is the result of venous congestion, and is a purely passive process.

Fluid may also be present in the abdominal cavity as the result of inflammation, or as the result of irritation from

diffuse malignant disease of the peritoneum. Tumours of the abdomen, especially solid tumours of the ovary, are often accompanied by ascites. The fluid in ascites may be clear or purulent or coloured with blood, and, if jaundice be present, bile-stained.

Ascitic fluids may be divided into transudates and exudates. Transudates are transparent yellow or greenish fluids, containing few cell elements. Speaking generally, they do not coagulate on standing, or, if they do, only after a long interval. If blood be present from accidental contamination, a gelatinous or membranous coagulum of fibrin may form. The amount of total solids in a transudate is small, the proteid percentage is at a maximum 1·5 to 2 per cent., and the specific gravity is low, 1012 or under.

Exudates, when serous, are like transudates in colour, but are richer in cellular elements. They coagulate spontaneously on standing, in twenty-four hours at latest. They have a higher specific gravity and a higher percentage of albumin. Peritoneal exudates have as a minimum a specific gravity of 1018, and a proteid percentage of 4·0–4·5. Ascitic fluid may assume a milky appearance—chylous ascites, when there is rupture of a lacteal. The microscope then shows the presence of oil globules. It may also assume a milky appearance in such conditions as tuberculous peritonitis, when there is no evidence of rupture of a lacteal, and no oil globules can be discovered under the microscope. This chyliform ascites has been ascribed to fatty degeneration of the cellular elements, or to the presence of lecithin.

Peritonitis.—Peritonitis is inflammation of the peritoneum. In addition to pain and general constitutional disturbance, there are physical indications of its presence. The patient lies on the back with his legs drawn up, in order to relax the abdominal parietes as much as possible. Respiration is almost entirely if not altogether thoracic, as any movement of the diaphragm increases the pain by the movement communicated to the abdominal viscera. The abdomen

is distended by the accumulation of gas in the intestines. This is known as *meteorism*, and results from paralysis of the muscular coat of the intestine, consequent on the inflammatory action. A further result of the paralysis is constipation. The abdomen, as might be anticipated, is tympanitic, but percussion must not be used more than is absolutely necessary, as there is extreme tenderness. In severe cases there is effusion of fluid, and if it is present in any quantity the physical signs indicative of its presence may be obtained; it does not, however, so readily change its position as in ascites from more mechanical causes, owing to the gluing together of the coils of the intestines to one another, and to the parietal peritoneum. In the form of peritonitis known as *tabes mesenterica* the enlarged abdomen has a doughy feel, and the enlarged glands may be felt.

The Stomach, which is flask or pear-shaped, occupies part of the left hypochondrium and the epigastrium. It is situated below the diaphragm and the liver, and above the transverse colon. A small part of its anterior surface is frequently in contact with the anterior abdominal wall.

The **cardiac orifice** lies behind the seventh costal cartilage an inch to the left of the sternum, and at a depth of about 4 in. from the surface. The **pyloric orifice** is about 3 to 4 in. below the xiphi-sternal articulation, and, if the stomach be contracted, in or immediately to the right of the middle line; when the stomach is distended, the pylorus moves to the right. The pyloric orifice is much nearer the anterior surface than the cardiac.

The superior limit of the **fundus of the organ** is found in the dome of the diaphragm at the fifth interspace in the mammary line. It is a little above and behind the apex of the heart. When the organ is moderately distended, the greater curvature reaches the level of the infracostal line.

Traube's halfmoon-shaped space is that portion of the thoracic wall which is in contact with the stomach, below the level of the lung and between the liver and spleen. In

health this space gives a tympanitic stomach sound to gentle percussion, in pleural effusion the sound is dull.

Inspection.—In the normal state inspection gives no information as to the state of this viscus. If, however, it be greatly distended, it leads to bulging of the abdominal wall, and its outline may be traced by the eye.

Percussion of the stomach is difficult, and at times gives unsatisfactory and misleading results. By percussion we desire to delineate the boundary between the lung and stomach, the liver and stomach, and the position of the greater curvature. On percussing over Traube's space and in the epigastric region a tympanitic stomach sound will be obtained. Using gentle percussion the delineation of the boundary between lung and stomach and liver and stomach can be got by percussing from below upwards. The position of the greater curvature is next got by using gentle percussion and percussing from below upwards in the parasternal line. The position of the greater curvature varies with the state of distention of the organ. In moderate distention it lies about the level of the infracostal line. On percussing from below upwards a tympanitic sound will be got from the small intestine, the colon, and the stomach, but a difference in the pitch will be noted, the pitch of the stomach sound, if the organ contain gas, being identical with the pitch obtained over Traube's space. If the stomach contain much fluid the sound from the lower border may be dull. The percussion of the fundus of the stomach where it rises into the dome of the diaphragm and is covered by lung is not satisfactory, but may be elicited by strong percussion, the sound obtained having a muffled tympanicity. The student must remember that in percussion of the stomach the important point is to delineate the position of the greater curvature. The position of the other boundaries is dependent upon the condition of the neighbouring organs; thus, if the liver be enlarged, the position of the liver boundary is altered, if there be pleurisy with effusion

Traube's space is obscured, if there be chronic fibroid phthisis with retraction of the lung the area is increased. The position of the greater curvature is variable, but unless the stomach be dilated or displaced it is never below the umbilicus.

By percussing to the left, the outer limit of the great curvature can often readily be defined. If, however, the colon be distended with gas, the delimitation of this border is more difficult, but may be made out by noting a change in the pitch of the sound as we pass from stomach to colon. From this it will be understood that the distended fundus lies under the costal cartilages and the ribs, below and to the left of the cardiac dulness, as represented in Fig. 80. The precise area over which tympanicity may be elicited varies, of course, with the degree of distention of the stomach with gas, the greater the distention the larger the area of tympanicity.

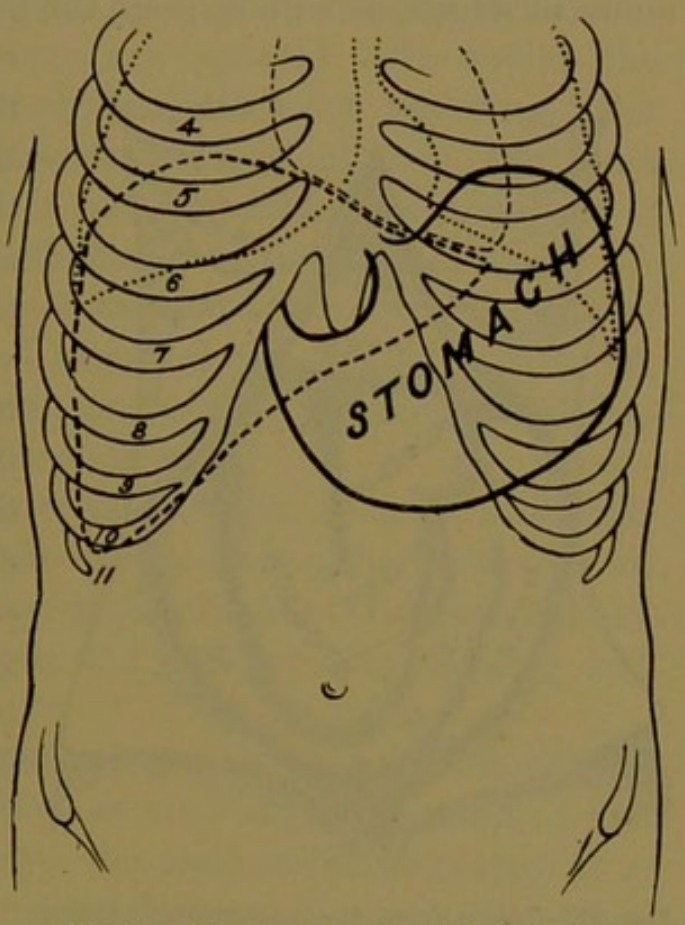


FIG. 80.—Showing a moderately distended stomach.

To accurately delineate the stomach by percussion it is frequently necessary to distend the organ with air. This can easily be done with the stomach tube. If the greater curvature of the stomach be found below the umbilicus it indicates displacement or dilatation.

The stomach may be so enlarged that it occupies a great

part of the abdominal cavity, reaching even as low as the pubes. Its condition, however, varies. It may be blown up like an inflated bladder, and project forwards when the abdomen is opened, or it may not be distended to anything like this degree, but, instead, be stretched and dragged downwards. This latter condition is illustrated in the following diagram, taken from a case in the post-mortem room, in which, as will be seen, the greater curvature of the stomach was as low as the pubes. Owing to the practical fixity of the cardiac orifice, and comparative fixity of the pylorus, when the viscus becomes stretched in its longitudinal diameter, without being distended in circumference, it assumes the U shape seen in the diagram. The diagram represents an extreme case, but minor degrees of the same type are commonly seen.

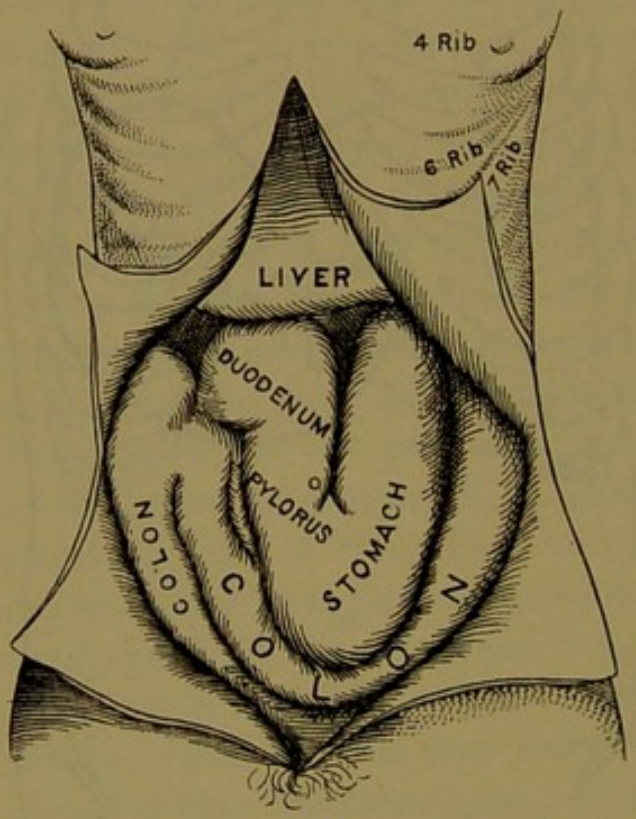


FIG. 81.—Taken from the post-mortem room, showing the U shape often assumed by the enlarged stomach.

The stomach may be dislocated — displaced without being dilated, the condition known as gastro-enteroptosis. It is impossible to recognise this with certainty by percussion unless the organ be first distended with air or water, when its exact position can be delineated. The transverse colon is displaced with the stomach; in the case from which the diagram was taken it was in the pelvis.

Palpation.—When the stomach is the seat of ulceration or malignant growth there may be pain or tenderness on

pressure; this, however, always leads to a tense state of the abdominal muscles, which hinders a satisfactory examination being made. The tumour of a malignant affection of the pylorus can at times be distinctly felt; it is known by its anatomical position, and by the signs of obstruction present.

By palpation of the stomach, when it contains both fluid and gas, a splashing sound may be elicited. Both hands are placed on the abdomen, and by a gentle shaking movement the contents of the organ are agitated. In health the splashing sound may be elicited above the umbilicus after a meal, when a considerable amount of fluid has been imbibed. If the sound is obtained *below* the umbilicus it indicates displacement or dilatation of the stomach.

The causes of dilatation of the stomach are pyloric obstruction, gastric catarrh, and atony of the muscular coat. A degree of dilatation is common in cases of general debility, irrespective of its immediate cause.

Diminution in the size of the stomach occurs to a marked degree in obstruction at the cardiac orifice, the most common cause of stricture being malignant disease. It also occurs in old and debilitated people, and in some long-standing and exhausting diseases.

Auscultatory percussion of the stomach is one of the most satisfactory methods of determining the borders of the stomach. The receiver of the stethoscope is placed at a point where a tympanitic stomach sound is got on percussion at Traube's space, or better, at the angle between the xiphisternum and the costal margin. Percussing very gently near the stethoscope the sound is auscultated and noted. Keeping the stethoscope in its original position, the observer begins at the lower part of the abdomen and percusses upwards towards the greater curvature of the stomach. Whenever the pleximeter finger reaches the stomach the sound heard can at once be recognised as of the same pitch and quality as the original stomach sound. By this method the organ can be delineated. It depends upon the fact that

vibrations set up within the column of air in the organ resound all through the organ.

The Small Intestine. — **Inspection.** — In intestinal obstruction gas accumulates in the small intestines, and leads to great distention of the abdomen; and if the abdominal wall be thin, the position of the coils of intestine is shown on it in transverse ridges and furrows, the "ladder" pattern. When this appearance is present it often denotes that the stricture is at the cæcum or lower part of the ileum. The degree of distention may sometimes help to indicate roughly the position of the stricture, for the nearer the stricture is to the duodenum the less is the distention. The vermicular movement of the intestine may also be visible through the emaciated parietes.

Palpation.—In thin subjects hard fæcal lumps are readily felt in the small intestine; they must not be mistaken for tumours.

Percussion.—In the normal condition the percussion sound is tympanitic; when greatly distended with gas, as in peritonitis and obstruction, the sound is lower in pitch but retains its tympanitic character.

The rumbling sounds produced in the intestines are known as *borborygmi*, and are caused by the passage of gas and semi-fluid matters.

The Colon.—The position of the various sections of the colon is represented in Frontispiece I. It is necessary to bear in mind that the posterior aspect of the ascending and descending portions are not covered with peritoneum, and are united to the posterior abdominal wall by loose connective tissue. This is important, as purulent inflammation set up, say, at the cæcum, may spread upwards in this tissue without implicating the peritoneum; while, on the other hand, a purulent inflammation beginning in the loose connective tissue round the kidney may extend downwards behind the colon, and reach the cæcum or sigmoid flexure.

Owing to the position of the transverse colon, immediately

inferior to the stomach, distention of it with gas gives a percussion sound which often cannot be distinguished from that of the stomach. Not infrequently the colon is distended when the stomach is unusually small, and when this is the case its percussion sound is almost certain to be mistaken for that of the stomach. In some cases, however, a difference in the pitch or in the amplitude of the percussion sound between the two structures may be made out. While the differential percussion of stomach and transverse colon is thus surrounded by possibilities of error, we may by repeated examination satisfy ourselves as to the condition of the stomach. Perhaps the most conclusive proof that it is the stomach with which we are dealing, is to elicit the splashing sound referred to under the examination of that viscus. The combined method of auscultatory percussion will also give valuable information.

Malignant stricture of the colon may occur at any point, but its most common sites are the cæcum, the hepatic, splenic, or sigmoid flexures. In all these the distention of the part above the stricture aids the diagnosis, and can be made out by palpation and percussion. The physical examination of the colon is of great value in other cases, for patients are often found to have one of its sections, most commonly perhaps the ascending portion, loaded with fæces, and if the condition is not recognised we miss giving them the relief they seek. The outline of the distended ascending transverse or descending colon can be made out with sufficient definiteness by means of palpation and percussion, but it must not be expected that it has the hardness and firmness either of a solid tumour or of an enlarged liver; it is more doughy in feeling than either of these. When distended it pushes the small intestine aside, and comes much more to the front than in the normal condition.

The Cæcum, with the vermiform appendix, is a part of the colon which requires special attention, as it is peculiarly liable to inflammatory attacks,—the condition is known as

appendicitis. Tenderness is then present at McBurney's point, midway between the anterior-superior spine and the umbilicus; there is muscular rigidity and increased sense of resistance, and a tumour may be recognisable in the cæcal region.

A gurgling sound can be elicited in the cæcum by palpation, in typhoid fever, but also in other and less important conditions: it simply denotes the presence of fluid or semi-fluid matter and gas in the part, and for obvious reasons it is not a desirable sign to attempt to elicit in typhoid fever.

The Anus and Rectum.—The anus and rectum have often to be examined; the former for piles, fissures, fistulæ, condylomata, etc. The rectum is frequently the seat of cancer, and in all conditions where there are complaints, specially referred to the lower bowel, it is desirable to make a digital examination; more especially is this necessary if treatment has not been successful in removing the symptoms complained of. Malignant stricture or malignant growth is frequently within reach of the finger. Another condition which we have seen producing extreme discomfort is the presence of a large and rounded scybalous mass in the rectum, coming down whenever the patient went to stool, and in which the history of diarrhœa and straining were extremely misleading. Such a mass must be broken down by the finger, and its hardness makes this no very easy or agreeable task.

The Sigmoid Flexure is situated in the left iliac region: accumulation of fæces in it is therefore within easy reach of palpation. When stricture occurs below it, it becomes enormously distended with fæces or gas; we have seen it so greatly distended as to occupy the lower part of the abdomen, stretching across to the opposite side, and even occupying the right iliac region.

The Abdominal Lymphatic Glands.—In some cases of malignant disease of the abdominal organs, the enlarged mesenteric and retroperitoneal glands are felt, and aid in

forming the diagnosis. In tuberculous peritonitis the enlarged glands may also be felt. They are also enlarged in cases of diffuse tuberculous disease of glands, without being necessarily accompanied by peritonitis; also in lymphadenoma.

The Liver.—The anatomical position of the liver is represented in the Frontispieces. Its superior surface is in contact with the diaphragm from before backwards. Its anterior, posterior, and lateral aspects are in contact with the ribs and the abdominal wall. Superiorly, where it lies under the right vault of the diaphragm, it reaches the level of the fourth space or lower border of the fourth rib. Its inferior limit practically coincides with the lower edge of the ribs, except in the epigastrium, where it crosses the subcostal angle from the ninth right to the eighth left costal cartilage about midway between the base of the xiphoid process and the umbilicus, but the precise position of this part of the lower edge varies greatly in different individuals. The organ in its upper part has the lung interposed between it and the thoracic wall; it is, however, uncovered by lung, and comes into immediate contact with the parietes, where the lung ends, and in the mesial line, at the base of the xiphoid process.

The exact position of the liver varies with respiration, descending with the descent of the diaphragm and receding with its ascent.

It will aid in the comprehension of the position of this organ and its variations if it be remembered that, as Symington has pointed out, and as is seen in the Frontispiece and in Fig. 80, it is wedge-shaped, and that the long edge of the wedge runs along the lower margin of the ribs on the right side, and leaves it to cross the epigastrium obliquely.

Inspection.—Great enlargement of the liver leads to fulness and projection of the ribs covering it, and of the adjoining part of the abdomen. The projection of large malignant nodules, a hydatid cyst, or an abscess may also be visible.

Palpation.—In the normal condition the edge of the liver cannot, as a rule, be felt; this is true even of palpation in the epigastrium, owing to the thinness of its edge, and the little sense of resistance consequently given by it. When, however, the organ is enlarged, the part of its surface in contact with the abdominal wall and its interior edge can be readily felt. It can also be felt when it is displaced downwards by the pressure of effusion into the right pleural cavity; and in pronounced cases of emphysema, the emphysematous lung pushing both the diaphragm and it downwards. When enlarged the surface may be smooth or nodulated. If the enlargement is due to congestion, fatty infiltration, or waxy degeneration, the surface is smooth; when to malignant disease, it is usually more or less nodular, and in some cases the umbilication of the nodules is felt. Over a hydatid cyst or an abscess there is fluctuation, or a feeling of elasticity. When the abdomen is distended, but not tensely, by fluid or gas, the anterior surface of the liver, if enlarged, may be felt by placing the fingers firmly over it and then suddenly pressing inwards; by this means the fingers are brought into sudden contact with the hard and firm organ, and we are conscious of the difference between it and the softer parts overlying it. The edge of the enlarged organ may be felt under similar conditions by pressing the fingers first inwards and then suddenly upwards; this may be done with either hand, the observer standing opposite the thorax of the patient.

Percussion.—Percussion of the liver gives two areas of dulness,—deep or comparative, and superficial or absolute dulness.

The **deep or relative dulness** is found by beginning to percuss in the second or third right intercostal space in the mammary line. We begin at that point to be sure of getting a good lung sound for purposes of comparison; we then percuss downwards, space by space, until *the slightest difference* in the character or the quality of the sound is observed,

and this point indicates the position of the uppermost part of the right lobe as it lies under the vault of the diaphragm and behind a considerable thickness of lung tissue. If greater precision be required, the two ribs above this space may be percussed to see whether the exact line be the space, or the rib above it. Here, as in the percussion of the lungs, ribs must not be compared with interspaces. The difference in the sound is slight, and is therefore called *comparative dulness*, but its accuracy may be relied upon, for we are in the habit of frequently checking this on the post-mortem table. This upper limit is almost invariably placed at too low a level. Percussion *requires to be strong*, as in the percussion of all solid structures lying behind air-containing organs. If desired, the same procedure may be followed on the lateral and posterior aspects of the thorax; but this is not as a rule done, although in some cases, especially in hydatid tumours or abscesses developing from its superior surface, it is of great service, as it indicates the presence of these projections, while they are beyond the reach of palpation. To find the inferior limit, percussion is begun in the nipple line, sufficiently far down in the abdomen to ensure getting a tympanitic sound, and continued upwards until a very slight difference is observed: this marks the lower edge. Percussion in this case *must be very light*, as the lower edge of the organ is thin and lies over air-containing intestine, and if not exceedingly light the sound from the intestine is brought out and quite masks the dulness of the liver. The measurement between these two points is that which is usually given as the *vertical liver dulness.*

The **superficial** or **absolute dulness** is taken from the point where the lung ends, so that what is really necessary in this case is to find the inferior margin of the right lung in the mammary line. This is done by percussing from above downwards, with extreme lightness, until all trace of clearness disappears, and the sound is absolutely dull even to light percussion. Now it will be found that from the point

where the percussion sound changes slightly to the point at which it becomes absolutely dull, the sound presents several degrees of dulness, but, if the percussion is sufficiently light, there need be no hesitation in proceeding downwards until all trace of clearness is passed. The lower edge is taken in the manner already described. The vertical measurement in the mammary line, as in deep dulness, is the one given and is known as the *superficial dulness.*

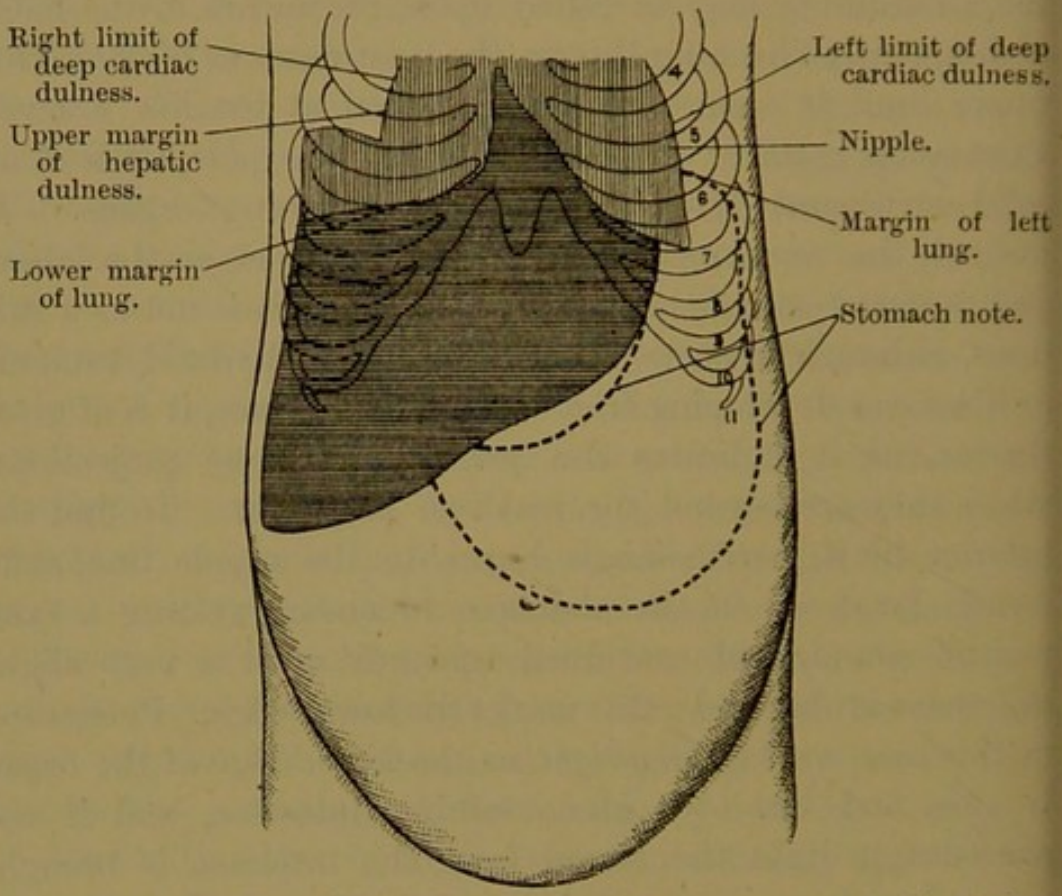


Fig. 82.—From the post-mortem table, showing enlargement of the liver and distention of the stomach.

While the above is all that is usually necessary in clinical work, it is as well to state that in many cases it is quite practicable to trace the outline of the entire organ. As the lower edge of the heart lies on the liver, with the diaphragm only interposing, this is by no means an easy undertaking; but, when the ear and hand have become trained, it can be noted that there is a difference of tone or of pitch in the dulness over the two organs which enables them to be separated.

The diagram at page 208, taken from a photograph of a patient after the heart and liver had been percussed, gives the relations of these organs, and shows that accuracy is quite attainable.

Enlargement of the liver occurs from congestion, waxy degeneration, fatty infiltration, malignant disease, hydatids, abscesses, and various other rarer conditions. The accompanying diagram (Fig. 82) shows the results of percussion in a case of enlarged liver from malignant disease, the stomach also being dilated. The percussion here was done on the post-mortem table, and the lines were found to be absolutely correct.

Diminution in size occurs in cirrhosis, acute yellow atrophy, in prolonged venous congestion, and in diseases accompanied with starvation, as in obstructions of the œsophagus.

The Gall Bladder.—The gall bladder projects from the under surface of the liver opposite the ninth right costal cartilage, as shown in Frontispiece I. Its position varies somewhat: we have seen it in the parasternal and in the mammary line, or at any point between these. This, apart from individual modifications, is due to the fact that the liver is capable of lateral movement, and can be pushed to the right as far as its suspensory ligament will allow, probably by a distended stomach, as Symington has pointed out, and as we have frequently verified.

Inspection.—A distended gall bladder may form a distinct projection on the abdominal wall, but only when the distention is great.

Palpation.—The normal gall bladder cannot, as a rule, be felt. An enlarged one is recognised by its more or less pyriform or rounded shape, by its relation to the edge of the liver, by the fact that it moves with respiration, and by its elasticity. Its position is usually as indicated above, but we have seen it in a line with the sternum.

Causes of Distention.—Distention is due to obstruction

to the outflow of the bile. The obstruction may be caused by catarrh of the duodenum, gall stones, the *Ascaris lumbricoides*, one of the *Tæniæ*, or malignant disease of the head of the pancreas.

The Great Omentum is sometimes infiltrated with malignant growth, forming a solid cake-like structure lying on the intestines, when it is readily mistaken for an enlarged liver. The presence of a tympanitic note above it would exclude the idea of its being liver. We have also seen it drawn up, thickened, and adherent to the abdominal wall, and feeling exactly like the lower edge of the liver and mistaken for it. Further, it may be quite drawn up and form a hard thick ridge attached to the transverse colon: this occurs in some cases of diffuse malignant disease of the abdomen.

The Pancreas.—The position of the pancreas, as seen from before and behind, is represented in the Frontispieces.

It crosses the vertebral column opposite the twelfth dorsal and first lumbar spines, and corresponds anteriorly with a point about three inches above the umbilicus. It is to be remembered that it is an elongated structure from one to about two inches broad, the head of which lies on the vertebral column, and a little to its right, and in the curve made by the duodenum, while its tail touches the spleen. The second part of the duodenum is thus to its right, while the third part is below it. The cœliac axis is given off above it. In close apposition to it posteriorly are the inferior vena cava, the vena portæ, and the common bile duct.

Lesions.—The most important lesion to which the organ, so far as is known, is liable is malignant disease, which affects its head chiefly. When thus affected the head forms a hard, more or less rounded swelling, which can sometimes be felt in the position already indicated. Its deep situation serves to distinguish it from a distended gall bladder. When affected, the glands in its neighbourhood may be involved in the morbid process.

Results.—Owing to the relations of the common bile duct to it, there may be obstruction to the outflow of bile, and consequent jaundice. The pancreatic duct is also occluded, and there is at times an increase of fat in the stools as a result. An increased quantity of fatty matter in the fæces is, however, much more commonly the result of hepatic disorders, or of some affection of the intestinal mucous membrane. Pressure on the vena portæ leads to ascites, but this is not constantly present, and when present it is not usually extreme.

The Spleen.—The spleen is situated behind the ninth, tenth, and eleventh ribs on the left side, with its long axis parallel to the eleventh rib. Posteriorly it is in close proximity to the spinal column; anteriorly it reaches a line parallel with but a little posterior to the mid-axillary line. It measures about 5 in. in length, and 3 to 4 in. in breadth, but its size varies within considerable limits. It lies in contact with the diaphragm as the latter arches upwards. This separates it from immediate contact with the chest wall, while posteriorly the inferior edge of the lung also lies between the organ and the surface. It is in contact with the greater curvature of the stomach; and inferiorly it touches the splenic flexure of the colon. Its relations to the kidney and pancreas need not be specified, as they have no bearing on diagnosis. The organ usually presents a notch on its anterior border towards its lower end, the presence of which is of diagnostic value when the organ is enlarged and projects beyond the ribs.

Inspection.—The normal spleen gives no evidence of its presence by inspection, but when much enlarged it causes a local bulging, and may distend the greater part of the abdomen.

Palpation.—The normal spleen cannot be felt. When it is enlarged, however, it may project beyond the free edge of the ribs, and can then be felt in the left hypochondrium. It can sometimes be brought within touch of the fingers by

pressing firmly with the left hand in a direction inwards and downwards over the ribs covering it. It may be so enlarged as to extend into the right side of the abdomen and downwards into the pelvis, while at the same time it may push the diaphragm upwards to such a degree that the percussion dulness reaches into the axillary region. The enlarged organ is firm, hard, and smooth to touch, and presents a well-defined notch on its anterior margin. It moves slightly during forced respiration, being pushed downwards during inspiration by the descending diaphragm, and retreating during expiration.

Percussion.—In percussing the spleen and delineating its borders, percussion should pass from the resonant organs around to the dull area of the solid organ. Percussion may be performed with the patient lying inclined to the right side, or in the sitting or standing posture. Percussion should be begun sufficiently high up in the posterior half of the axillary or infra-axillary region to elicit the pulmonary percussion sound, and continued downwards until the dull sound obtained from the organ is received. A mark should be made at this point. Percussion is continued in the same vertical line until a tympanitic note informs us that we have passed from the solid spleen to the intestine. This inferior limit does not quite reach the margin of the ribs if the organ be not enlarged; this fact enables us, when there is difficulty in fixing the limit of dulness inferiorly, to check our observations by means of palpation. Difficulty is experienced when the dull note of the spleen merges into and is continuous with the dull note of the colon distended with solid matter. To define the anterior limit percuss from before backwards in a line at the level of and parallel to the direction of the tenth rib. Posteriorly the organ cannot be satisfactorily percussed beyond the scapular line, as behind this line it is separated from the chest wall by lung and diaphragm. Here also it is well to begin with the lung sound, and to percuss outwards and somewhat downwards.

In some acute diseases it is undesirable to move the patient on to his side. Under these circumstances we must be satisfied with the vertical dulness in the infra-axillary region, and the anterior limit of the dulness which can be obtained with the patient lying on his back. This gives a sufficient index to the size of the organ, and is ample for all practical purposes.

Percussion requires to be light, for if done vigorously the tympanitic sound from the adjacent stomach or colon is brought out, and is apt to mislead the observer.

The spleen is enlarged in all febrile diseases, especially ague, in pernicious anæmia, lymphadenoma, leucocythæmia, general venous congestion, portal congestion, waxy disease, and rickets.

No observations have been made as to affections in which diminution in size might be regarded as of clinical importance.

The Kidneys.—The kidneys being situated behind the abdominal cavity are not as a rule palpable. They are lodged one in each side mainly in the epigastric and hypochondriac regions, but the right usually extends into the umbilical and lumbar regions.

The lower border of the right kidney reaches a point an inch above the level of the umbilicus, that of the left kidney 1½ in.; the upper border reaches the level of the sixth or seventh costal cartilage, the right being half an inch higher than the left. The upper part of the kidney lies nearer the middle line than the lower. Posteriorly the upper end of the right kidney reaches the level of the eleventh dorsal spine, the lower end 1 in. above the iliac crest. The left kidney lies half an inch higher than the right. Like all abdominal organs, the kidneys are liable to variations in size and shape. In the female they are situated slightly lower than in the male. In the child they are relatively of large size, and are placed more symmetrically than in the adult.

Palpation.—The kidneys are palpated by pressing one

hand firmly into the loin covering them, and the other deeply from the front, so as to try to feel the organ between the two hands. The normal kidney, even in very thin persons, can only be indistinctly felt, but even when it cannot be satisfactorily felt, the production of pain by palpation is often of great diagnostic value in renal calculus, and in the early stages of tuberculous disease before the organ is much enlarged. When they are much enlarged they can be distinctly felt. If they are the seat of hydronephrosis, pyelonephrosis, or if their substance is destroyed by large abscesses, as in tuberculous disease, not only can their enlargement be felt, but distinct fluctuation can be obtained with the hands in the position as directed above. They are also readily felt when enlarged from malignant disease.

Percussion.—Percussion of the normal kidneys is unsatisfactory, and is not relied upon in clinical work. The lower limit of the organs can, however, be made out posteriorly owing to their relation to the colon, the commencement of its tympanitic sound marking the inferior extremity of the kidney. In the same way the convex surface of the organs may be made out. Percussion requires to be strong, and is perhaps best performed with a plessor and pleximeter. When the organs are enlarged percussion is more feasible; when the enlargement is great the dull percussion sound can then be readily elicited even in front, but even then greater reliance is placed on palpation.

Floating or Loose Kidney is a condition which is occasionally met with. It occurs most commonly on the right side. It results from the looseness of the renal connections permitting the organ to leave its normal position, and to come forwards until it can be felt lying under the edge of the liver; or it can be pushed out of its normal position by one hand in the loin, while it is felt through the anterior abdominal wall by the other. It can sometimes be pushed across the middle line. That the organ is the kidney is recognised during life by its mobility, position, shape, and

sometimes by the sensation of nausea caused by grasping it firmly. The flank may be less dull on percussion than the other one. The percussion sound over the kidney itself, when it is near the anterior abdominal wall, is of course dull. Attacks of peritonitis sometimes occur round it, and cause pain and tenderness on palpation.

The Suprarenal Capsules.—These organs are situated on the antero-superior surface of the kidneys. They may be the seat of malignant or tuberculous disease, and if their enlargement is considerable they may, in thin persons, be felt by careful palpation.

Abdominal Aneurysm. — The abdominal aneurysms which come under the care of the physician are those of the abdominal aorta or of the cœliac axis. The most commonly affected part of the aorta is just below the diaphragm, and in this situation it involves the cœliac axis. Aneurysm is recognised by the presence of abnormal pulsation; by the presence of a tumour, the pulsation of which is distensile; sometimes by the presence of a thrill; and by the presence of a systolic murmur over it. The time and the fulness of the pulse in the femoral arteries, as compared with the arteries in the upper limb, may be of assistance; in aneurysm the pulse in the former may be delayed slightly, and be less full. In addition, aneurysm may produce great pain by pressure on nerves and on the vertebral column, the bones of which may become eroded. When this last occurs there is tenderness on pressure over the spine, and there may even be curvature. If the aneurysm be large, it may exercise pressure on other important structures, such as the large veins in the abdomen.

Bladder. — In many diseases it is important to pay particular attention to the state of the bladder. More particularly is it necessary to bear in mind that retention of urine occurs in various injuries and diseases of the spine, in the delirious stage of the acute fevers, and in all more or less comatose states. The distention of the viscus is recog-

nised by dulness on percussion above the pubes; in extreme distention it may produce a very marked bulging of the hypogastrium, and may reach even as high as the umbilicus. Care must also be taken that the constant dribbling away of the urine does not lead one to think that the viscus is emptying itself, when only *the overflow* is passing away.

Contents of the Alimentary Canal

Under the above head fall to be considered the examination of matters voided by the stomach and rectum, and the investigation more especially of the contents of the former.

Vomiting — Causes. — Vomiting may be due to very diverse causes, the investigation and differentiation of which are often surrounded with difficulty and uncertainty. It may be due—(*a*) To local disorders of the stomach. (*b*) To affections of other parts of the digestive system, as in intestinal obstruction, biliary colic, and peritonitis. (*c*) To affections of the central nervous system, as in uræmic poisoning, meningitis, tumour, and hæmorrhage at its onset, while in rare cases it is periodic and epileptoid in type. (*d*) To organs not concerned in digestion, as the uterus, pregnancy being often associated with very troublesome vomiting, and the kidney, renal colic being commonly accompanied with vomiting; in these the action is a reflex one. (*e*) To the poisons of some of the acute fevers, and poisons introduced into the stomach, given as emetics, taken accidentally, or administered with criminal intention.

Time and Manner.—The time and manner of vomiting provide important indications as to its cause. It may occur almost immediately after taking food, and may or may not be accompanied or preceded by pain, when it indicates that some local irritative condition of the stomach exists. It may be delayed until one, two, or more hours after a meal, when it is usually preceded by a sense of distention, perhaps pain, acidity, flatulence, or heartburn; in such cases the

digestive process in the stomach has been imperfectly performed, or the digested material has not passed out of the stomach. In the first the local cause may be catarrh, ulceration, or malignant disease of the body of the viscus; in the latter it is due to obstruction at the pylorus, from malignant thickening or from the cicatricial contraction following upon the repair of a simple ulcer. Relaxation with dilatation from simple atony also leads to retention of the gastric contents. In all these vomiting is preceded by some premonitory symptom referable to the stomach, as pain, nausea, or discomfort; when due to other than stomachic causes, there is usually no symptom referable directly to it. In biliary and renal colic the vomiting accompanies the pain, and is a reflex of it. When it is neurosal in origin it is usually unaccompanied by nausea. In this form, and in that due to colic, it, as a rule, occurs independently of taking food; but this is by no means invariably the case, for it can often be noted that, even when purely neurosal, as in meningitis, the vomiting may occur immediately after taking food, or even water, into the stomach, although it may reveal its true nature by occurring also at times when food has not been taken.

Investigation of the contents of the Stomach.—Before drawing off the contents of the stomach for examination, it is usual to give a test meal. A suitable test breakfast consists of 2 to 3 oz. of bread and 10 to 12 oz. of weak tea. Such a meal contains albuminoids, sugars, starches, non-nitrogenous extractives and salts, the weak tea in addition acting as a stimulant to gastric secretion. The contents of the stomach are drawn off an hour after the breakfast is taken. A test dinner may consist of soup, scraped meat, and wheaten bread, the contents of the stomach being withdrawn after an interval of four to six hours. As a matter of practice, it is probable that more information is obtained by drawing off the contents of the stomach after such a meal as the patient is in the habit of taking in the ordinary

course, provided that the meal contains a fair amount of proteid matter.

To obtain the contents of the stomach for analysis, a soft red rubber œsophageal catheter, with a rounded end and two lateral eyes, is used. This is firmly attached by a glass joint to a length of tube and a glass funnel. A large size of tube is desirable, as it is better grasped by the muscles of the pharynx. If the patient be intelligent, and the object in view is appreciated, there is frequently but little difficulty in passing the tube; but if the patient be nervous, and the pharynx irritable, it is well to spray the parts with a 5 per cent. solution of cocaine five minutes before passing the tube. Caution the patient to breath through the nose while the tube is being passed. The head being thrown slightly back, the tube, lubricated with water, is introduced over the tongue into the pharynx, and very gently and slowly passed down into the gullet. When the tube enters the stomach, there is usually an escape of gas. The funnel at the end of the tube is then depressed, and as a rule some of the stomach contents will escape. If not, the patient is directed to retch, and if this is not successful gentle pressure may be exerted over the epigastrium. It is seldom that it is necessary to introduce water into the tube to exert a syphon action. Before withdrawing the tube pour in a little warm water to clear it of stomach contents, and to prevent injury to the mucous membrane of the stomach by suction into the eyelets of the tube. While the tube is *in situ* it is sometimes desirable to estimate the capacity of the stomach by filling it up with warm water, and noting the amount that can be introduced without causing pain. When it is desired accurately to map out the size and position of the stomach, this can readily be done by distending it with air through the stomach tube. Remove the proximal tube and funnel at the glass joint, and substitute the ball syringe of a spray producer. The stomach can then be distended at will. The method is simple and easy, the amount of air introduced is

under command. There are none of the obvious objections that can be urged against the old method of administering bicarbonate of soda and tartaric acid.

The **character** of the stomach contents and vomited matter varies much with the time the food has remained in the stomach, the nature of the food, and the changes which have supervened. The unfiltered contents should be stood for a short time in a conical glass before noting the appearance. On standing, the material usually separates into layers, the solids below, the liquid—an almost clear fluid—above. In dilatation of the stomach, at times three layers are formed—the solids below, covered by a layer of almost clear fluid, and on the top a thick scum of a greyish or brownish colour, consisting of sarcinæ and yeasts.

As a rule the contents of the stomach are of a pale yellow colour, but this varies with the nature of the food. Bile may give a distinct green or yellow colour, while blood may vary from dark brown to bright red. In intestinal obstruction the vomited matter may have a fæcal appearance, from the presence of the contents of the small intestine.

The amount of the vomited matter or the food withdrawn varies in health with the amount of the previous meal and the time which has elapsed since ingestion. From one to two pints is not an excessive amount to withdraw from the stomach from five to seven hours after a meal. In gastric dilatation, where there is undue retention of the food in the stomach, enormous quantities may be withdrawn or vomited.

The **odour** of the healthy contents of the stomach is sour. In disease, when fermentation is present, this is disagreeably accentuated. When butyric acid is present the odour is that of rancid fat, and is very characteristic. Acetic acid gives a vinegar smell. Ingested poisons, such as carbolic acid, may give a distinct odour to the vomit.

The **specific gravity** of the filtered stomach contents obtained after a meal is from 1010 to 1020. In hyperacidity it may rise above 1020.

Mucus is present normally in small quantities, but is much increased in catarrhal affections of the stomach, in hyperchlorhydria and in ulcer. When in excess it is easily recognised, from the stringy, tenacious character of the gastric contents.

Blood may appear in the vomited matter, bright and clotted, or in various stages of digestion. The condition is

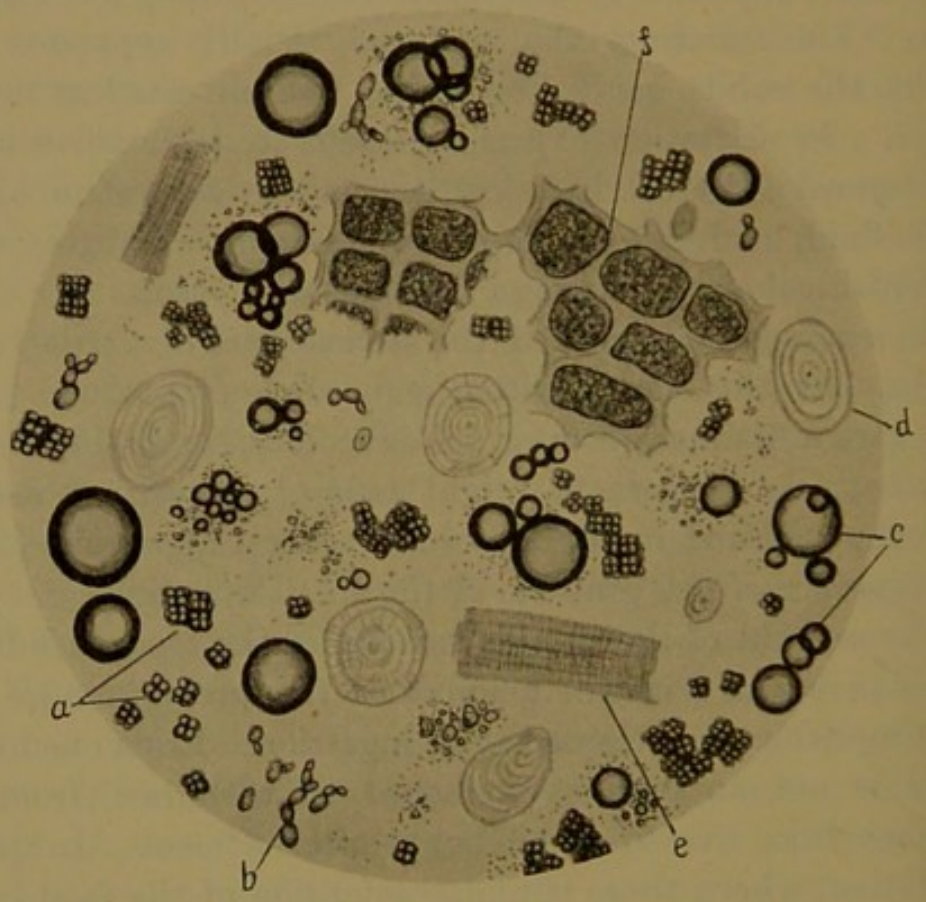


FIG. 83.—The microscopic appearance of the gastric contents from a case of gastrectasis. *a*, Sarcinæ; *b*, yeast; *c*, oil globules; *d*, starch granules; *e*, muscle fibres; *f*, vegetable cells.

known as hæmatemesis. When the bleeding is from a large vessel and profuse it is bright red. When it is retained in the stomach a short time it is dark and clotted, while if it be retained for longer it becomes dark brown or black in appearance, like "coffee grounds," not in clots.

When in small quantity in the stomach contents, the microscope is the best means of definitely determining its

presence. The guaiacum and ozonic-ether test is of little or no value, as the reaction is given by the saliva, bile which may be present, and many of the common vegetable constituents of the food. Undigested food may be present in the stomach contents, such as muscle fibre, coagula of milk, fruit skins, etc. Remarkable cases are on record where, in gastric dilatation, cherry stones, fish bones, and such indigestible matters have been retained in the stomach for months.

Pus is rarely present in such quantity as to be recognisable by the naked eye, but it may occur in phlegmonous gastritis or where an abscess has ruptured into the stomach.

Microscopic examination of the stomach contents may reveal the presence of columnar and squamous epithelium, white blood corpuscles, and red blood corpuscles affected by the gastric juice. From the food, muscle fibres with thin transverse striæ, fat globules, and starch granules. Fungi of various kinds—moulds, yeasts, and fission fungi may all be present. The most important fission fungi are *Sarcinæ ventriculi*, which have the appearance of corded bales of wool. They stain brown with the iodo-potassic-iodide solution, and readily take up aniline dyes.

The bacterial forms found in the stomach contents are very numerous. They will be noticed under Clinical Bacteriology.

The Chemical Examination of the Stomach Contents is of the greatest importance in practical medicine. The first point is to determine the **total acidity.** This comprises the acidity due to acid salts, to mineral acids, and to organic acids, in a free state or combined with proteids.

The estimation is carried out by titrating a known quantity of the stomach contents with a decinormal solution of caustic soda. A litre of decinormal soda solution contains 4 grms. of caustic soda, and will be neutralised by 3·65 grms. hydrochloric acid. Therefore 1 c.c. of decinormal soda

solution will contain 0·004 grms. NaOH, and will neutralise 0·00365 grms. HCl.

In titrating it is necessary to have an indicator, a body which will change colour with the altered reaction of the fluid. The best for the purpose is an alcoholic solution of phenol-phthalin, which in acid solutions is almost colourless, in alkaline of a pink colour. The titration is carried out as follows :—

Take 10 c.c. of the unfiltered stomach contents in a flask, and dilute with about 100 c.c. of water. Shake well to break up as far as possible all solid particles. To this add a few drops of a 2 per cent. alcoholic solution of phenol-phthalin. Take a Mohr's burette, graduated in fifths of a c.c., and fill up to the desired point with the decinormal soda solution. Turn the stop-cock of the burette, and allow a drop or two to escape in order to fill the nose of the burette. Place the burette in a holder. The flask with the stomach contents is now placed under the burette upon a sheet of white paper. The point at which the soda solution is standing in the burette is noted on paper, and the titration is begun. A pink colour at once appears on dropping in the soda solution, but disappears on gently shaking. Titration is continued till a uniform delicate pink tinge is obtained throughout the fluid, which shows that it has become very faintly alkaline. The level of the soda solution is now noted. Say that 4 c.c. of the decinormal solution have been used to neutralise the 10 c.c. of stomach contents, 40 c.c. would be required to neutralise 100 c.c. stomach contents. According to Ewald's notation, the acidity in the given case could be expressed as 40, a convenient clinical method of expression. We know that 1 c.c. decinormal soda solution contains 0·004 grm. NaOH, and corresponds to 0·00365 grm. HCl.

In the case under discussion—

$$4 \text{ c.c. } \frac{N}{10} \text{ NaOH} = 10 \text{ c.c. stomach contents.}$$

4 c.c. $\frac{N}{10}$ NaOH = 0·016 grm. NaOH.

4 c.c. $\frac{N}{10}$ NaOH = 0·0146 grm. HCl.

∴ 10 c.c. stomach contents = 0·0146 grm. HCl.

100 c.c. stomach contents = 0·146 grm. HCl.

Total acidity as expressed in HCl = 0·146 per cent.

Normally the total acidity of the stomach contents, one hour after a test meal, ranges between 40 to 65; any figure above or below this may be considered pathological.

Qualitative tests for free acids are not entirely satisfactory. Several are recommended. **Congo red** is best used in the form of congo red papers, strips of filter paper dipped in a saturated watery solution of the dye, and dried. In the presence of free acid the paper becomes of a blue colour. Acid salts do not affect it, but organic acids do, so that it cannot be considered an accurate reagent for indicating the presence of free hydrochloric acid. **Benzo-purpurin** test papers are of a purple colour. In the presence of free hydrochloric acid they become dark blue, and the colour is not affected by ether. With free organic acids a brownish-black colour is got, which is removable by ether. If a mixture of free organic and inorganic acids is present, the colour is a dark brown tint, and is only partially removable by ether.

Of the tests for **free hydrochloric acid** alone, Gunzburg's vanillin-phloroglucin method is the most applicable to general work, and is very delicate.

The test solution consists of—

℞	Phloroglucin	30 grs.
	Vanillin	15 grs.
	Alcohol	1 oz.

A few drops of this solution and an equal quantity of the stomach contents are mixed on a porcelain basin and evaporated to dryness over a water bath, or by cautiously

heating *over* the flame of a spirit lamp, care being taken not to burn and brown the mixture. If free HCl is present, a bright rose-pink colour develops. The test is accurate and very delicate, the colour forming when a single drop of stomach contents is used, if it contains as much as 0·01 per cent. free HCl.

The **resorcin** method is not quite so delicate. The test solution consists of 5 grms. resorcin, 3 grms. of cane sugar, dissolved in 100 c.c. weak alcohol. Used in the same manner as the phloroglucin-vanillin solution, the colour struck is not so brilliant, but more of a pink tinge.

The presence of the organic acids, when in a free state, may be detected by *Uffelmann's* test. Take a solution of carbolic acid, and to it add a drop of perchloride of iron solution. A deep blue-black colour develops. The solution is then diluted with *distilled* water, till the colour is of a depth similar to that of claret. In the presence of lactic acid, the blue colour is discharged and a canary-yellow developed. Lactic acid, however, is seldom present alone in the stomach. Hydrochloric acid, when free, discharges the colour of the reagent. When combined with a proteid, it produces a yellow-brown colour. Acetic acid also produces a brown tint. Butyric acid discharges the colour from the solution, and generally gives it a greyish opalescent tint. Citric and tartaric acid give the same reaction as lactic acid.

A more delicate and accurate test is obtained by adding a drop or two of a dilute solution of perchloride of iron to 50 c.c. distilled water. If lactic acid be present, a few drops of the stomach contents dropped into this solution will turn it, from almost colourless, to a light yellow colour. Hydrochloric, butyric and acetic acids will not produce this colour. Acetic acid can be recognised by the "vinegar" smell. Butyric acid has a very characteristic and disagreeable smell. If a drop or two of strong acetic acid and some alcohol be added to the stomach contents in a test tube, and boiled in

the presence of butyric acid, butyric ether forms, giving an agreeable odour of "pine-apple rum."

The methods for the quantitative estimation of free hydrochloric acid, combined hydrochloric acid, and the volatile and non-volatile organic acids, are too elaborate in the procedure and in the apparatus required to be discussed within the scope of the present volume. For their consideration the student is referred to special works.

In considering the significance of the results obtained from the chemical examination of the stomach contents, the student must remember that one of the earliest changes in digestion is a combination of the hydrochloric acid with the proteid bodies of the food. The presence of free hydrochloric acid, therefore, indicates a power on the part of the gastric glands to undertake further work. In normal digestion free hydrochloric acid is always present in the stomach contents an hour after a test breakfast. Free hydrochloric acid is diminished in acute and chronic gastritis, in gastric atony and dilatation, and in general conditions, such as fevers, pernicious anæmia, diabetes, Addison's disease, and chronic wasting diseases. Free hydrochloric acid is absent or present only in very small quantities in gastric carcinoma, but it may also be absent in atonic dyspepsia. The entire absence of free hydrochloric acid cannot therefore be considered as diagnostic of carcinoma. The total acidity may be increased either from excess of hydrochloric acid, hyperchlorhydria, or from an increase of organic acids. Hyperchlorhydria is most commonly met with in gastric neurosis, but it is also a pronounced feature in ulcer of the stomach.

Lactic acid may occur as sarcolactic acid, a product of normal digestion after a meal rich in meat. In disease it occurs as the result of fermentation, hence its presence points to a lesion with which fermentation is associated. Its presence was, at one time, thought to be pathognomonic of gastric carcinoma, but this is not the case. It is true, however, that the absence of free HCl, with the presence of

lactic acid and a loss of motor power of the stomach, are a combination of signs seldom met with apart from gastric carcinoma. The absence of lactic acid does not, however, exclude the presence of gastric carcinoma.

The activity of gastric digestion is shown by the presence of albumoses and peptone in the stomach contents. These are seldom absent from the stomach contents after a test meal. Their presence can be demonstrated by the biuret and other reactions, which will be found in the section on the Proteids in the Urine. The activity of gastric digestion may best be tested in the following manner. Take small discs of coagulated egg albumin, cut with a double-bladed knife to a uniform size and thickness. A stock of such discs may be preserved in glycerin till required. Four portions of the filtered stomach contents (about 5 c.c.) are placed in four small beakers, and an albumin disc placed in each. To the first nothing is added; to the second two drops dilute hydrochloric acid (B.P.); to the third 3 grs. pure pepsin; to the fourth both acid and pepsin. Place the beakers in an incubator, or in any warm place. The rate at which digestion proceeds in the different beakers will give an index of the digestive power of the specimen of gastric contents, and if defective, whether one or both the active agents is deficient. It must, however, be remembered that in the contents of a normal stomach all the HCl may be in a combined state, and cannot then exert any digestive power till free HCl is added.

Sugar and Starch.—The starch of the food is converted into maltose by the action of the saliva. Sugar can be detected by the reduction of Fehling's solution and other tests, the proteids first having been removed by heat and filtration. Starch may be recognised by adding a drop of the stomach contents to a mixture of iodine, iodide of potash and water, when a blue colour indicates the presence of starch, a violet that of erythrodextrin.

The Milk-curdling Ferment.—Rennet is present in the

stomach contents in health, absent in some diseased conditions, such as carcinoma. The simplest test for its presence is to add 2 to 5 drops of the filtered stomach contents to 10 c.c. raw milk. It is not necessary to neutralise the stomach contents, as such a relatively small quantity is used. If rennet be present, the milk coagulates on standing.

The Alvine Discharges.—It is always necessary to make inquiries regarding these, and not infrequently it is necessary to examine them.

The number of stools in health varies with the individual and the character of the food. A healthy male on an ordinary diet has one evacuation daily; in many women, who are apparently in good health, movement of the bowels may occur only every second or third day. When this infrequent movement of the bowels becomes pathological it is termed **obstipation.** The nature of the food and its amount, the quantity of fluid taken, and exercise all influence the number of the stools. When the bowels move frequently and the stools are loose, the condition is known as **diarrhœa.** Diarrhœa occurs in such conditions as intestinal catarrh, tuberculous and waxy disease of the bowel, dysentery and typhoid fever. Diarrhœa is frequently accompanied by straining, spoken of as **tenesmus,** a very prominent symptom in dysenteric diarrhœa.

The **amount** of the fæces varies with the quantity and nature of the food. In any disease which diminishes the digestion and absorption of the food, such as cholera, the amount of the fæces will be increased.

The **consistency and form** of the stools varies in health, but they are usually cylindrical and firm, what is termed a "formed motion." When very soft they are without form; they may be watery. When the fæces remain long in the alimentary canal the water is extracted, and they become hard and may form rounded or flattened masses known as **scybala.**

A pressure of a tumour upon the lower bowel, narrowing

its lumen, will produce an alteration in shape, the motion becoming flattened and ribbon-like or grooved.

The **odour** of the normal stool is due to the presence of indol, skatol, and fatty acids, and other products of bacterial action. The odour is exaggerated and disagreeable when there is obstruction to the entrance of the bile into the alimentary canal. It becomes very offensive when altered blood is present, and in some affections of the intestine such as typhoid fever. In infants with gastro-intestinal catarrh the odour is sour and unpleasant.

The **colour** varies much in health with the diet of the individual. A meat diet gives a darker stool, vegetable diet a paler stool, while pure milk diet produces almost colourless stools. Medicines affect the colour; thus, bismuth and iron produce a black colour, calomel a green from the discharge of biliverdin, santonin, rhubarb and senna a yellow colour, and hæmatoxylin a red.

The presence of **blood** in the stools, the condition known as melæna, may give a distinctive appearance. When the hæmorrhage is from the lower bowel the blood may be red, when from higher up the blood is altered, it becomes dark or even black, giving the stool a characteristic "tarry" appearance.

When the bile duct is obstructed and the bile prevented from entering the intestine, the stools become pale and clay-like, and are usually very offensive.

Diarrhœic stools are usually paler than normal; in cholera they are quite colourless, watery, and slightly opalescent; in young children the normal stool is pale yellow; in intestinal catarrh it may become green. In typhoid the stools have a semi-fluid consistency and a yellow colour, the well-known "pea-soupy" stool. The presence of abnormal constituents may be noticeable on inspection. Undigested food may be present in intestinal affections, *e.g.* white curds of milk. Gall-stones may appear in cases of biliary colic. To search for gall-stones, the motion should be put in a jar

and shaken up with water, to which a dash of formalin has been added to obviate unpleasant odours. The motion is then strained through a sieve. If stones are found they will be facetted if multiple, smooth and rounded if single.

Mucus in any quantity in the stool indicates the existence of catarrh of the bowel. The mucus may be intimately mixed, as in dysentery, when the frequent small stool consists of little but mucus and blood. When the mucus is in shreds and covers scybala the catarrh is usually in the large intestine. In cholera the particles of mucus look like boiled rice, giving the stool the characteristic "rice watery" appearance.

Pus in the stool indicates extensive ulceration of the bowel or rupture of an abscess into the bowel.

Microscopical examination will under different conditions reveal the presence of—(1) Fragments of undigested food, vegetable cells, starch granules, muscle fibre, and connective tissue; (2) epithelial cells, squamous and columnar; (3) blood corpuscles; (4) crystals, such as phosphates, oxalate of lime, fatty acids, cholesterin, hæmatoidin, and Charcot-Leyden crystals; (5) bacteria of different varieties; (6) the presence of intestinal parasites.

Intestinal Parasites

The parasites which are found in the alimentary canal, and whose presence is recognisable in the fæces, belong to the Protozoa and worms.

Protozoa.—Several Protozoa occur in the stools, but the most important clinically is the **Amœba coli.** Its presence in large numbers is frequently associated with a form of tropical dysentery and liver abscess. It is easily recognised under the microscope, resembling in appearance the fresh-water amœba.

Under favourable circumstances it is actively amœboid. Structurally it consists of a clear outer layer—the ectosarc,

of which the pseudopodia are mainly constituted, and a granular greyish central portion, the endosarc, which contains a nucleus, nucleolus, one or two granules and included foreign matters, such as bacteria, blood corpuscles and débris of various sorts. Its diameter is from 15 μ to 20 μ, or equal to that of three to five blood corpuscles. The protozoa occur in the healthy stools of man and certain animals. They are present in large numbers in the stools in certain forms of dysentery. To demonstrate the amœba a thin film should be

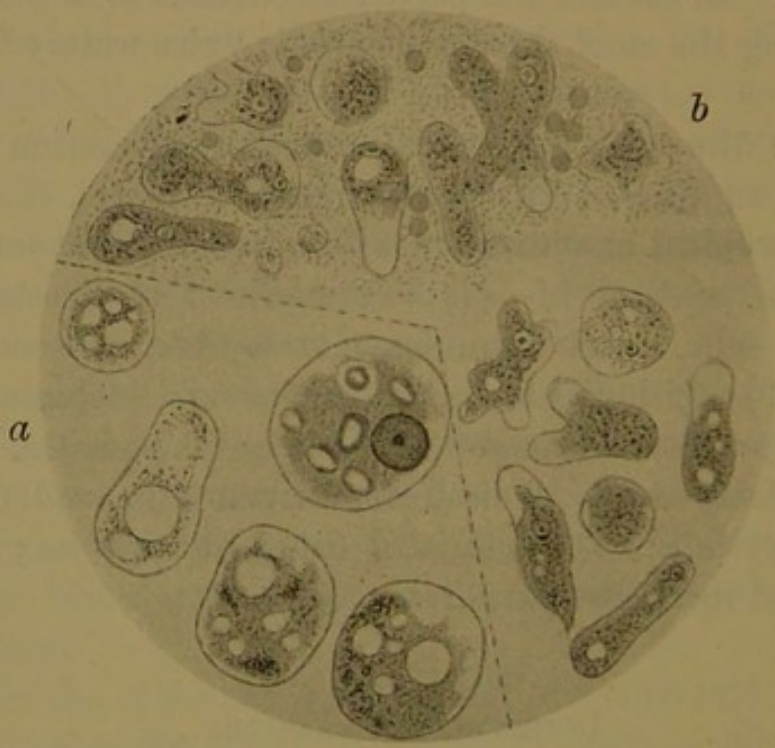


FIG. 84.—*Amœba coli.* (*a*) *A. dysenteriæ*, fixed and stained.—Councilman. (*b*) *A. dysenteriæ*, in stools.—After Lösch.

made by pressing, between a cover-glass and a slide, a portion of the mucus from the suspected stool. If the film be examined on a warm stage the parasite can easily be recognised by its form and characteristic amœboid movements. If a warm stage is not available it can easily be extemporised by making use of a strip of tin or copper plate, towards the end of which a hole has been cut. This is placed on the stage of the microscope with the preparation over the aperture, and heat is applied to the long arm by

means of a spirit lamp. Below a temperature of 75° F. the parasite has a sharply outlined spherical form; when warmed it becomes actively amœboid, throwing out pseudopodia.

Of **Infusoria**, the *Lamblia intestinalis* and the *Balantidium coli* have been found in the fæces, but they are not of sufficient clinical importance to merit description.

Worms occur in the intestine, as the *Cestoda* or tape-worms, the *Trematoda* or flukes, and the *Nematoda* or round worms.

Cestoda, or tape-worms, are long ribbon-like parasites. Each possesses a minute bulbous head—the scolex, and a long, flat, jointed body consisting of segments, the proglottides, which are developed serially from the posterior part of the head. The head is provided with suckers and hooklets by which the parasite is attached to its host. The young proglottides, small and immature, are next the head, the segments increasing in size and development the farther they are from the head, the most mature being at the tail end of the worm. The terminal joints, containing the mature ova, break away and are expelled in the fæces. The segments disintegrate and the ova are set free, and may be acquired by the intermediate host. Three forms of tape-worm are of sufficient clinical importance to require detailed description.

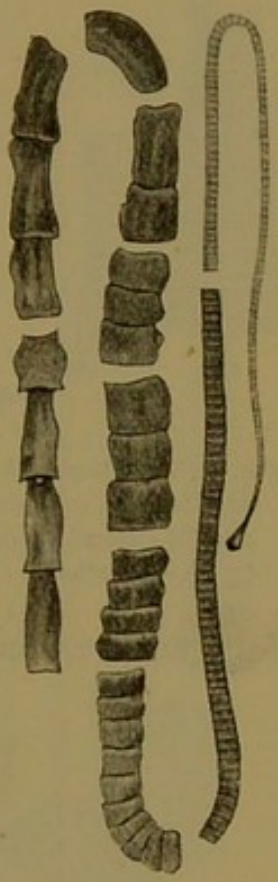


FIG. 85. — Tape-worm form of *Tænia saginata s. mediocanellata*.—Leuckart.

Tænia mediocanellata (T. saginata) has its immature or cystic stage in the muscles of the ox. When mature it measures from 12 to 30 ft., and is made up of from 1200 to 1500 segments. The broadest segments are at the centre of the worm, and they gradually taper upwards to a very fine neck. The head is flattened on the top and provided with four suctorial discs. There is no ring of hooklets, but

there may be a few isolated hooklets on the top of the head. The head is about 2 mm. in diameter. The mature segments, of which there are 100 to 200 in the chain, show a uterus with a longitudinal mesial stem, and on either side twenty to thirty lateral twigs which branch repeatedly. The genital apparatus opens at a marginally situated papilla, which lies somewhat behind the middle of the length of the proglottis. The mature ova are discharged in the ripe proglottides. They measure about 38 μ by 30 μ, have a striated shell, and contain a minute six-hooked embryo.

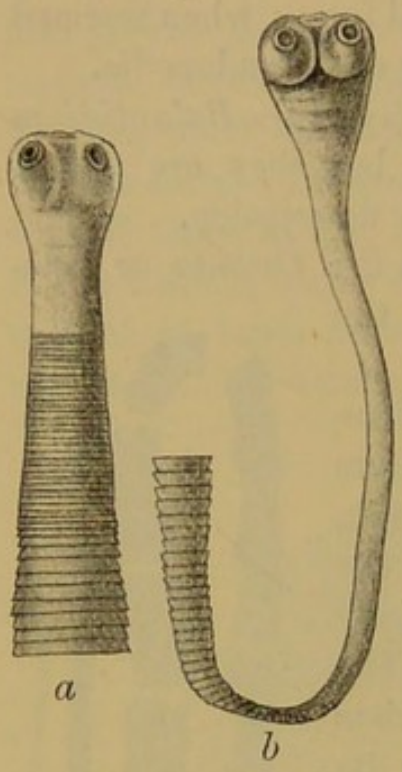


FIG. 86. — Head of *Tænia saginata* in (*a*) contracted and (*b*) extended condition. (× 8).—Leuckart.

T. solium, or the pork tape-worm, has its cystic phase in the pig. The adult shows a head furnished with four suckers and a double row of twenty to twenty-eight

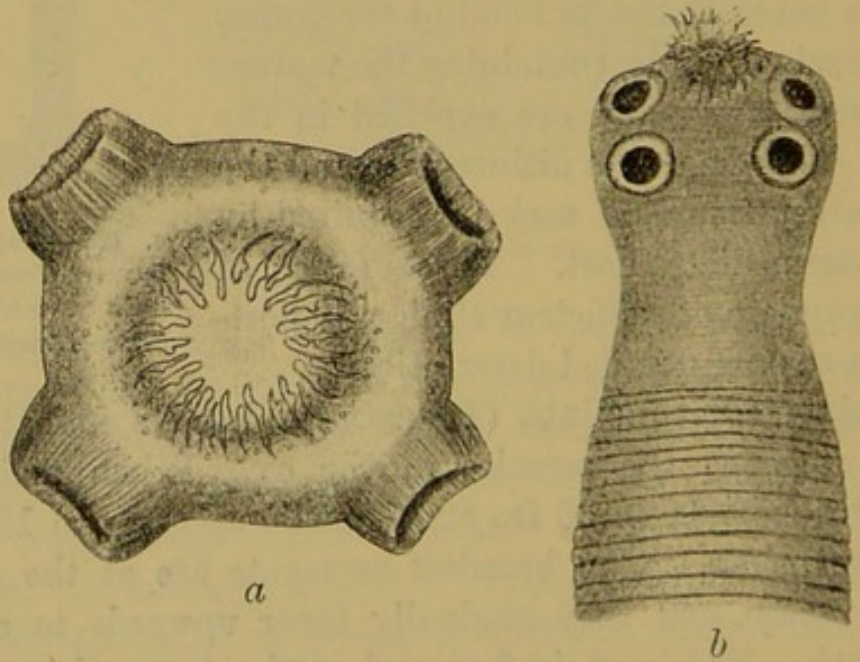


FIG. 87.—(*a*) Apex and hooks of *T. solium*; (*b*) head of *T. solium*. (× 35).—Leuckart.

hooklets, which surround the somewhat prominent rostellum or proboscis-like projection. To the naked eye the head is about the size of the head of a common pin. The scolex is succeeded by fine segments, which gradually increase in size on passing farther from the head. The largest segments, about the middle of the worm, measure about 8 mm. in breadth, the narrower but more elongated terminal joints being 8 to 12 mm. in length by 5 mm. in breadth.

The uterus has a longitudinal trunk with lateral branches, but the lateral branches divide dendritically, thus differing from *T. mediocanellata*. The ovum is almost round, measuring 30 μ to 35 μ, has a thick striated shell, and encloses a six-hooked embryo.

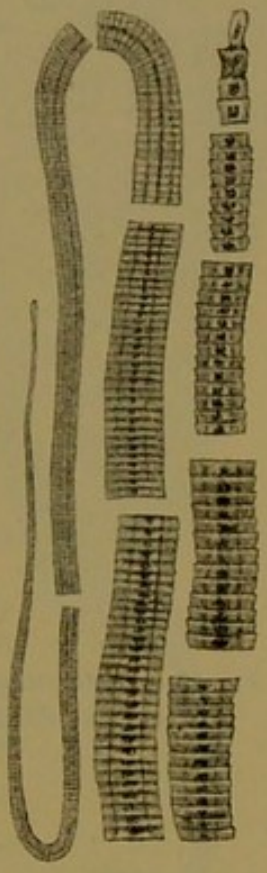


Fig. 88. — *Bothriocephalus latus.*— Leuckart.

Bothriocephalus latus, the fish tapeworm, may attain the enormous length of 30 ft. It is proportionally broad (10 to 12 mm.), but the proglottides are relatively short (4 to 5 mm.), and in a full-grown specimen number 3000 to 4000. The terminal segments are narrower and longer than those higher up the strobila. The head is flattened, club-shaped, and furnished with two well-marked lateral suctorial grooves. The uterus when distended with ova shows a series of radially arranged folds which give it a rosette-like appearance. In front of the uterus, on one surface of the segment, are the genital pores. Each ovum is enclosed in a brown shell, is oval in form, 0·07 mm. long, and 0·045 mm. broad. The shell is opercular, and the embryo, which has a thick ciliated cuticle and some hooklets, escapes by raising the lid of the shell when the ovum reaches water.

Trematoda, with the exception of the *Bilharzia*, are flat, fluke-shaped, hermaphrodite parasites. **Distomum hepaticum**

is a long, brown, flat, leaf-shaped parasite covered with minute spines. In the sheep the embryos find their way into the bile ducts and there develop. It is rare in man.

FIG. 89.—*Distomum hepaticum* (magnified).

D. lanceolatum is a smaller parasite than the preceding, and is very rare in the human subject.

Bilharzia hæmatobia has its habitat in the portal veins. It is a bisexual distomum of filiform shape. The ova are minute brown bodies, provided with a characteristic terminal spine and contain a ciliated embryo. They escape from the host by way of the fæces and urine. When the ova are mixed with water the embryo escapes by longitudinal rupture of the capsule and swims free.

The Nematoda, or thread-worms, are long, slender, bisexual organisms with a body tapering toward the extremities, at one of which is situated the mouth, at the other the anus.

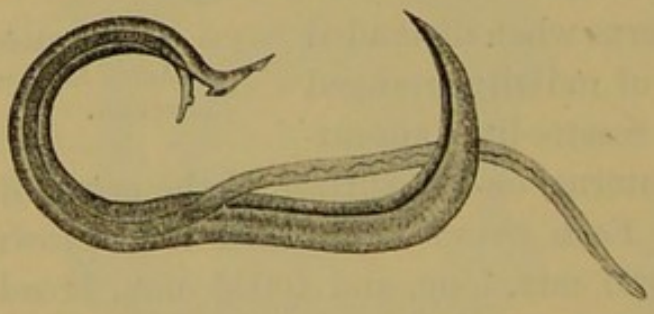


FIG. 90.—*D. hæmatobium*, male and female, the latter in the canalis gynæcophorus of the former.—Leuckart.

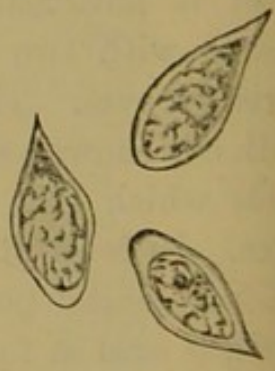


FIG. 91.—Ovum of *Bilharzia*.

Ascaris lumbricoides, the round worm, inhabits the upper part of the small intestine, occasionally wandering into the

stomach or lower part of the bowel. Both male and female are plump, cylindrical worms of a grey or pinkish colour, and transversely striated. The female is larger than the male. As a rule the worm occurs singly or in small numbers, and during its life numerous ova can be recognised in the fæces of the host. The eggs are oval, 0·05 to 0·06 mm. long, and have a thick chitinous capsule. When shed they are further enclosed in an albuminous capsule, which is either clear or stained brownish from the intestinal pigments of the host.

Fig. 92.—Free embryo of *Bilharzia.*

A. mystax is sometimes found in man, though normally parasitic in carnivora. It is a much smaller worm than the preceding, and has a cutaneous wing on each side of the head.

A. vermicularis, or thread-worm, occurs most frequently in children. The ova are swallowed, hatched in the stomach, and pass into the intestine, where they rapidly attain maturity. After sexual intercourse the male worm usually dies, the female moving on to the cæcum, where she remains till ovulation is complete. She then passes on to the rectum. At times the worms emerge from the anus, and wandering over the skin give rise to irritation; scratching ensues, the worm is ruptured, and the eggs may be transferred by the fingers to the nose or mouth, to be swallowed and in time developed. A cycle is thus kept up.

Fig. 93.—*Ascaris lumbricoides,* female.—Leuckart.

The female worm measures 9 mm. to 12 mm. in length, the male from 3 to 5 mm. To the naked eye the female

worms appear like small pieces of thread in the stool. The male is so small as to be easily overlooked. The eggs generally contain a differentiated embryo, the shell has a treble outline, and is slightly flattened on one side. On the convex side the middle layer of the shell is wanting.

Ankylostomum duodenale, the tunnel-worm, is an important parasite on account of its frequency in certain districts, and the grave

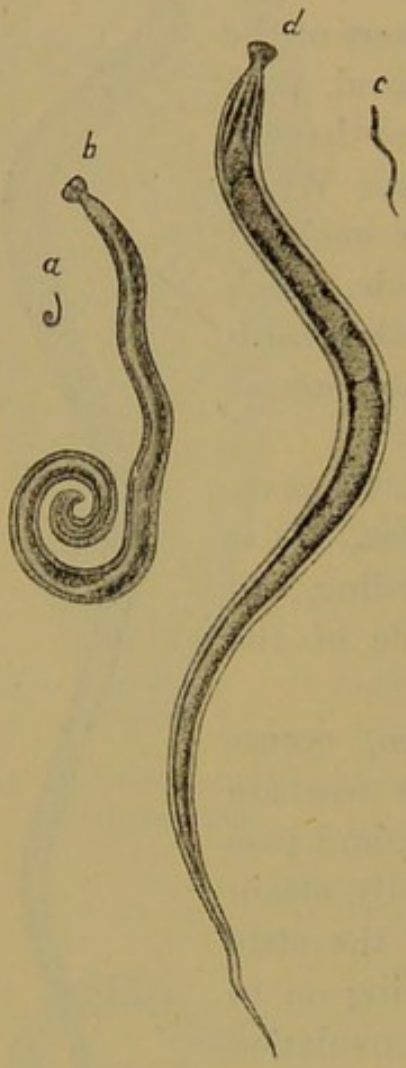


Fig. 94.—*Oxyuris vermicularis.* *a*, Male (nat. size); *b*, male (magnified); *c*, female (nat. size); *d*, female (magnified). —Leuckart.

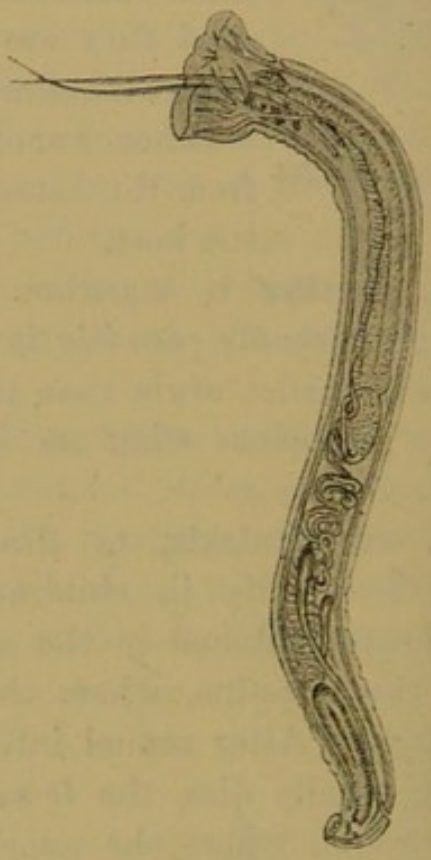


Fig. 95.—Male *Ankylostomum duodenale.*

cachexia to which it gives rise. It is common in many warm countries, and, though rarer in Europe, has given rise to characteristic and fatal epidemics of anæmia. The parasite inhabits the upper part of the jejunum, and to some extent the duodenum and ileum, attaching itself firmly to the

mucous membrane. It derives its nourishment from the blood of the host, which it freely ingests. The male worm measures from 6 to 11 mm., the female 7 to 15 mm. They are cylindrical, the head is bent backwards, the tapering neck ends in a cup-shaped expansion, which carries the oral aperture, which is furnished with four hooks and chitinous teeth. The caudal end of the female worm terminates in a short spine which can be protruded or retracted. The anus opens at the base of this, the vagina considerably anterior. The caudal end of the male is expanded into a trilobed, umbrella-like structure; at the bottom of this is the cloaca, from which two long delicate spicules emerge. The eggs are very numerous, oval, with a delicate, single outlined, transparent shell, enclosing two to eight greyish yolk spheres. The ova escape, develop in stagnant water or moist earth, are introduced into man by drinking water, and reach maturity in the intestine.

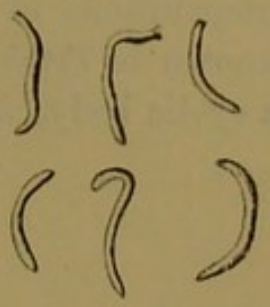


FIG. 96. — *Ankylostomum duodenale*, male and female. —Blanchard.

FIG. 97.—*A. duodenale.*—Sonsino.

Trichocephalus dispar, or the whip-worm, inhabits the cæcum. Both sexes are about 40 to 50 mm. in length. The anterior part is thin and thread-like in both sexes, and by it the parasite sometimes attaches itself by transfixing the mucous membrane. The posterior part of the body is fuller and thicker. In the male the hinder portion of the body is curved spirally. The eggs are oval, about 0·05 mm. long, have a thick shell which bears at each pole a rounded, translucent knob.

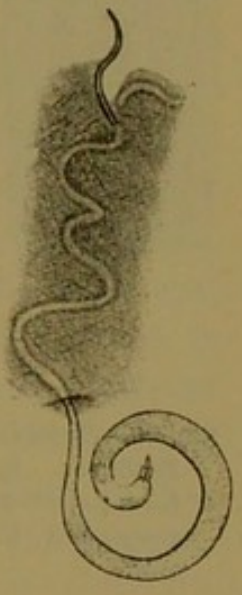


FIG. 98.—*Trichocephalus dispar, in situ.*—Leuckart.

Trichina spiralis is a nematode which is acquired by man

through eating raw or imperfectly cooked pork or other preparations of swine flesh, and gives rise to the disease known as *Trichiniasis* or *trichinosis*. The *Muscle trichina* is a cystic body, which contains a coiled-up cylindrical embryo possessing an alimentary canal but no sexual organs. Digestion sets free the embryo, which rapidly develops sexual organs The male intestinal trichina is about 1·5 mm., the female 3 mm. in length. The male is provided with two caudal appendages, by which the female is secured during coitus. This function completed, the male dies and the female continues to grow, and, in six to seven days after the ingestion of the muscle trichina, produces young, and continues to do so for six to seven weeks. The young are lancet-shaped organisms, rounded anteriorly and tapered toward the tail. These penetrate the intestinal walls and arrive at the muscles of the host, where they become encysted. Each female intestinal trichina is said to produce from 1200 to 1500 live embryos. Many of these are discharged with the fæces and die, but a large number pass to the muscles and become encysted. The gravity of the constitutional disturbance from the passage of the embryos to the muscles varies with their number, and reaches a height in from two to five weeks of the original ingestion of the muscle trichina.

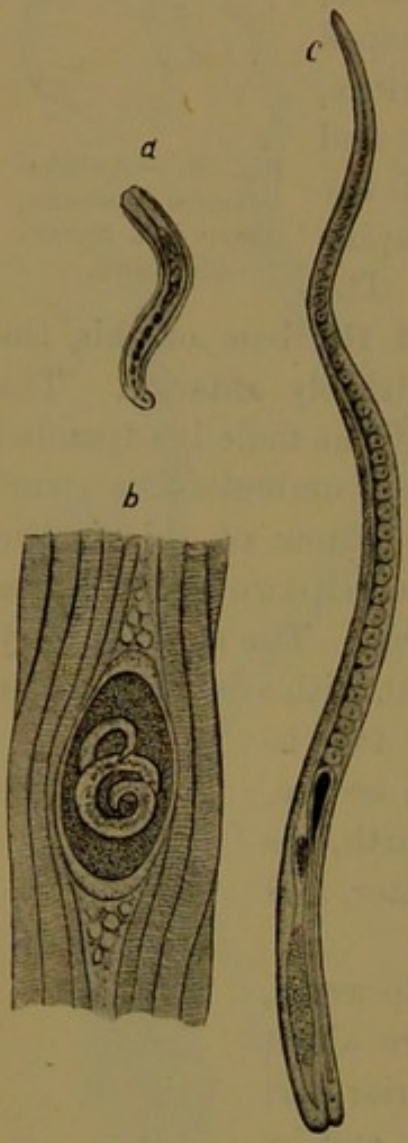


FIG. 99.—*Trichina spiralis*. *a*, Embryo; *b*, intermediate form; *c*, sexual form (unimpregnated female). — Leuckart.

CHAPTER IX

THE URINARY SYSTEM

Subjective Phenomena.—Pain may be complained of in different portions of the urinary tract. Pain at the end of the penis, especially towards the end of micturition, may be due to a calculus in the bladder. The pain is increased by movement. In prostatitis there is pain at the end of the penis and in the perineum towards the end of micturition. In women, when a urethral caruncle is present, there is severe pain at the urethral orifice during micturition.

Pain in the urethra, of a smarting and burning character, during and following micturition, is a prominent symptom of urethritis. Pain over the pubis and in the perineum before micturition, exacerbated by the act, and accompanied by tenesmus, occurs in cystitis. In renal calculus, when the calculus produces irritation of the pelvis of the kidney, the pain is intense in the loins, passing down into the groin and testicle or labia of the affected side. When the calculus is lodged in the substance of the kidney the pain is localised to the angle formed by the last rib with the erector spinæ. It can be increased by palpation or exercise. Pain in the loin of a dragging, sickening character occurs in enteroptosis or dislocation of the kidney.

Increased frequency of micturition may result from the increased secretion of urine, as in diabetes or chronic interstitial nephritis. This frequency, especially in nephritis, is prominent during the night. Frequency of micturition

may also be due to irritative lesions of the urinary tract, such as cystitis.

The disorders of micturition, due to disease of the nervous system, will be discussed under that section.

The Urine

The results obtained by examination of the urine serve a twofold purpose. In their wider significance they furnish evidence in regard to the processes of tissue change in the system at large, while in their narrower application they provide the most important, and frequently the only, guide to the condition of the urinary organs. A thorough acquaintance with the methods of ascertaining the physical and chemical characters of the urine is therefore of the highest value. In the following pages attention will be called to the various modes of procedure to be adopted in its clinical investigation.

Method of Examination.—In making a systematic examination of the urine some plan should be adopted, such as the following:—

Determine the daily quantity.

Observe the appearances as to colour, clearness, and deposits.

Ascertain if there is any peculiar smell.

Take the specific gravity.

Note the reaction.

Proceed to the chemical examination, especially with regard to the amount of urea, and the presence or absence of albumin and glucose.

Examine the deposit by means of the microscope.

Method of obtaining Samples.—In order to have a true estimate of the state of the urinary secretion, it is necessary to examine specimens taken from the total quantity passed during a period of twenty-four hours. The patient should be directed to empty the bladder at a certain hour, and to

reject the specimen then passed; after which the urine is to be collected until the same hour of the following day, when the last sample is to be added to the vessel employed for the purpose. The total quantity of the mixed urine is to be measured, and a portion taken for analysis. It is often desirable to have the urine passed during the night and the day, separately examined; and it may in certain cases be advisable to determine the characters of that secreted before and after food, for which purpose separate specimens must be collected. It may be necessary to ascertain whether deposits have their origin in the upper or lower urinary passages; and, with this end in view, the patient should be ordered to pass the first two ounces of urine into one vessel, and the rest into another. By this means it is easy to distinguish the products of the urethra from those of higher origin, as the portion first passed will contain almost all the substances which have accumulated in the urethra.

Physical Characters of Healthy Urine. — The quantity of urine passed by an adult in twenty-four hours varies with the amount of fluid taken and the amount of water excreted by other channels, and in each individual case these two factors must be considered. The average amount is about 1500 c.c., or about 50 oz. When freshly passed the urine is a clear fluid having a yellowish colour, a peculiar aromatic smell, an acid reaction, and a specific gravity depending on the amount of solids in solution, and therefore very variable, being frequently, in perfectly normal conditions, temporarily as low as 1005 and as high as 1030. The average specific gravity is about 1020. On standing in a glass vessel for eight or ten hours a faint cloud may gradually form in the lower portion of the urine. This is composed for the most part of *mucus*.

If the urine is alkaline it is somewhat cloudy when passed, from the deposition of *earthy phosphates*. These at once begin to fall down and form a hazy stratum, which gradually subsides to the bottom of the vessel. The true nature of

this appearance may be detected by the addition of a few drops of an acid which causes the cloud to disappear.

The urine may, soon after being passed, become muddy from the precipitation of *mixed urates*, which are not so easily soluble in cold as in warm water. The turbidity in this case passes gradually away, from the falling of the urates to the bottom of the vessel. Urates are at once recognised by the fact that on heating the turbidity disappears.

Chemical Characters of Healthy Urine.—The amount of solids contained in the urine of twenty-four hours depends largely upon the amount of food taken. On an average diet it varies between 60 and 70 grms., or 900 and 1050 grs. Rather more than one-half of this consists of **organic substances.** These are numerous, but those which alone possess any importance in clinical medicine are urea, uric acid, purin bases, oxalic acid, kreatinin, hippuric acid, the chromogen of urobilin, and organic sulphur-containing bodies, such as indican. Urea forms by far the largest proportion of the organic constituents, its amount, which depends on the nitrogen in the food taken usually in a normal diet, being from 28 to 40 grms., or from 420 to 600 grs., in twenty-four hours. The other organic substances taken together scarcely amount to 3 grms., or 45 grs., in the same period.

The **inorganic constituents** form, roughly speaking, rather less than half of the total solids. The most important of these are chlorides and phosphates of the alkalies and alkaline earths, sulphates, with small quantities of iron, free ammonia, and carbonic acid. Sodium chloride forms a large proportion of the inorganic constituents of the urine, the daily quantity passed depending on the salt taken in the food, and varying from 10 to 15 grms., or from 150 to 225 grs.; that is, about one-half of the inorganic, or about one-fourth of the total solids.

Variations in the Physical Characters of the Urine.—Alterations in the physical properties of the urine

depend on changes in its chemical composition, and serve to point out the directions in which such chemical modifications have taken place. It will be necessary to describe variations in the quantity, colour, smell, reaction, and density of the urine.

1. **Variations in the Quantity of the Urine.** — The amount of fluid taken and the amount excreted by other channels, is the great source of variations in the quantity. The entrance of fluid into the system is followed by an increase, and any great loss by the skin or bowels by a diminution in the amount of urine. Changes in the structure of the kidney give rise to alterations in the amount of its secretion. The urine may be greatly increased in quantity in waxy disease, and entirely suppressed in acute inflammation. From the conditions of its secretion, it is clear that any modification of the circulation must be followed by a change in the amount of the urine. Increase of the arterial blood pressure and increase in the rapidity of circulation through the kidney causes an increase; while diminished blood pressure and retarded flow through the kidney, as in cardiac diseases, lessens the amount. Nervous influences acting on the circulation cause variations in the secretion of urine. Amongst these may be mentioned hysterical polyuria and oliguria, the increase which is sometimes found as a symptom of cerebral lesions, and the conditions known as diabetes mellitus and diabetes insipidus. Suppression of urine may be caused by some obstruction in the urinary channels, as by calculi in the ureters, or it may be caused by acute inflammation of the kidney, or by lowering of the blood pressure, as in shock and collapse.

In health there is a distinct and important relation, between the day and night secretion of urine. The amount secreted during the daytime, is six times as great as the amount secreted during the night. In many diseased conditions, such as cardiac disease, wasting diseases, but especially in kidney disease, this relation is altered, and the amount of

night urine is increased till it may equal or even double the day urine. The symptom is one of the earliest complained of in chronic interstitial nephritis.

2. **Variations in the Colour of the Urine.**—Changes in colour may be caused by alterations in the relative proportions of the colouring matters and the water of the urine, or by the presence of abnormal substances.

Pale urine is usually of low density and contains a small proportion of solid matter. High coloured urine is usually of high density. The depth of the colour may be affected by the reaction of the urine; other things being equal, an acid urine will have a darker colour than one that is alkaline. Four substances form the bases of the colouring matter of normal urine—urochrome, urobilin, uroerythrin, and hæmatoporphyrin.

Urochrome in aqueous solutions gives a tint like that of urine, and like normal urine, when examined with the spectroscope, shows no absorption bands. It appears to be derived by oxidation from urobilin.

Urobilin is, as a rule, present in normal urine as a chromogen—a body which gives a colour on the addition of a strong reagent, but at times the pure pigment is found in the urine. It is identical with the pigment of the fæces—stercobilin. It is derived from the pigments of the blood and bile.

Uroerythrin is best known as the colouring matter of pink urates. The quantity excreted is very small, but with a high tintorial power.

Hæmatoporphyrin is found in normal urine in minute traces, but in pathological conditions, such as drug toxæmias, *e.g.* sulphonal poisoning, this iron free derivative of hæmatin may be present in considerable amounts.

Chromogens are found in the urine. They are substances which on the addition of a strong reagent are capable of yielding a pigment. Two are recognised—(1) *Indoxyl*, which can be oxidised to indigo-blue, or to the isomeric substance, indigo-red. It is very rare for the urine to be pigmented

with indigo-blue. It is excreted in the form of a conjugated sulphate commonly known as "indican," and requires to be set free before it produces a pigment. Indoxyl is present in the urine in increased amount, whenever putrefactive decomposition of albumin is taking place, as in the increased putrefaction in the intestine seen in constipation, intestinal obstruction, typhoid fever, etc., or in suppuration in abscess cavity or serous sac. The best test for the recognition of indoxyl is that of Jaffé. The urine is mixed with an equal bulk of strong hydrochloric acid, by which means the sulphate (indican) is decomposed and the indoxyl liberated. With a pipette a few drops of a solution of chloride of lime are added, when by oxidation of the indoxyl, indigo-blue is formed. By shaking up the mixture with chloroform, the blue pigment separates out and the chloroform assumes a beautiful blue colour. It is necessary to add the oxidising agent, the hypochlorite, with great care, or the indigo-blue will become oxidised and the blue colour disappear. Obermeyer's test is devised to obviate this difficulty. The urine is first treated with acetate of lead and then filtered. To the filtrate is added an equal bulk of strong hydrochloric acid, containing two to three parts per thousand of ferric chloride. The mixture is shaken for a short time and the liberated pigment taken up, as before, in chloroform. The ferric chloride acts as a mild oxidising agent sufficient to form, but not to destroy, the pigment.

(2) *Urorosein* is a chromogen which gives a red colour on addition of an oxidising reagent, or on treating the urine with hydrochloric acid. It is easily decolorised by treating with alkalies, and is not taken up by chloroform. It is thus easily distinguished from indigo-red.

In diseased conditions the colour of the urine may be altered. **Pale urine** may result from increased excretion of water, as after copious drinking or in the polyuria of nervous conditions; or may depend upon a diminished excretion of pigment, as in diabetes mellitus. **Dark yellow** or **brown-red**

urine is excreted notably in febrile conditions. The colour is due to excess of urobilin and urochrome. It also occurs after internal hæmorrhage, excessive blood destruction, and in some cases of cirrhosis of the liver.

Red or **reddish urine** results from hæmoglobinuria and urinary hæmorrhage.

Brown and **brown-black** urine may be caused by the presence of acid, hæmatin, or methæmoglobin. The latter is the more common, occurring in the condition known as "paroxysmal methæmoglobinuria."

Black urine may result from the presence of melanin, as in melanotic sarcoma.

A dark colour develops in the urine on standing, in the condition known as **alkaptonuria.** If the urine be kept in a urine glass the colour first appears at the upper part and spreads downwards. The colour is deepened on addition of an alkali. On the addition of the urine to boiling Fehling's solution a brown colour, gradually deepening to black, develops, and on prolonged boiling there is a distinct copper reduction. The property of the urine of developing a dark colour on exposure to the air, from the taking up of oxygen, is associated with the presence of pyrocatechin, homogentisic acid and uroleucic acid, aromatic bodies which, when oxidised, develop a colour. The phenomena occurs in health, but is also seen in tuberculosis and other morbid conditions.

Greenish-yellow, greenish-brown, approaching black, discoloration of the urine occurs in jaundice when bile pigments are present.

A **milky urine** results from the presence of finely divided fatty matters in the condition known as chyluria, and from the presence of pus. In chyluria, on standing, a layer of finely divided fatty matter forms on the surface of the fluid. Blood corpuscles can always be detected with the microscope, and albumin is also present. Sometimes fatty matter is present in such a form as to present the appearance of drops of oil on the surface of the urine. If the milky appearance

be due to pus a characteristic deposit will form, at the foot of the vessel, on standing.

Pigmentation of the Urine due to Drugs.—The excretion of certain drugs by the kidneys gives the urine a definite colour. Senna and rhubarb, by means of their chrysophan, give a deep yellow colour to acid and a bright carmine to alkaline urines. Santonin and picric acid produce a brilliant yellow, while juniper gives a greenish colour. Logwood, with alkaline urine, gives a blue or violet colour. Carbolic acid in its different compounds gives a greenish-black colour, which deepens on exposure to the air. The colour is due to the excretion of conjugate sulphates (pyrocatechin-sulphuric acid, hydrochinon-sulphuric acid), which though colourless in themselves readily undergo decomposition and become oxidised, forming the pigment. Methylene-blue, fuchsin, and other aniline preparations pigment the urine.

3. **Variations in the Odour of the Urine.**—Little is accurately known in regard to the smell of the urine. Under ordinary circumstances it has an odour which may be termed aromatic, and which varies in its intensity very considerably. When the alkaline fermentation begins there is an ammoniacal smell, and in cases in which decomposition of the urine takes place, before it is passed, the odour may be intensely putrid, or even sulphurous, from the presence of sulphuretted hydrogen.

The urine frequently assumes the smell of substances which have been taken internally. Radish, onions, and other vegetables give the urine a characteristic odour, while asafœtida, valerian, and many other aromatic drugs act in a similar manner. It must not be forgotten also that turpentine and its congeners produce a smell not altogether unlike that of violets.

4. **Variations in the Reaction of the Urine.**—In health the reaction of the urine is almost invariably acid, probably from the acid phosphate of sodium. The reaction varies, however, at different periods of the day, and becomes less

acid or even alkaline after a full meal. The citrates of potash and soda contained in the vegetables of the food are excreted as soluble carbonates, and give the urine an alkaline reaction. It is a well-known fact that in carnivora the urine is highly acid, while it is alkaline in herbivora.

The acidity of the urine may be shown by its action on blue litmus paper, and may be estimated quantitatively by means of a standard solution of soda, by the same method as is employed in estimating the total acidity of the stomach contents by titration. In the case of the urine cochineal should be used as indicator.

The acidity of the urine may be increased relatively or absolutely. Whenever the quantity of the urine is diminished, as in hot weather from free perspiration, it becomes more highly acid. The acidity may be absolutely increased in disorders of the functions connected with food digestion and tissue change. Increased albuminous metabolism, as in fever or from an excessive meat diet, increases the acidity of the urine. The acidity may be diminished relatively by the increased ingestion of water. It is diminished absolutely in diminished albuminous metabolism, as in anæmic and cachectic states; by the consumption of carbonates and the salts of vegetable acids, which are oxidised and excreted as carbonates; during digestion by the removal of acid from the blood during the secretion of hydrochloric acid by the gastric glands; by the rapid absorption of alkaline transudations and exudations.

Alkalinity of the urine may be caused by the presence of a fixed or volatile alkali, and may be shown by its action on red litmus and on turmeric paper. Permanently alkaline urine, so called from the fact that the blue stain given by it to red litmus paper does not disappear on drying, is due to the presence of carbonates of potassium and sodium in excess. The causes of the excessive elimination of these salts are to be found in the internal use of alkalies and their salts, taken either in the food or as drugs.

Temporary alkalinity of the urine is produced by the presence of carbonate of ammonium, and the blue colour which it gives to red litmus paper fades on drying. Decomposition of urea while the urine is in the bladder, or after it has been passed, is the sole cause giving rise to this volatile alkalinity of the urine. It has its origin in the presence of micro-organisms, and when present in freshly passed urine indicates an unhealthy state of the urinary tract. The ammoniacal fermentation of the urine always gives rise to the presence of deposits of calcium phosphate and ammonio-magnesium phosphate. It is accompanied by a film, usually somewhat iridescent in appearance upon the surface, which consists mainly of these two earthy phosphates.

An amphoteric reaction is sometimes given by the urine, by which blue litmus is turned red and red litmus blue. It is due to the simultaneous presence of monosodium phosphate and disodium phosphate, acid and alkaline phosphates.

5. **Variations in the Specific Gravity of the Urine.**—The specific gravity of the urine is ascertained by means of a modification of the hydrometer specially adapted for the purpose, and termed therefore a *urinometer*. The scale which it contains should begin with the specific gravity of distilled water, taken as 1000, as zero, and should register as far as 1050. The instrument should be carefully tested in distilled water before it is employed to examine urine.

The specific gravity depends upon the relative amount of the fluid and solids of the urine, and in health usually varies between 1015 and 1025.

The proportion of solids in the urine, as we have already seen, is on an average 60 grms. in 1500 c.c., or 1 in 25—that is, 4 per cent. As the normal specific gravity of the urine is 1020, we find a simple method of estimating the approximate quantity of solids by multiplying the last two figures of the specific gravity by 2·2, in order to obtain the bulk of solids in 1000 c.c. Having done this, it is easy to calculate the amount of solids in any given quantity of urine.

This calculation is commonly known as the *co-efficient of Trapp*; in urine with a specific gravity exceeding 1025, more accurate results are obtained by using the *co-efficient of Haeser—i.e.* by multiplying the last two figures by 2·33.

Marked and persistent deviations from the average specific gravity point to the probable presence of certain diseases. The specific gravity may be much increased, while the amount of urine undergoes little or no change, in certain disorders of metabolism resulting in the production of an excess of urea. Increased specific gravity, with lessening of the quantity of urine, is found in acute febrile disorders. Greatly increased specific gravity, with an increased flow of urine, points to the probability that diabetes mellitus is present. Diminution of the specific gravity, without an increase in the amount of urine points either to diminished metabolism, as may be seen towards the fatal termination of a fever; or to the products of metabolism not being excreted by the kidneys, through circulatory disturbances. It is a grave sign in the course of a nephritis, and may herald an attack of uræmia. Diminished specific gravity, with increased quantity of urine, points to a cirrhosis of the kidney with cardiac hypertrophy; to waxy disease; to diabetes insipidus, in which the total solids are not absolutely diminished; or to hydruria from nervous or other causes, in which the solids are absolutely lessened. The urinometer in these last two disorders affords great aid in diagnosis.

6. **The Naked-eye Characters of the Deposit.**—When normal urine stands for some time a mucous cloud develops. It has the appearance of a semi-transparent, filmy cloud, occupying the lower fourth of the glass. If the specific gravity of the urine be high, the mucous cloud may float in the upper part of the fluid. The substance is probably not true mucin, but a nucleo-proteid.

Phosphates may separate out of the urine and form a

deposit if the urine be neutral or alkaline. The deposit is flocculent and colourless. If treated with acetic acid it passes into solution.

Pus forms a dense white deposit, denser than the phosphate deposit, and not showing the flocculent character. Acetic acid does not dissolve the deposit, and the addition of a caustic alkali renders the deposit ropy. Pus and phosphates are often associated.

Urates may form a distinct sediment even in health. The deposit is usually of a reddish-yellow or pink tint, from the presence of uroerythrin. The deposit may, however, be quite white. Urates are characterised by the fact that they are dissolved on heating the urine, and reappear on cooling; they are unaffected by acetic acid, but dissolved by strong acids, to form crystals of uric acid. The deposit consists of the amorphous quadriurates of potassium, sodium and ammonium.

Acid urate of soda is rare as a deposit, but it may occur as a yellowish, sandy looking sediment in acid urine. It is not readily soluble on heating.

Urate of ammonia occurs in ammoniacal urines, forming a yellowish granular deposit, soluble in acetic acid. It is associated with the presence of triple phosphates.

Uric acid appears as a scanty deposit, in its free crystalline form, in the shape of small grains of reddish colour, somewhat resembling sprinkled cayenne pepper.

Oxalate of lime crystals are usually seen, not as a thick deposit but entangled in a mucous cloud, giving it a glittering appearance when the glass is held up to the light. It must be noted that the presence of crystals of calcium oxalate indicates merely precipitation, not increased excretion, of the salt. Tyrosin, cystin and calcium carbonate, rarely form deposits.

Variations in the Chemical Characters of the Urine.—The urine may depart from the healthy condition in two ways—on the one hand, by alteration in the quantity

of the normal constituents; and, on the other, by the presence of abnormal substances.

The normal constituents of the urine, to which, for practical purposes, attention must be directed, are—(1) The products of nitrogenous metabolism; (2) the inorganic constituents.

The Products of Nitrogenous Metabolism.—The total urinary nitrogen amounts on an average diet to 15 grms. in twenty-four hours, the greater part, in fact, of the nitrogenous loss of the body.

The urinary nitrogen is distributed in various compounds. About 86 per cent. of the whole is in the form of urea, 3 per cent. as ammonia, 3 per cent. as kreatinin, 2 per cent. as uric acid and the allied xanthin bases, while the remaining 6 per cent. is, in varying proportions, in hippuric acid, indol, skatol, urinary nucleo-albumin, the pigments and unknown bodies.

For the estimation of the total nitrogen, Kjeldahl's process is employed. A portion of the urine is heated with concentrated sulphuric acid, organic substances are oxidised, and all the nitrogen not originally present in combination with oxygen, is converted into ammonia. The resulting ammonia is liberated with a caustic alkali and distilled into a measured quantity of a standard acid solution. The acid is then titrated with a standard alkali, and from the loss of acidity the nitrogen can be calculated.

The details of the process are—

Five c.c. of filtered urine are taken in a small conical flask, 20 c.c. of strong sulphuric acid and a small globule of mercury added. The flask is then placed over the flame of a bunsen and gently boiled till all the colour is discharged, which takes place in about an hour, showing the complete oxidation of the organic substances. The nitrogen present has now been converted into ammonia, and the ammonia has combined with the sulphuric acid to form ammonium sulphate. The fluid is now allowed to cool, and when cool

is transferred to the large flask of a distilling apparatus. The small flask is washed with distilled water, and the washings added to the original fluid. To the acid mixture add next about 100 c.c. of a saturated solution of caustic soda, and 10 c.c. of a saturated solution of potassium sulphide. The flask is then connected with the condenser of the distilling apparatus. The end of the condenser passes into a U-shaped tube. In this tube is placed 50 c.c. of a decinormal solution of sulphuric acid. The distilling flask is heated, and the ammonia being set free by the alkali passes through the condenser and is received in the decinormal acid solution, and combines with part of the acid. After all the ammonia has passed over, which is as a rule after about an hour's heating, the U-tube with the acid solution is disconnected, and after this has been done the flame under the flask is extinguished. The ammonia having combined with the sulphuric acid, the decinormal acid solution has lost in acidity by the amount of acid in combination, and it is only necessary to find how much acid has combined with the ammonia to ascertain the amount of the ammonia, and thus the amount of nitrogen. This is done by titrating the acid solution with a decinormal soda solution, using cochineal as an indicator.

Suppose that in the titration 20 c.c. decinormal soda solution are used to neutralise the acid solution, this indicates that of the original 50 c.c. acid solution, 30 c.c. have been neutralised by the distilled ammonia.

Thirty c.c. N/10 acid solution = 30 c.c. N/10 ammonia solution = 3 c.c. normal ammonia solution. 1 c.c. normal ammonia solution contains 0·017 grms. ammonia, of which 0·014 grms. are nitrogen.

In the case under discussion, 5 c.c. urine contains $0{\cdot}014 \times 3 = 0{\cdot}042$ grms. nitrogen. If the patient is passing 1500 c.c. urine in twenty-four hours—

$$\frac{1500 \times 0{\cdot}042}{5} = 12{\cdot}6 \text{ grms. N.}$$

The amount of the total nitrogenous excretion in the urine depends upon the intake of nitrogen in the food, and the metabolism of proteids in the body. It is not much affected by exercise. It is markedly increased under conditions which produce increased metabolism, as in fever, diabetes mellitus, etc. It is diminished when the excretory powers of the kidneys are impaired, as in nephritis.

Urea forms 86 per cent. of the total nitrogen excretion. The products of metabolism are carried to the liver probably as lactate of ammonia, there to be converted by the liver cells into urea.

The presence of urea can always be demonstrated by evaporating the urine, if of ordinary specific gravity, to about half its bulk, and on cooling adding an equal quantity of nitric acid, when shining rhombic crystals of nitrate of urea separate out. This reaction, as will be seen subsequently, is apt to mislead the observer in testing for albumin. If the urine is of high specific gravity, it is often unnecessary to concentrate it by evaporation, previous to the addition of nitric acid.

Estimation of urea.—There is no known method for the quantitative separation of urea as urea. The best method of estimation is to separate the other nitrogenous constituents, and, by determining the nitrogen remaining, the amount of urea present can be calculated. A useful method is that of Mörner and Sjöquist. Five c.c. of urine is treated with an equal volume of a saturated solution of barium chloride containing 5 per cent. caustic baryta; 100 c.c. of an alcohol-ether mixture (alcohol 2 parts, ether 1 part) is added, and the whole allowed to stand for twenty-four hours in a closed flask. The mixture is then filtered, and the filtrate evaporated at a low temperature (below 60°), and the nitrogen of the residue determined by Kjeldahl's method. All the nitrogenous substances are precipitated and removed by the filtration, save ammonia and urea. The ammonia passes off in the process of evaporation, the only nitrogenous

body remaining for the final estimation being urea. The percentage figure of nitrogen multiplied by 2·143 gives the percentage of urea.

A rough and useful volumetric analysis by decomposition, may be performed by means of a freshly prepared solution of hypobromite of sodium or chlorinated soda. The former reagent is to be preferred. It must not be forgotten that other nitrogenous bodies besides urea are broken up in this process, but it is also to be remembered that there is also a deficiency in the volume of nitrogen evolved as tested with a pure solution of urea.

For this process a special apparatus is necessary. It consists of—

A flask furnished with an indiarubber stopper, through which a glass tube is inserted.

A basin of water into which the flask may be put.

A small test tube graduated to 5 c.c.

A tube about 40 cms. in length, with a calibre of about 1·75 cm.

This tube is drawn out at one end to form a smaller tube, connected with the flask by a piece of indiarubber tubing. The tube, placed with the small end uppermost, has a zero mark upon it about half an inch below the upper end. From this zero mark downwards a space equal to 55 c.c. is divided into 30 parts of equal capacity. A volume of nitrogen, filling one of these divisions and evolved from 5 c.c. urine, is exactly equivalent to 0·1 per cent. of urea, or 0·1 grm. of urea in 1000 c.c. of urine.

A glass cylinder, to be filled with water, in which the graduated tube is held by means of a clamp.

A graduated measure with a mark for the quantity of bromine, which is 1·3 c.c., and another for the solution of caustic soda, which is 13·7 c.c., the total bulk of the solution of hypobromite of sodium employed being 15 c.c.

One of the usual forms of this apparatus—that of Russell and West—is figured.

The solutions required are—

Bromine.

A solution of caustic soda, containing 100 grms. of caustic soda in 250 c.c. of water.

The process is to be carried out in the following manner:—

Measure out the 1·3 c.c. bromine, add the 13·7 c.c. caustic soda solution, and after mixing them well pour the fluid into the flask.

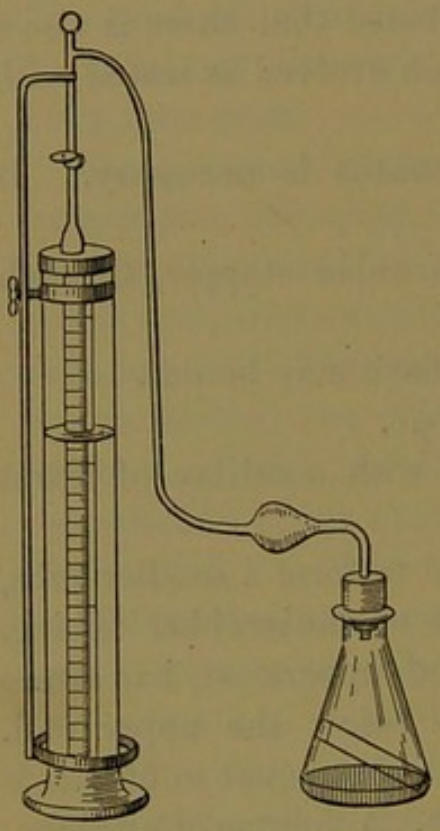


Fig. 100.—Ureometer of Russell and West.

Measure out 5 c.c. urine in the small test tube, and by means of a forceps, or the point of the finger, lower it into the flask, taking care that none of it spills into the hypobromite solution. Fix the stopper firmly in the flask, which will then be in communication with the graduated tube.

See that the water in the glass cylinder and in the graduated tube stands at the zero mark on the latter.

Leave the apparatus for ten minutes until the temperature becomes uniform throughout the system, and then incline the flask so that the urine flows from the small test tube into the flask, gently shaking to thoroughly mix the two fluids. A considerable amount of effervescence ensues with the development of heat, and the water sinks in the graduated tube from the evolution of nitrogen. The completion of the reaction will be known by the cessation of effervescence.

During the process the flask must be kept cool by immersion in cold water. Fifteen minutes after the reaction is at an end, when the apparatus and contents have cooled down to the surrounding temperature, the water is to be

brought to the same level in the tube and outside of it. The index is then to be read, and the number of spaces occupied by nitrogen gives the percentage of urea expressed for each space as 0·1 grm. urea in 100 c.c. urine.

If, for instance, fifteen spaces are filled by the gas, the urine contains 1·5 per cent. of urea, and if there be 1600 c.c. passed in twenty-four hours, then

$$100 : 1600 : : 1{\cdot}5 : 24$$

or the quantity of urea is 24 grms. in that period.

This process, as was referred to above, falls short of the absolute amount of urea by 8 per cent.; that is, at 0° C. and 760 mm. pressure, 1 grm. of urea evolves only 342·9 c.c. of nitrogen, instead of 372·7 c.c., which is the theoretical amount. Knowing this fact, it is perfectly easy to calculate the exact quantity of urea contained in any given urine.

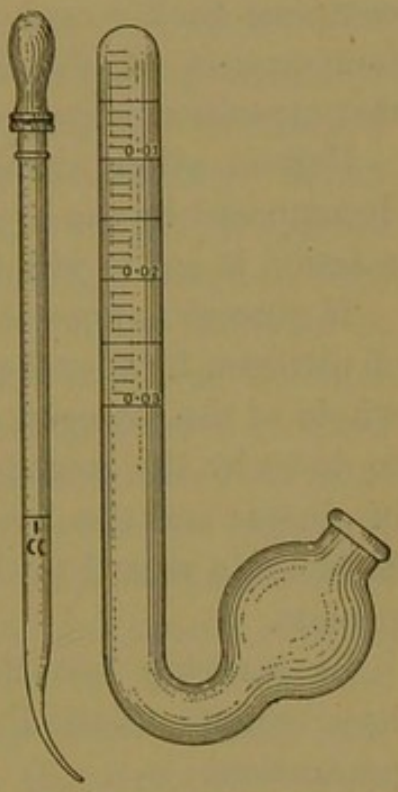


Fig. 101.—Ureometer of Doremus and Thursfield.

A very simple and exceedingly useful modification of this process has been introduced by Doremus and Thursfield. The apparatus consists of a graduated tube and a pipette, which are shown in Fig. 101. The tube is closed at the upper end, curved at the lower end, and terminates in a bulb. The upper part is graduated, each division indicating 0·001 grm. of urea in 1 c.c. of urine, and a mark at the bend shows how much of the reagent is required. The pipette has a mark indicating 1 c.c., and an indiarubber bulb for suction. In order to use the apparatus, pour the solution of sodium hypobromite up to the mark, filling the graduated arm, and add sufficient water to fill the bend and lower part of the bulb. Draw the urine into the pipette as far as the mark on

it. Pass the pipette into the tube as far as the bend, and slowly squeeze the indiarubber bulb so as to eject the urine, which rises through the hypobromite of sodium solution, evolving the gas which rises to the upper part of the tube. Each division indicates 0·001 grm. of urea from the 1 c.c. of urine, and the percentage is obtained by multiplying the result by 100. The objections, however, to this method are twofold: it is exceedingly difficult to prevent the nitrogen escaping back round the bend of the instrument, and the temperature of the fluid cannot be controlled while the decomposition is proceeding.

Certain other nitrogenous substances found in urine are decomposed by the hypobromite of sodium, and this chemical reaction is apt to vitiate the result.

If glucose is present in the urine it increases the amount of nitrogen by 6 or 7 per cent.—that is, it causes almost the whole of the nitrogen of the urea to be evolved. It appears to do so by the great heat which is produced by the mixture of glucose and the hypobromite solution, and hence the precautions in regard to the cooling of the apparatus during the process.

In carrying out the volumetric method with sodium hypobromite, Noël Paton urges attention to the following precautions:—

(1) That the same apparatus and same strength of hypobromite solution be always used, and that they should be first tested upon a standard solution of urea.

(2) That the hypobromite solution should be freshly prepared each day.

(3) That the urine, when concentrated, should be diluted to a specific gravity of between 1010 and 1020.

(4) That full time be allowed for the temperature throughout the apparatus to become uniform before and after the decomposition.

(5) That the volume of gas be corrected for temperature and pressure.

(6) That throughout the decomposition the temperature of the flask be kept low by submerging it in a large basin of water at the temperature of the room.

The daily quantity of urea is chiefly dependent on the amount of nitrogen taken in the food. This is a fact which should never be forgotten by the physician.

Ammonia.—The urine of man contains ammonia in the proportion of from 0·3 to 1·2 grms. in twenty-four hours. When the proportional acid production in metabolism exceeds the supply of bases in the food, urinary ammonia increases at the expense of urea. This may also be brought about by the administration of acids by the mouth. When bases taken by the mouth are in excess, ammonia disappears from the urine, and there is a corresponding increase in urea excretion. Abnormal acid production, with increase in ammonia excretion, is seen in such conditions as diabetes and fevers, and where the hepatic cells possess a reduced activity for the hydrolysis of ammonium carbonate into urea. For the estimation of ammonia, 25 c.c. of urine are placed in a basin with vertical sides, and 20 c.c. milk of lime added. A glass triangle is placed over the basin, and upon it a small vessel containing 20 c.c. of one-fifth normal sulphuric acid solution. The whole stands upon a glass slab covered with a bell-glass, which fits air-tight, on the slab. Absolute air-tight fitting may be ensured by smearing the rim of the bell-jar with lard. The ammonia of the urine is liberated by the lime without the decomposition of the other nitrogenous constituents, and in the course of two or three days has all been absorbed by the sulphuric acid, the diminution in the acidity of which can be estimated by titration with decinormal soda solution with a cochineal indicator.

Uric acid is closely related to urea, which it yields under a combined process of oxidation and hydrolysis. It belongs to the class of substances known as diureïdes, in which the residue of two urea molecules are united to a carbon-con-

taining nucleus, which in the case of uric acid contains a chain of three carbon atoms.

Uric acid in the urine is present in a proportion far in excess of the amount soluble in an equal volume of water. The presence of neutral salts and also of urea enhance the solubility of the acid, but it is probable that the acid is present not as free acid but as a soluble salt. In normal urine, after its secretion, all the acid is present as a hyper-acid salt, a quadriurate. In the presence of water these salts are unstable and tend to decompose, forming free uric acid and biurates. The biurates, however, are in the presence of acid phosphates, and are therefore unstable, and quadriurates are again formed, and become subject to the same conditions as before. This cycle persisting, the whole uric acid is in time set free. For this precipitation of uric acid it must always be remembered that an excess of acid phosphates is necessary. If there be a balance between acid phosphates and basic (monohydrogen) phosphates, uric acid is not precipitated, for there is not the acid phosphate present to convert the biurates into quadriurates.

Uric acid is thrown out of its combinations with bases by the addition of strong hydrochloric acid, and is then, on account of its insolubility, precipitated; it may in this way be procured for the purpose of examination. It can be recognised chemically by means of the murexide test, which is also applicable to the salts which it forms with alkalies and alkaline earths. This test is performed with crystals of these substances, or with the residue obtained by evaporating urine. It consists in the addition of a drop of strong nitric acid and subsequent heating to dryness; there is left a reddish-brown deposit on the porcelain capsule, and this, when touched with a glass rod, which has been dipped into a strong solution of ammonia, at once assumes a crimson tint. If caustic potash is used instead of ammonia, a violet colour is obtained.

Uric acid, when examined microscopically, is found in

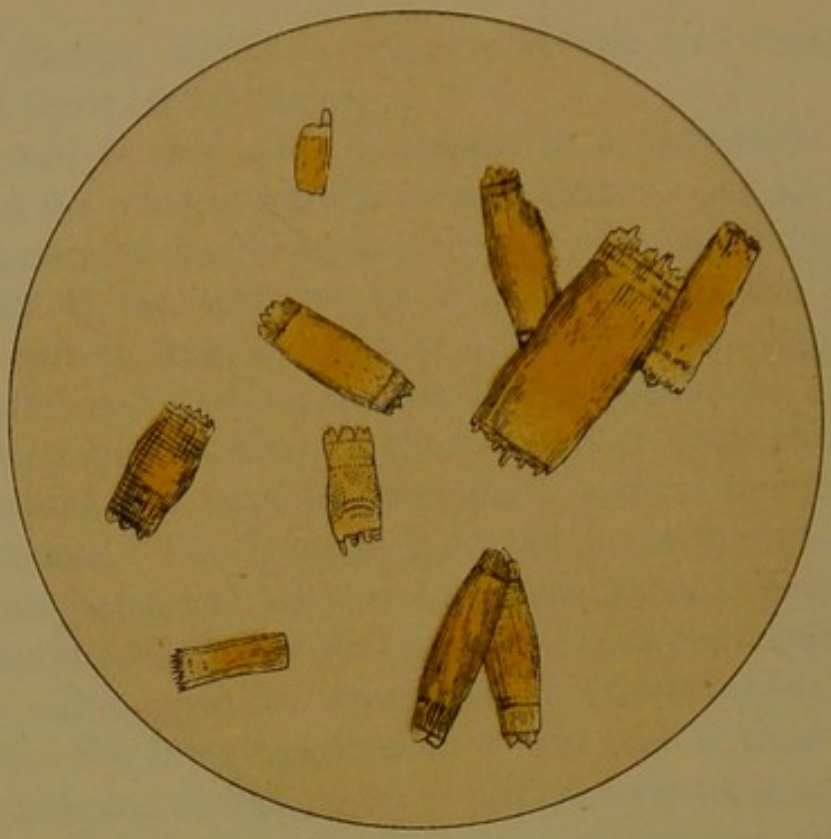


Fig. 102.—Uric acid.

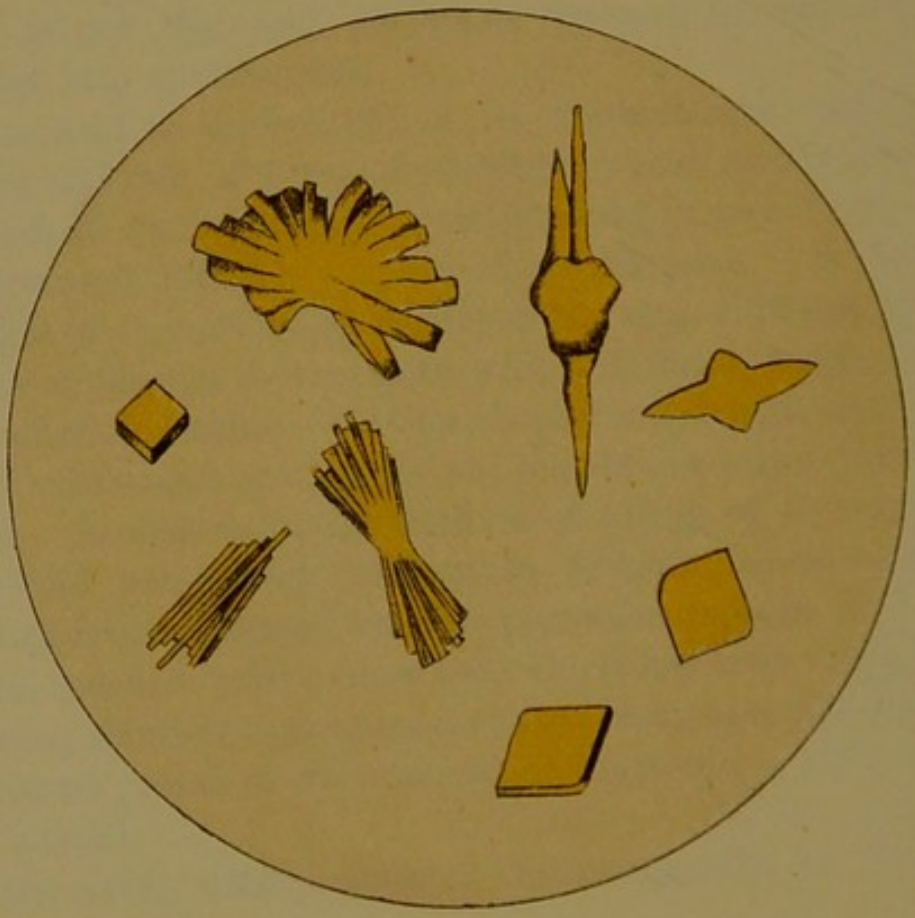


Fig. 103.—Uric acid.

rhombic crystals of many different forms, some of which are shown in Fig. 102 and Fig. 103. They are nearly always of a brown colour. Lozenge and oval-shaped plates and spikes or rods are the most common forms of crystal; these are frequently grouped together to form sheaf-like or fan-shaped bodies, along with stellate and rose-formed collections.

Quantitative estimation of uric acid is best carried out by *Hopkins'* method, which depends upon its separation as an ammonium salt. The danger of reduction is thus obviated. *Estimation in a normal urine without deposit,*—100 c.c. of urine are taken, and powdered ammonium chloride added till practical saturation is obtained; about 50 grms. ammonium chloride are required. The mixture is allowed to stand for two hours, with occasional stirring. The precipitate is filtered through a thin filter paper, and washed two or three times with saturated ammonium chloride solution. The filtrate should remain clear and bright.

With a jet of hot water the pigmented urate is next washed off the filter paper into a clean beaker, and heated just to boiling point with excess of hydrochloric acid. It is then put aside in a cool place to allow the separation of the uric acid. Two hours should be enough for this. The precipitated uric acid is then collected on a filter paper and washed with cold distilled water. Before washing, the filtrate must be measured, and 1 mgrm. added to the final total for each 15 c.c. of the filtrate, which need never exceed 20 to 30 c.c. in amount.

The acid is next washed off the filter with hot water, warmed with the addition of sodium carbonate to the solution, till all is dissolved and the solution made up to 100 c.c. It is next transferred to a flask of sufficient capacity and mixed with 20 c.c. strong sulphuric acid, and immediately titrated with one-twentieth normal potassium permanganate solution. The titration must be done slowly towards the end of the reaction, the close of which is marked by the first appearance of a pink colour which is

permanent for an appreciable interval. The flask should, of course, be agitated throughout the operation.

The permanganate solution is prepared by dissolving 1·578 grms. of pure potassium permanganate in 1 litre of water, 1 c.c. of this solution is equal to 0·00375 grms. of uric acid. The addition of the sulphuric acid to the solution produces a temperature suitable to the reaction.

An abbreviated and fairly accurate method may be used in clinical work. A filter is prepared by packing a small plug of glass wool tightly into the neck of a small glass funnel; 20 c.c. of urine are taken and saturated with ammonium chloride, put to stand for two hours and then filtered through the filter, which is then washed with a saturated solution of ammonium sulphate. This removes chlorides, which would interfere with the titration. After washing with the sulphate solution, the plug of glass wool with the entangled precipitate is placed in a small flask and boiled with a little sodium carbonate solution, and titrated with the permanganate after the addition of sulphuric acid, as in the full process. A weaker solution of permanganate should be used—one-fiftieth normal, 1 c.c. of which equals 0·0015 grms. uric acid.

Say that in titration 2·5 c.c. permanganate solution have been used, 2·5 = 0·00375 grms. uric acid.

$$20 : 0{\cdot}00375 :: 100 : x$$

$$\frac{100 \times 0{\cdot}00375}{20} = 0{\cdot}1875 \text{ grms. uric acid per cent.}$$

The quantity of uric acid excreted in relation to diet is not yet fully established. The rôle played by the albumin and globulin of the diet is not certain, but there is no doubt that the ingestion of nucleo-proteids increases the urinary uric acid. In the adult an average excretion is from 0·5 to 1 grm. in twenty-four hours. This is increased by excessive exercise and diminished by rest. Drugs affect the excretion. The action of alkalies is still disputed. Isolated doses may increase, but continued administration appears to diminish

the excretion. Salicylates increase urinary uric acid, and the same is true of pilocarpine; the action of the latter drug being probably effected by the production of a leucocytosis. In leukæmia there is marked increase. In gout the question of increased excretion is still undecided. In disease where leucocytosis is present the excretion is increased. In general, therefore, in disease, whenever there is destruction of nuclein going on in the body, uric acid excretion increases. The separation of uric acid or urates from the urine on standing does *not* indicate an increased excretion of uric acid, though increase may be present.

The Purin bases—bodies which are present in the urine in small amount—are closely related to uric acid.

The amount of purin bases in the urine is about one-tenth of the uric acid present, 0·1 to 0·05 grms. in twenty-four hours. The method of estimating these bodies is not applicable to clinical work.

Variations in the amounts of the purin bases closely follow the variations in uric acid, and are dependent for the most part on the same influences. They are increased by a diet rich in nucleins and, pathologically, in leukæmia.

The Kreatinin of the urine is a derivative of the kreatin of muscle. Its presence in urine may be demonstrated by adding a small quantity of a dilute solution of nitroprusside of soda, and then drop by drop a dilute solution of caustic soda, when a ruby-red colour develops, varying in intensity with the amount of kreatinin present. This colour soon passes into a straw-yellow tint, becoming blue on the addition of ammonia.

The quantity of kreatinin in the urine averages 1 grm. in twenty-four hours. Variations follow closely those of urea, and the quantity largely depends upon the amount of kreatin taken with the food. It is increased pathologically in febrile conditions and diabetes, diminished in progressive muscular atrophy and pseudo-hypertrophic paralysis.

Kreatinin has the power of reducing copper sulphate on boiling in alkaline solutions.

Indoxyl potassium sulphate and skatoxyl potassium sulphate, through nitrogenous substances, are closely related to the aromatic bodies. *Indoxyl* sulphate of potash is derived from oxidation in the body of the indol absorbed from the bowel and linking to sulphate of potash, and its amount in the urine is increased by all causes which promote the bacterial decomposition of proteids in the intestine, and by causes, such as constipation, which promote the absorption of indol. *Skatoxyl* sulphate of potash is derived from skatol, and goes hand in hand with the indoxyl sulphate, being also a conjugated sulphate. By oxidation, indoxyl forms indigo-blue and indigo-red, while skatoxyl yields red pigments.

The **phenol** of the urine is in very small amount, and mainly as its homologue **kresol,** and isomeric forms of the latter such as **parakresol. Pyrocatechin** and **hydrochinon** are isomeric substances, the former constant in normal urine, the latter only under exceptional circumstances. **Inosit,** or "muscle-sugar," contains a benzine nucleus and belongs to this group. It appears frequently in normal urine, when polyuria is induced by diuretics or copious drinking. It may occur in diabetes. It is crystalline, being not unlike cholesterin in appearance. It is optically inactive, and does not ferment. Of the aromatic oxyacids a number have been identified in the urine. Pathologically, these aromatic substances are important. In carbolic-acid poisoning many are increased in the urine; pyrocatechin and hydrochinon may be present in large amounts, and by their oxidation give rise to the pigmented appearance known as "**carboluria.**" In certain diseases other members of the group are increased in amount, and produce the condition known as "**alkaptonuria.**" The urine, on standing, develops a dark colour. The phenomenon is due to the presence of uroleucic acid, homogentisic acid or pyrocatechin. The quantity of phenol

in the urine is greatly increased in constipation and intestinal obstruction.

Hippuric acid in the urine amounts, on a mixed diet, to about 0·7 grm. per diem, while on a diet rich in fruit it may increase to three or four times this amount. It forms four-sided prismatic crystals ending in four facets, and often grouped in clumps.

The Inorganic Constituents of the urine to which it is necessary to call attention are chlorides, phosphates and sulphates.

Hydrochloric acid (HCl) is passed out in the urine chiefly in combination with sodium, but also to a smaller extent combined with potassium and ammonium. As the chlorides are soluble they never form urinary deposits, but if a few drops of urine are evaporated in a watch glass, octahedral crystals and rhombic plates of chloride of urea and sodium are formed.

Chlorides may be detected by the addition of a solution of nitrate of silver (1 to 8), which throws down a white precipitate of silver chloride, but, as phosphates also form new compounds with solutions of nitrate silver, a few drops of nitric acid should be added, which prevents the precipitation of phosphate of silver. All albuminoids should also be removed before testing.

Quantitative estimation of chlorides can be carried out by means of Mohr's nitrate of silver method. The method depends upon the fact that when, to a neutral fluid containing chloride and phosphate of sodium and a neutral salt of chromic acid, a solution of nitrate of silver is added, there first occurs a precipitation of chloride of silver, when all the chlorine in the chloride of sodium is precipitated, then if more silver be added a red chromate of silver forms.

The solutions required are—

A solution of nitrate of silver, prepared by dissolving 29·075 grms. of fused silver nitrate in 1 litre of water. This solution is of such a strength that 1 c.c. is

equivalent to 0·01 grm. of sodium chloride, or 0·006 grm. of chlorine.

A saturated solution of neutral potassium chromate to show the end-reaction.

To conduct the process, take 10 c.c. of the urine after it has been filtered, add a few drops of the solution of potassium chromate, stir the mixture well, and gradually add from a burette the silver nitrate solution. Reddish spots appear as the solution falls, but disappear on stirring, so long as any chloride of sodium remains in solution. As soon, however, as all the chlorine of the sodium has combined with the silver, a permanent colour forms from the presence of chromate of silver.

The number of cubic centimetres of the silver nitrate solution employed gives the amount of sodium chloride (and hence of chlorine) in 10 c.c. of urine; from which the calculation of the daily excretion is easy. If, for instance, 5 c.c. of the solution are used, then $5 \times 0{\cdot}01 = 0{\cdot}05$ grm. of sodium chloride, or $5 \times 0{\cdot}006 = 0{\cdot}03$ grm. of chlorine in 10 c.c. And if the daily amount of urine is 1600 c.c., then

$$10 : 1600 : : 0{\cdot}05 : 8$$

the amount of sodium chloride is 8 grms., or

$$10 : 1600 : : 0{\cdot}03 : 4{\cdot}8$$

the quantity of chlorine is 4·8 grms. in twenty-four hours.

The quantity of chlorides excreted in health varies in direct proportion to the quantity taken with the food, and may be said to lie within the limits of from 5 to 8 grms. daily.

In disease the quantity may depart greatly from the normal. Ague, during the cold and hot stages, causes a considerable increase, but this is the only febrile affection in which there is any augmentation. In every other disease attended by pyrexia the amount is diminished, especially in acute pneumonia, where a total disappearance of chlorides

may occur, as well as in diseases accompanied by exudations, such as pleurisy with effusion.

Phosphoric acid (H_3PO_4) is the only acid of phosphorus which occurs in the urine, and as a tribasic acid forms three orders of salts. Acid phosphates or dihydrogen phosphates, where two hydrogen atoms of the radicle are intact, are soluble and give an acid reaction to litmus. Monohydrogen, where two hydrogen atoms are replaced by a base, and normal phosphates, where all are replaced by a base, are alkaline to litmus. While all the phosphates of sodium, potassium and ammonium are freely soluble in water, only the dihydrogen phosphates of the alkaline earths are at all freely soluble, the monohydrogen and normal phosphates being insoluble.

In addition to the phosphates of the alkalies and alkaline earths, a certain amount of phosphoric acid is excreted as glycero-phosphoric acid.

Phosphoric acid in the urine is derived in great part from the food, but a proportion results from the breaking down of phosphorus-containing tissues, such as bone and nervous tissue, by the oxidation of nuclein, lecithin and protagon.

Its presence may be demonstrated by the addition of a solution of ammonium molybdate in nitric acid and water, which gives a yellow precipitate. On boiling urine which contains a considerable quantity of phosphates there may be, even when it is acid, a distinct cloud or turbidity, which disappears on cooling and on the addition of a dilute acid. The usual explanation of this fact is that the heat dissipates the carbonic acid of the urine, which is believed to keep the calcium phosphate in solution. This does not explain the disappearance of the cloud on cooling, and it seems more probable that the cause of these appearances is, as Smith has suggested, an alteration of the proportions in which the alkaline earth bases unite with the phosphoric acid. On the addition of an alkali to the urine the earthy phosphates are precipitated, calcium phosphate as a rule in an amor-

phous form, and magnesium phosphate as a fine crystalline deposit. It must be borne in mind that when the urine is alkaline, phosphates are apt to be precipitated in the bladder, and in this case the first urine voided is clear, while that which is passed towards the end of micturition is thick and muddy.

Quantitative estimation of phosphates is effected by means of an acid solution of uranium nitrate, which precipitates an insoluble phosphate of uranium; and the completion of the process is known by the fact that the nitrate gives a reddish-brown colour with ferrocyanide of potassium, while the phosphate causes no change in colour. An acid solution of acetate of sodium is employed in the process in order to make sure of the entire precipitation of the uranium phosphate.

The following are the solutions required:—

Solution of uranium nitrate prepared by dissolving 33 grms. of yellow oxide of uranium in nitric acid of 1200 specific gravity, adding water to 1100 c.c., and adjusting, by operating on a solution of sodium phosphate of known strength, the solution to such a strength that 1 c.c. is equivalent to 0·005 grm. of anhydrous phosphoric acid.

Solution of acetate of sodium prepared by dissolving 100 grms. of the salt in 100 c.c. of dilute acetic acid, and adding water to 1 litre.

A saturated solution of ferrocyanide of potassium.

To conduct the process take 50 c.c. of urine, add 5 c.c. of the sodium acetate solution, and heat and keep heated just short of the boiling point during the titration. Add by means of a burette the uranium nitrate solution, stirring meanwhile until no precipitate is formed, and when this point is reached place a drop of the mixture on a porcelain plate along with a drop of the ferrocyanide solution. If no colour is produced, enough uranium nitrate solution has not been added. The uranium has a greater affinity for phosphates than for the ferrocyanide, and while phosphoric acid

remains unsatisfied will not unite with the ferrocyanide. Continue the titration till the ferrocyanide indicator gives a reddish-brown colour, showing the presence of slight excess of the uranium solution. The number of cubic centimetres of the uranium nitrate solution required represents the quantity of phosphoric acid in 50 c.c. of urine, from which the daily amount may be calculated. For instance, if 14 c.c. are used, $14 \times 0{\cdot}005 = 0{\cdot}07$, and if the daily amount of urine is 1600 c.c., then

$$50 : 1600 : : 0{\cdot}07 : 2{\cdot}24$$

the daily excretion of phosphoric acid is 2·24 grms.

This process gives the total amount of phosphoric acid, whether combined with alkalies or alkaline earths, but not the glycero-phosphoric acid.

The usual quantity of phosphoric acid which is excreted daily is from 2 to 4 grms. It varies considerably with the nature and quantity of the food and the state of the nutritive processes.

Sulphuric acid (H_2SO_4) appears in the urine as preformed sulphuric acid in organic combination, the so-called conjugated or ethereal sulphates which contain organic radicles, or as inorganic compounds with potassium and sodium, and, as its compounds with these metals are very soluble, no urinary deposits of this nature are to be met with. Normally, the conjugated sulphates form one-tenth of the sulphuric acid present. Most of the aromatic substances are present in the urine as conjugated sulphates, and the proportion of the sulphuric acid present in this form depends upon the factors which increase or decrease these aromatic substances. The chief salts are indoxyl-, skatoxyl-, and kresyl-sulphuric acid.

Sulphates may be detected by means of a solution of barium chloride or nitrate, which throws down, even in acid solution, a white precipitate of barium sulphate insoluble in acids.

The quantitative estimation of the sulphates of the urine, while important, is, when accurately carried out, an elaborate process, and necessitating, as it does, the use of laboratory apparatus, it is inapplicable to clinical work.

The daily average of sulphuric acid passed in the urine depends upon the proteid of the food, from the sulphur of which it is derived. As a general rule the total sulphuric acid in the urine of an adult on a mixed diet averages 2 to 4 grms. The quantity is increased on a proteid diet, and after the use of certain vegetables which contain sulphur. It is increased by any condition which hastens the rapidity of tissue metabolism, such as fever. The organic sulphates are increased proportionally by the putrefactive decomposition of proteid in the alimentary canal.

The Abnormal Substances of the Urine

Proteids.—Normal urine contains but traces of substances belonging to the proteid group. Minute quantities of nucleo-proteid are derived from the cells of the urinary passages, but the quantities are so small as to be very difficult to demonstrate. It may, however, be present in appreciable quantity in the urine of apparently healthy people. The proteids usually present in disease are serum albumin, serum globulin, albumoses and peptone. Serum albumin may, under certain circumstances, become changed into acid albumin or alkali albumin. This may take place spontaneously if the urine is highly acid or highly alkaline.

In testing for proteids the urine should be filtered if turbid. If very highly acid it should be rendered less so by liquor potassæ. If alkaline it should be made slightly acid by means of acetic acid.

Tests for Serum and Albumin and Globulin.—(1) **Heat Test.**—The heat test depends upon the fact that serum albumin and globulin are coagulated at a temperature

of 75° C. and over. Coagulation takes place more readily in the presence of excess of a neutral salt.

Half an inch of urine is taken in a test tube and acidulated by the addition of a few drops of acetic acid and about 20 drops of a saturated solution of acetate of potash or of sodium chloride added. The mixture is then heated. If albumin or globulin be present, precipitation occurs. If the urine be not acidulated phosphates may separate out on heating, but this does not occur if the test be properly performed. If the urine be very alkaline serum albumin and globulin may have undergone a transformation into alkali albumin, which is not precipitated by heat. If the urine be highly acid the serum albumin may have been converted into acid albumin, which does not coagulate on heating. If the test be properly performed and a precipitate form, serum albumin or globulin must be present in the urine.

(2) **The Nitric Acid Test (Heller's).**—Half a drachm of strong nitric acid is placed in a test tube, and the suspected urine is slowly floated over this, or the urine may be placed in the tube and the acid gently poured down the side so as to lie below it. A cloud of coagulation immediately above the junction shows that some proteid is present. The acid coagulates both serum albumin and serum globulin as well as albumose. It does not coagulate peptones. If a resinous body is present in the urine, as after the ingestion of copaiba, styrax and turpentine, it causes a cloud at the line of junction, while uric acid and urates give rise to a zone of coagulation somewhat higher up in the tube. In concentrated urines this test may produce crystals of nitrate of urea, occurring in large scales above the contact line. Clouds formed by resins, uric acid, urates, and nitrate of urea are dissolved by heat, which does not cause the disappearance of the cloud produced by the proteids.

(3) **The Biuret Reaction.**—Half an inch of urine is mixed with an equal volume of strong caustic soda solution. On the addition of a drop or two of a very weak solution of sulphate

of copper a violet colour develops if albumin or globulin be present, or rose-pink with albumose or peptone.

(4) **The Picric Acid Test (Johnson's).**—A saturated solution of picric acid is floated upon the surface of the urine, and the tube is gently shaken in order to cause a slight mixture of the reagent with the urine. Cloudiness of the zone where the two fluids are mixed shows that one or other of the four proteids mentioned is present, unless the individual whose urine is under examination has been taking quinine or other alkaloid, which produces a substance coagulable by picric acid. In this test the precipitate caused by peptones and alkaloids dissolves on heating and reappears again on cooling.

(5) **The Xanthoproteic Reaction.**—Add nitric acid and boil; in the presence of a proteid body a yellow colour forms, which is turned orange by ammonia.

Other tests similar to these might be mentioned, but those which have just been described are the best of the kind in common use, and nothing need be said about the others.

Serum albumin and serum globulin are usually associated in any albuminous urine; they may be distinguished by the behaviour of the latter in neutral solution on the addition of magnesium sulphate. If the urine be rendered neutral or very faintly alkaline with liquor potassæ, and a saturated solution of magnesium sulphate be poured down the side of the test tube, a white ring appears at the line of junction of the two fluids. By saturating the neutralised urine with sulphate of magnesium, all the serum globulin may be precipitated, and after filtration and acidulation a precipitate of serum albumin may be obtained on boiling when it is present.

Albuminometry, or the **quantitative estimation of the important proteids,** has within the last few years been much simplified, and the old gravimetric process, as well as the titration method by means of perchloride of mercury and

iodide of potassium, has been entirely superseded for clinical purposes.

By means of *Esbach's process* the amount of proteids—which in most cases may be assumed to be composed of serum albumin and serum globulin—can be easily determined with considerable accuracy. The reagent employed is obtained by dissolving 10 grms. of picric acid and 20 grms. of citric acid in 800 or 900 c.c. of hot water, and, after solution, adding enough water to make 1000 c.c., or 1 litre when cold. It is employed in a special tube holding about 20 c.c., which is marked by a line and the letter "U," to indicate the quantity of urine to be used, and another line higher up with the letter "R," to show how much of the reagent is to be added to the urine. At the lower part of the tube are lines marked by the figures 1, 2, 3, 4, 5, 6 and 7, to indicate the result. The tube is provided with an indiarubber stopper.

The method of using the tube and reagent is as follows:—Fill the tube with the filtered urine as far as the letter "U," and add the reagent until the fluid reaches the letter "R." Close the tube by means of the stopper, gently turn it upside down twice without shaking, and put it aside for twenty-four hours; at the end of that period read off the height of the coagulation, as shown by the figures at the lower end of the tube. The figure represents the number of grammes of proteids contained in a litre of urine.

In using this method it is of importance to attend to two points. The urine must, first, be quite acid. If it should happen to be alkaline or neutral, acetic acid must be added until it turns litmus paper to a bright red tint. The results, secondly, are more exact when the quantity of proteids is small, and in the case of highly albuminous urines it is well to dilute them so as to double or quadruple their bulk, taking care when recording the number of grammes in a litre to correct the result. If the amount is above 0·6 per cent., or below 0·05 per cent., the result is not accurate.

In order to determine the relative amount of serum albumin and serum globulin in any urine, all that it is necessary to do is to estimate, by means of the Esbach process, the total quantity of proteids (which, as already mentioned, may be considered to be solely composed of serum albumin and serum globulin) in the urine, and to estimate the quantity in it after complete precipitation of the serum globulin by shaking the neutralised urine with powdered magnesium sulphate. The result of this second estimation gives the amount of serum albumin, and the difference between it and the total quantity of proteids is the amount of serum globulin. Six days must elapse before the tube is read off, since the high specific gravity of the magnesium sulphate solution delays the precipitation of the albumin. Tapping the side of the tube generally gets rid of air-bubbles and allows the precipitate to fall. It is a good rule to allow the tube to stand until the reading of the precipitate on two successive days is the same.

In making the calculation it must be remembered that some expansion has taken place in the original amount of urine from the addition of the magnesium sulphate. The urine and magnesium sulphate should be measured, and allowance made, or the reading of albumin will be too low.

Crystalline Globulin.—A form of globulin, precipitating spontaneously from the urine on standing, has been described. It occurs as a white precipitate, which when mixed with the supernatant urine gives it the appearance of watered silk. The crystals are of elongated rhombic form, lying singly or in rosettes. They vary greatly in size, but are much longer than tyrosin, and terminate in angular extremities.

Proteoses.—Primary and secondary albumoses are found in the urine under pathological conditions, but it is still doubtful if true peptone ever occurs. From a clinical standpoint it is scarcely necessary to differentiate the albumoses, but the reactions which are given by the different forms differ somewhat.

Albumoses are not precipitated from their watery solution by heat, but are precipitated by alcohol, by tannic acid and by metaphosphoric acid. They give the biuret and xantho-proteic reactions.

On the addition of picric acid to a urine containing albumose a cloud forms, which disappears on heating. The presence of the alkaloids of quinine, antipyrin and certain resinous acids also give this reaction.

Protoalbumose and heteroalbumose are precipitated on saturating the urine with sodium chloride; deutero-albumose is not precipitated by saturation with sodium chloride alone, but is precipitated by saturation in the presence of acetic acid.

Protoalbumose gives a precipitate with cold nitric acid, which diminishes or disappears on heating; the secondary albumose, deutero-albumose, is probably only precipitated in the presence of excess of salt.

Peptone gives the general proteid reactions, the biuret and the xanthoproteic reactions in a similar manner to the albumoses, but it is distinguished from the albumoses by not being precipitated on saturation of the solution with ammonium sulphate.

Nucleo-albumin.—A trace of nucleo-albumin is present in normal urine derived from the cells of the urinary tract. It is a phosphorus-containing proteid which forms a precipitate on the addition of acetic acid to the urine, and this more readily, if the proportion of salts in the urine be lowered by the addition of water or by dialysis. The precipitate formed with acetic acid is soluble in excess of acid. If nucleo-albumin be abundant, a precipitate is given with Heller's nitric acid test and the ferrocyanide test.

Mucin, a phosphorus-free mucoid, forms part of the mucus cloud of healthy urine. If the urine be alkaline, mucus remains in solution, and can be precipitated by the addition of acetic acid, the precipitate not being soluble in excess of acid. With the nitric acid a diffuse haze may form. It is

precipitable on saturating the urine with magnesium sulphate.

Histon, described as a product of the decomposition of nucleo-histon, a proteid contained in the leucocytes of the blood and lymphatic glands, has been found in the urine. It is now generally regarded as the proteid part of nucleo-proteid.

The Separation of the different Proteid Bodies.—The total urine of twenty-four hours is taken, and if serum albumin and globulin are present they are precipitated by heat, the urine being evaporated on the water bath down to about a litre. The urine is then filtered. The filtrate, which should be neutralised, is saturated with ammonium sulphate, and the precipitate removed by filtration. The filtrate is put aside to be tested for peptone. The precipitate, which should contain the albumoses, is dissolved in water. Divide the solution into two portions. The first is tested for nucleo-albumin. It is placed in a dialyser till the ammonium sulphate disappears. If hetero-albumose be present, it will have separated out, and the precipitate can be removed by filtration. If nucleo-albumin be present, it will give a precipitate with acetic acid, dissolving in excess or in the presence of a mineral acid.

The second portion is to be tested for albumoses. To a small quantity of the fluid add an equal volume of a saturated solution of common salt, and then acetic or nitric acid, so long as the precipitate which may form increases in volume. The mixture is then boiled, and if the precipitate passes entirely or partially into solution, so that when filtered hot the filtrate at first clear becomes cloudy, it may be concluded that albumoses are present. To determine which albumose, take the dialysed fluid and add acetic acid to precipitate the nucleo-albumin, and filter. The filtrate is next neutralised and saturated with rock salt. If a precipitate form it is primary albumose. If the fluid remains clear, add acetic acid which has been saturated with sodium chloride. If

deutero-albumose is present it is precipitated. The fluid which was originally put aside is next tested for peptone. Apply the xanthoproteic test, if the reaction be present the proteid is peptone.

The **clinical significance** of these proteids is still a matter of discussion. Serum albumin and serum globulin are almost invariably associated together, and it is almost beyond doubt that they pass out by way of the glomeruli. It is almost equally certain that if the epithelium of the glomeruli is intact no transudation of proteids can occur, and the presence of serum albumin and serum globulin in conditions of apparent health—such as we find in what is called "functional albuminuria," "cyclical albuminuria," or "the albuminuria of adolescence"—may be taken as evidence that on account of some transitory disturbance of the kidney, or the blood vessels, or the blood, there is a state of abnormal permeability of the glomerular epithelium. Albuminous substances are found in the urine as the result of febrile affections and toxic agents; venous stasis from heart and lung disease or pressure on the renal veins; temporary stoppage of a ureter; several chronic wasting diseases, such as anæmia, leukæmia, and tuberculosis; and certain diseases of the nervous centres, such as cerebral hæmorrhage. They are constant constituents of pus and blood, and are thus found in the urine when these bodies are present. In these affections there may be no definite recognisable lesions of the kidney. But the great cause of albuminuria is nephritis, in its various forms, and it is of the highest importance to be able to determine in any case by means of the whole of the symptoms whether there is evidence of renal disease or not. In lardaceous disease of the kidney albuminuria is a constant sign. In the class of cases termed "functional," the albumin may be present at one period of the day and absent at another; hence the utility of testing a mixed specimen of the urine of twenty-four hours, and of specimens of the urine passed at different hours.

Albumoses are found in the urine under different conditions. They are not normally present in the blood, when they occur they are excreted by the kidneys in the same way in which any other foreign substance is excreted. If there be a lesion of the alimentary canal, such as in ulceration of the intestine or gastric carcinoma, the albumoses, the products of proteid digestion, pass into the circulation and are excreted in the urine. Albumosuria is also common in fever, especially in the infectious fevers, in osteomalacia and carcinomatous affections of the bones. Albumoses are found along with serum albumin and globulin in nephritis. It is exceedingly doubtful if in these cases they can be considered as *per se* evidence of kidney disease. In some cases albumoses may result from the digestion of proteids in the bladder.

Nucleo-albumin may be detected in normal urine, if a sufficient quantity be used and be concentrated by evaporation. It occurs in considerable quantity in leucocythæmia, and in catarrh of the urinary passages. It has been found experimentally in dogs after compression of the renal artery, probably from destruction of the kidney epithelium, and after the intravenous injection of proteid. When serum globulin is present in the urine, and there is at the same time an excretion of fibrinogenic substance by the kidney or genito-urinary tract, *fibrin* is formed, and appears in the urine. Fibrin may be formed after the urine is passed, when it appears as a firm clot, or it is formed in the bladder and appears as shreds in the urine when passed. Such a condition is seen in chyluria, hæmaturia, infective nephritis and tuberculous disease of the bladder.

Blood Pigment in the urine gives it a tint varying in degree with the quantity which is present. Blood pigment present in the urine in the proportion of 1 in 2000 gives a smoky tint, and 1 in 500 produces a bright cherry colour. When blood is present the urine gives characteristic reactions with the tests for proteids,

as serum albumin and serum globulin are always to be detected.

The colour of the urine is in most cases sufficient to determine the presence of blood, but it may be proved by spectroscopic, microscopic, and chemical examination.

Spectroscopic Examination.—If much blood is present, a specimen of the urine placed in a narrow test tube and held in front of the slit of the spectroscope may obscure the whole field of the spectrum, but when diluted with a little water some of the red and orange appears, and when still more diluted the green begins to show itself, while between the orange and green a dark space is seen. If the position of this absorption band is compared with Frauenhofer's lines, it will be found to extend from beyond D towards the red side to a point beyond E towards the violet side of the spectrum. On further dilution this broad band is found to be composed of two smaller bands; one near D being narrower but more strongly marked; the other near E being broader but less definite at its edges. By adding more water the bands can be narrowed, and that near E disappears, leaving the other to be faintly seen; and still further dilution finally removes it also. Reducing agents added to the urine cause the disappearance of these two absorption bands, which are replaced by one broader but less defined band midway between D and E. The addition of ammonium sulphide to urine containing blood will effect this change.

In what is known as hæmoglobinuria the spectrum of methæmoglobin is usually present.

Microscopic Examination.—Blood corpuscles retain their natural size and form for a considerable time in urine which is slightly acid, but at last become irregular in outline and cease to form rouleaux. When the urine is alkaline the form speedily alters, and the colouring matter makes its escape.

Chemical Examination.—(1) The guaiacum test is the most generally used chemical test for blood in urine, but it

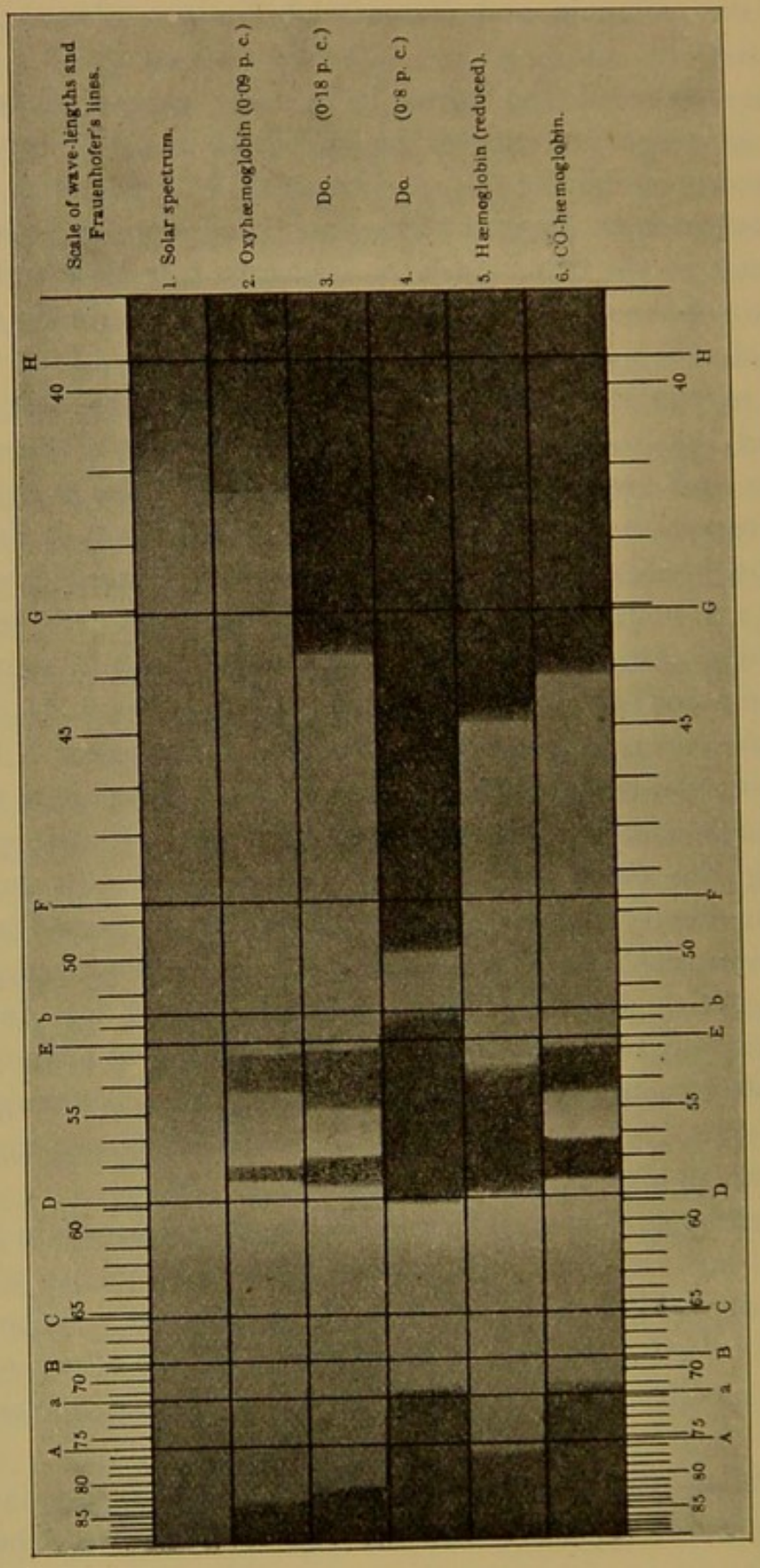


Fig. 104.—The spectrum of haemoglobin.

must be remembered that it is not conclusive. The reagents required are a freshly prepared tincture of guaiac, which must be made with rectified spirit, the ammoniated tincture of the Pharmacopœia being useless for the purpose, and ozonic ether, that is, an ethereal solution of hydrogen peroxide. The test is performed by placing 1 drm. of urine in a test tube; adding a drop of the guaiac tincture, thoroughly mixing them, and gently shaking with as much ozonic ether as will equal the quantity of urine. When blood is present a bright blue colour is seen in the ozonic ether when it separates, or it appears at the junction of the fluids. The test depends upon the transference by hæmoglobin of ozone from the ozonic ether to the guaiacum, which becomes oxidised and assumes a blue colour. This test is not to be relied on absolutely, as saliva and nasal mucus and pus produce the same blue ring, and the urine of patients taking iodide of potassium gives a similar reaction.

The following two tests are usually given for the recognition of blood in the urine, but are of little value.

(2) **Heller's Test.**—If the urine be rendered alkaline by the addition of a few drops of liquor potassæ, a flocculent precipitate of earthy phosphates forms on boiling. Normally this precipitate is white in colour, but if blood be present it assumes a beautiful blood-red colour. If small quantities of hæmoglobin be present, the colour reaction may not be visible till the precipitate sinks to the bottom of the test tube. The reaction depends upon the formation of hæmatin, which gives the colour to the earthy phosphates. If too little or too much alkali be added, or when heat has been too rapidly applied, the precipitate may be rust coloured. If the urine be originally alkaline, no precipitate may form; but if a calcium salt be added, say by the addition of a little normal urine, the reaction can be brought out.

(3) **Hæmin Test.** — The precipitate obtained by the Heller's test may be used. It is collected on a filter paper, washed with water, and dried. A small fragment is then

laid on a glass slide, a minute crystal of common salt added, a cover-glass applied, and the space between the two glasses filled up with glacial acetic acid. The preparation is then gently heated for a minute over a small flame till the fluid gives off minute bubbles, care being taken not to overheat. Any acid lost by evaporation is replaced by the occasional addition of a drop. In the presence of hæmatin the fluid becomes gradually brownish-red. Allow the preparation gradually to cool at about 45° C. till all the acetic acid has evaporated. This is easily done by removing the specimen a little distance from the flame. In the presence of blood in the urine the microscope shows the characteristic crystals of hæmatin, mahogany-brown rhombic crystals, which are more readily brought out by running glycerin under the cover-glass.

Hæmaturia may arise from a lesion of any portion of the urinary tract, and the appearance of the urine will vary with the site of the hæmorrhage. Blood arising from the walls of the urethra is not mixed with the urine, but appears as a small clot at the beginning of micturition. Blood from the prostatic veins usually finds its way back into the bladder, and may be mixed with the urine, or appear towards the end of micturition. When the bleeding is from the bladder, the first part of the urine passed is frequently unaltered, but towards the end of micturition the urine becomes deeply blood-stained and commonly contains clots. Clots in the urine are rare when the hæmorrhage is from the ureters or the pelvis of the kidney. If present, they are usually decolorised and moulded into an elongated shape. When blood comes from the kidney it is intimately mixed with the urine. If blood corpuscles are present, they are readily recognised under the microscope.

The colour of the urine varies much in hæmaturia, and the urine is never clear. Small quantities of blood give a "smoky" tinge, and from this the pigmentation varies up to a deep red or brown shade; with an equal quantity of

blood an acid urine will give a deeper tint than an alkaline. In hæmaturia albumin is always present. On standing the urine deposits a reddish or brownish sediment. When the pigment of the blood alone is present the condition is known as **hæmoglobinuria.** The pigment is, as a rule, methæmoglobin, and gives the urine a dark brown colour resembling porter. The spectrum shows four absorption bands, one well marked between C and D ; the other three, in the yellow, green, and blue being fainter.

Hæmaturia may be caused by renal affections, such as nephritis, pyelitis and cancer ; by inflammation, and simple and malignant neoplasms of the urinary channels ; and by morbid conditions of the blood, such as scorbutus and purpura, or the presence of the *Bilharzia hæmatobium.*

Hæmoglobinuria, in which the blood is disintegrated within the circulatory system, follows the absorption of certain toxic agents, such as chlorate of potassium, carbolic acid and pyrogallic acid ; it may be the result of insolation, syphilis and rheumatism. It is well exemplified in the disease termed "paroxysmal hæmoglobinuria," which may possibly be malarial in origin.

Hæmatoporphyrin, the iron free derivative of hæmatin, is normally present in the urine in very small quantities ; when in excess it gives the urine the colour of port wine. The spectroscope shows four absorption bands in alkaline solution and two in acid. Hæmatoporphyrin is found in the urine in excess in sulphonal poisoning, sometimes in Addison's disease, enteric fever, cancer, cirrhosis of the liver, lead poisoning and some other toxæmias.

Bile.—The constituents of bile, the bile pigments and bile acids, appear in the urine in jaundice, constituting the condition known as "choluria." Icteric urine is a yellowish green to a porter-like brown colour. On shaking, the froth is yellow. Any sediment which may be present is stained a vivid yellow colour.

Gmelin's Test for the Bile Pigments is performed by

placing a few drops of the urine to be examined on a white porcelain plate, and near it a few drops of fuming nitric acid. The two fluids are gently brought into contact, when the play of colours, and particularly the green tint, must be looked for. The best method of applying this test has been introduced by Rosenbach. Filter the urine through a small filter paper. If little urine be available pass it several times through the paper. With a glass rod place a drop of nitric acid on the paper; around the drop a beautiful play of colours forms if bile pigment is present. The play of colours results from the oxidation of biliverdin by the nitric acid, and is best brought out if the nitric acid contains some peroxide of nitrogen, as is seen when it has stood for a time and has acquired a yellow colour.

The **Bile acids** cannot be directly demonstrated in the urine. Pettenkofer's test with sugar and sulphuric acid is interfered with by the presence of substances which give a similar play of colours.

Oliver's Peptone Test depends on the precipitation of peptone by the bile salts in solution, or their derivative cholate of sodium. The test solution consists of—

Pulverised peptone	30 grs.
Salicylic acid	4 ,,
Acetic acid	30 drms.
Distilled water to	8 oz.

The solution is rendered perfectly transparent by filtration. The urine should be freed from all deposit by boiling and filtering, rendered faintly acid when necessary, and diluted to a specific gravity of 1008. Half an inch to an inch of the reagent is taken in a test tube and about twenty drops of the urine added. In the case of normal urine a slight haziness appears, but if bile acids be present a distinct cloud forms, which will disappear on the addition of excess of acid. The test is difficult and somewhat uncertain in its application. Bile acids cannot as a rule be demon-

strated without being first isolated, a proceeding not applicable for clinical work.

Glucose ($C_6H_{12}O_6$). — Healthy urine usually contains traces of glucose, but in so minute a quantity that it may be regarded as an abnormal substance. If the specific gravity of any urine is 1030 or more, it raises an expectation that glucose is present; and if the urine is at the same time pale in colour and excessive in quantity, the expectation is almost a certainty. It may be detected by means of several tests, all depending upon the fact that when glucose is boiled with caustic potash it is oxidised—taking oxygen from any source available, and hence causing reduction.

Moore's Test consists in the mixture of equal parts of urine and liquor potassæ in a test tube, the upper layer of which is to be boiled. If sugar is present, a ruddy brown colour is developed in the heated zone with the formation of glucic and melasic acids. If nitric acid be now added, the brown colour is discharged, and an odour of caramel is given off. This is by no means a delicate test, and is apt to be vitiated by impurities in the solution of potash.

Trommer's Test.—The urine is treated with one-fourth of its volume of liquor sodæ, and solution of copper sulphate is added drop by drop, the mixture being shaken after each addition, till a small quantity of copper hydroxide remains undissolved. If the urine dissolves much of the hydrated cupric oxide, and assumes a beautiful blue colour, the presence of sugar is probable. The mixture is now gently heated just to the boiling point, the heat being applied to the upper level of the fluid in the test tube. If precipitation of copper suboxide takes place, as evidenced by the development of orange-yellow streaks or clouds, the presence of sugar is evidenced.

If the test be not carried out in this manner, and with very great care, error is liable to occur. It must be remembered—(1) That normal urine contains substances which are

able to dissolve hydrated cupric oxide, giving the blue colour to the solution. Such substances are uric acid, kreatinin and the salts of ammonia. (2) That normal urine contains substances which reduce hydrated cupric oxide, withdrawing a portion of its oxygen and forming the red copper suboxide or hydrated yellow suboxide. Such substances are uric acid, glycuronic acid, pyrocatechin and hydrochinon. The copper suboxide is not, however, precipitated; the colour of the solution changes, but no precipitation takes place for some time, for *normal urine contains substances which keep the suboxide in solution*, *e.g.*, uric acid, kreatinin, ammonia salts.

The test is only reliable when in its application the temperature is not raised to the boiling point, and when distinct precipitation of suboxide occurs.

Fehling's Test.—This is simply a modification of Trommer's test, in which the cupric oxide is held in solution by an alkali and an organic compound. The reagent employed is composed of—

Cupric sulphate, 34·64 grms. dissolved in 500 c.c. distilled water.

Neutral potassium tartrate, 173 grms. dissolved in 500 c.c. solution of caustic soda (specific gravity 1·12).

These should be kept in separate bottles, and mixed when required in equal proportions.

The strength of this solution is so adapted that 10 c.c. are reduced by 0·05 grm. of glucose; and, as will be mentioned below, this renders the test of use in the quantitative estimation of sugar in the urine.

As tartaric acid is apt, if long kept, to be changed into racemic acid, which will reduce cupric to cuprous salts, the Fehling's solution must be boiled before being employed as a test.

In order that the test may not be complicated by the reducing substances of normal urine, the urine must be added, and the reaction take place at a temperature below

the boiling point. It is therefore best performed as follows:—Take half an inch of the solution and a little urine in different test tubes, boil both, then remove from the flame, and wait for twenty to thirty seconds for the fluids to cool, then add some urine to the copper solution. If sugar be present, reduction of the copper takes place. The copper-reducing substances of normal urine do not react in the presence of tartaric acid when the solutions are at a temperature below the boiling point, while grape sugar under those circumstances has an actively reducing power on copper sulphate.

Pavy's Test.—The solution in this case is prepared by dissolving 4·158 grms. of cupric sulphate in 200 c.c. of distilled water by heat; 20·4 grms. of sodium tartrate, and the same quantity of caustic potash, in 400 c.c. of distilled water; mix the two solutions gradually; when cold, add 300 c.c. of strong ammonia (specific gravity 0·88), and add enough water to make a litre.

The solution is to be used in the same way as Fehling's solution. It has the advantage of giving no precipitate, as the ammonia holds the reduced cuprous oxide in solution. Its disadvantage is the giving off of ammonia fumes. The complete reaction is indicated by the disappearance of the colour.

In all the tests based upon the reduction of copper there are possibilities of error due to the presence of certain substances. Serum albumin and serum globulin must be removed if present. All the bodies belonging to the uric acid group, along with kreatinin, mucin, allantoin, milk sugar, bile pigments, pyrocatechin, and other less important substances, must be taken into account. The administration of certain drugs, such as chloral, morphia and chloroform, results in the excretion of glycuronic acid, which has a copper-reducing power.

Tests not depending on the oxidation of glucose when boiled with an alkali.—(1) The **phenyl-hydrazin test** is

one of the most satisfactory for glucose. It depends on the power of phenyl-hydrazin to form with glucose an osazon, which has a characteristic crystalline appearance. Two to 3 inches of urine is taken in a test tube, five drops of acetic acid and ten drops of pure phenyl-hydrazin are added, and the whole placed in a water bath or in a beaker of boiling water for twenty minutes to half an hour. On heating, the phenyl-hydrazin passes into solution. After remaining in the water bath for half an hour the tube is allowed to cool, and crystals of phenyl-glucosazon deposit if glucose is present in the urine. The crystals are in the form of yellow needles, detached or arranged in clusters and sheaves (Fig. 105). The test is exceedingly accurate and delicate.

(2) The **fermentation test** depends upon the fact that if saccharine urine is fermented by yeast, alcohol and carbonic acid are formed ($C_6H_{12}O_6 = 2C_2H_6O + 2CO_2$). The alcohol remains in solution, and the carbonic acid is given off and can be collected. The simplest method of carrying out the observation and of establishing a check is as follows :—Take three curved tubes closed at the upper end of the *long* tube —the ureometer of Doremus and Thursfield does excellently for the purpose. A quantity of the urine to be examined is shaken up with a small portion of fresh yeast about the size of a pea and filled into tube No. I., all air being excluded. Tube No. II. is filled with normal urine to which yeast has been added. Tube No. III., with normal urine, a pinch of powdered glucose and yeast. The tubes are placed in an incubator or warm place for twelve hours. If the yeast is active after twelve hours' fermentation has taken place in No. III., carbonic acid has developed and passed up to the upper part of the tube. In No. II. little or no gas should have developed ; while if sugar be present in the suspected urine, carbonic acid has developed in tube No. I. For the sake of further control some caustic alkali may be introduced into tube No. I., when, if the gas be carbonic acid, it will

disappear. If the urine to be examined be faintly acid or alkaline, a few drops of a 10 per cent. tartaric acid solution should be added to diminish putrefactive processes, which are inimical to alcoholic fermentation. The test may also be applied by observing the specific gravity of the urine before and after treatment with the yeast. In the presence of sugar there is a loss of specific gravity as the result of fermentation. Roughly, each degree of specific gravity lost represents 0·22 grms. of glucose in 100 c.c. of urine, or 1 gr. per oz.

If a mere trace of sugar is present in the urine no carbonic acid will be given off on fermentation, for the fluid absorbs the carbon dioxide.

(3) By **polarimetry** the presence of sugar may be detected with great readiness, and the quantity present can be estimated. The method is based on the fact that glucose has the power of rotating polarised light towards the right, and the amount of deviation is an index to the amount present.

There are several different kinds of instruments—the name of which is the *saccharometer*—all of which are constructed on similar lines. The light passes through one prism called the polarising, and afterwards through another called the analysing prism. If these be so placed that the oblique ends of the two prisms are parallel, the polarised part of the ray of light—that which is allowed to pass through the first prism, while the other part of the ray is absorbed—passes through the analyser without obstacle. If the analyser is turned on its axis the light will become more and more obscured until an angle of 90° has been reached, at which point it will be quite shut out. If it is rotated beyond this angle the light will gradually return until 180° is reached, when it is allowed to pass freely. It again diminishes to 270°, and increases to 360°.

If the prisms are so arranged that the light passes without obstruction, it will be found that a column of urine containing glucose causes partial obstruction by rotating the

light towards the right, and that if the analysing prism is rotated the light will pass when a certain point has been reached. The rotatory power of glucose at 20° C. averages 52°·6. If the length of the column of the fluid containing glucose is known, and the angle of deviation is noted, the percentage of sugar can be calculated.

If highly coloured, the urine must previously be freed from colouring matter by the addition of a solution of acetate of lead and subsequent filtration, or by clarifying it by passage through animal charcoal. If the solution of lead acetate has been employed, one-tenth must be added to the result.

If the length of the tube containing the urine is 1 decimetre, and the amount of deviation is 8·69, then

$$100 : 52{\cdot}6 : : 8{\cdot}69 : 4{\cdot}57$$

the urine contains 4·57 per cent. of glucose. It must be remembered, however, that all instruments are not graduated alike.

The quantitative estimation of glucose may be conducted most accurately by means of the titration process with Fehling's solution, of which, as has been already said, 1 c.c. is equivalent to 0·05 grm. of sugar.

For this method a graduated burette and stand are required. The process is conducted in the following manner :—

Take 10 c.c. of Fehling's solution, dilute it with 40 c.c. of distilled water, and boil. Place 10 c.c. of urine diluted ten times with water in the burette, and from this drop ½ cm. into the hot Fehling's solution. A yellow or red precipitate will fall at once to the bottom. After it has subsided, add another ½ cm., and so on until all the blue colour has disappeared. The exact moment of its disappearance must be noted. If 10 c.c. of diluted urine are required to decolorise all the diluted Fehling's solution, which, as above stated, is equivalent to 0·05 grm. glucose, then, as the urine was diluted ten times, 10 c.c. of undiluted urine

contains 0·5 grm. of sugar. From this it is easy to calculate the percentage—

10 : 100 : : 0·5 : 5

The urine contains 5 per cent. of glucose. To calculate the glucose in grains per ounce, multiply the percentage obtained in this way by 4·375 and the resultant figure, in this case 21·875, represents grains of glucose per ounce of urine.

The clinical significance of sugar in the urine is extremely wide, but there are two great classes of cases in which it appears. It may be present as a temporary constituent, caused by a surplus of carbohydrates in the system, or by some transient disturbance of the hepatic vasomotor apparatus; or it may be permanently present in cases of profound disturbance of the metabolic processes, as seen in diabetes mellitus.

Lactose, or **milk sugar,** may be found in the urine of nursing-women during the first days of lactation. It passes into the circulation from the mammary gland, and is excreted by the urine. Milk sugar in the urine reacts to Fehling's, Trommer's, and Bötger's test, but the reaction is not so rapid or energetic as in the case of glucose. It does not *readily* form an osazone with phenyl-hydrazin. When exposed to the influence of yeast it is slowly inverted into dextrose and galactose, and then undergoes fermentation.

Levulose, or fruit sugar, may occur in the urine in conjunction with glucose. It gives all the principal chemical reactions, including the formation of an osazone with phenyl-hydrazine. It is, however, levo-rotatory; when the urine is examined with the polarimeter, the polarised light is rotated to the left.

In certain conditions the peculiar odour of **Acetone** (C_3H_6O) may be detected in the urine. The substance may be recognised by several tests, but that which is most available is the nitro-prusside of sodium reaction. On the

addition of a solution (0·1 grm. to 15 c.c. of water) of nitro-prusside of sodium, along with caustic potash or caustic soda, to urine containing acetone, a ruby-red tint is produced, which slowly fades into a straw colour. The same colour is produced by these reagents in the presence of ethyl-diacetic acid ($C_6H_{10}O_3$), aldehyde, and kreatinin. In the case of the two last mentioned, however, the ruby-red rapidly becomes a straw-yellow, and on the subsequent addition of an acid no change occurs; while with acetone the addition of an acid produces a violet colour, and with ethyl-diacetic acid it gives a dark tint. If the solution of sodium nitro-prusside be employed along with nitric acid, a rose-violet colour is slowly given by acetone, and a ruby-red, slowly fading into straw-yellow, by ethyl-diacetic acid; no change occurs with aldehyde or kreatinin.

Lieben's test depends upon the formation of iodoform. The urine is acidulated and distilled. Five c.c. of the distillate is taken in a test tube, and five drops of a 10 per cent. solution of caustic soda added, and the whole warmed. Next add, drop by drop, a saturated solution of iodine in iodide of potash solution till a permanent yellow tint is obtained. More caustic soda solution is then added till the yellow colour is discharged, and if acetone be present a yellow precipitate of iodoform occurs. It must be remembered that alcohol will give this reaction; thus, in urine which contains sugar a fallacy may result by glucose being converted by fermentation into alcohol and carbonic acid.

The clinical significance of acetone in the urine is somewhat obscure. It probably owes its existence to excessive proteid metabolism. It occurs in the grave form of diabetes mellitus, and to it has been ascribed, though on doubtful evidence, a causal influence in diabetic coma. It occurs also in acute diseases such as pneumonia, in cancer, in nephritis, and in fasting and digestive disturbances. It may be found in the urine in health.

It may be added that in the case of Cetti, who starved in

Berlin for fourteen days, acetone was largely present in the urine, and Senator considers its presence an inanition symptom indicative of grave disturbance of the metabolism.

Along with acetone must be considered **Diacetic acid** or **Aceto-acetic acid** ($CH_3COCH_2CO_2H$)—acetic acid, where an atom of hydrogen has been replaced by the radicle acetyl, CH_3CO.

Its presence in urine may be demonstrated by the ferric chloride test. To 10 c.c. of urine add a drop or two of a weak solution of ferric chloride till no further precipitate of phosphate of iron occurs. Filter and add a few drops more of the iron solution. If diacetic acid be present a burgundy-red colour appears. This reaction is given, however, with salicylates, antipyrin, etc. Diacetic acid, however, is volatile and can be driven off by heat, and thus if it be the cause of the colour reaction it will not appear if a second sample of the urine be boiled for a time before adding the iron. Again, diacetic acid may be extracted with ether after acidulating the urine with sulphuric acid; and the test applied to the ethereal extract.

Oxy-butyric acid occurs along with acetone and diacetic acid in the urine in grave cases of diabetes. It is a levo-rotatory body.

The exact clinical significance of these bodies is not yet decided. They are found in the urine and also in the blood of diabetics, and are frequently associated with the onset of diabetic coma.

Microscopic Examination of Urinary Deposit

If the urine on standing shows a considerable deposit at the foot of the glass a portion of this can readily be taken up with a pipette, run on to a slide, a cover-slip placed in position, and the preparation examined under the microscope. If the deposit be small in amount it is necessary to centrifuge the urine to obtain a specimen for examination. When it

is necessary to examine the urine before any deposit has separated, the centrifuge is indispensable. No examination of the urine for tube casts, tubercle bacilli, etc., can, when negative, be considered complete unless the centrifuge be used. Deposits may be unorganised or organised.

Unorganised Sediments of the Urine consist of the various crystalline and amorphous forms of salts.

Uric acid occurs in acid urine. It is found in rhombic crystals of various forms (Figs. 102, 103). Lozenge and oval-shaped plates and spikes or rods are the most common forms; these are usually grouped together to form sheaf-like or fan-shaped bodies, along with stellate or rose-formed collections. The crystals have, as a rule, the characteristic reddish-brown hue of the pigment uroerythrin. The deposit gives the murexide reaction. When the deposit occurs in irregular crystals with pointed processes it is said to indicate rapid precipitation, and a consequent tendency to calculus formation.

Urates occur in both acid and alkaline urine.

Urates in acid urine form a clay-yellow to bright red powder often adhering to the glass, acid salts of uric acid, most commonly sodium urate, but also the urates of potassium and calcium. Under the microscope the deposit is seen to consist of amorphous granules in irregular heaps or moss-like branching rows. The deposit is readily soluble on heating, and on treating with acetic acid.

Crystalline deposits of urates may occur in both acid and alkaline urine. In acid urine crystalline urate of soda occurs as a greyish or yellowish deposit, which under the microscope shows crystals of irregular outlines with projecting spines; the deposit is uncommon.

In alkaline urine, urate of ammonium occurs as the sediment of alkaline fermentation. The crystals are large globules or aggregates of globules, mostly dark yellow in colour, and often coated with minute pointed crystals (hedgehog crystals). The crystals are readily soluble in acetic acid.

Fig. 105.—Phenyl-glucosazon.

Fig. 106.—Urate of sodium.

Oxalates. — Calcium oxalate occurs as a sparse light deposit ; most frequently the crystals are entangled as glistening specks in the mucus cloud. The form of the crystals is usually octahedral—two regular four-sided prisms placed base to base—the envelope form. More rarely the crystals are found as discs constricted across the short axis to form a dumb-bell like figure. The crystals are found, as a rule, in acid urine. They are unaffected by acetic acid or weak alkalies, but dissolve in hydrochloric acid.

Phosphates deposit in the urine as a white flocculent precipitate. Various phosphatic salts are found. Earthy phosphates, tri-calcium and tri-magnesium phosphate (basic phosphate of lime and magnesia), occur in alkaline urine of a fixed alkalinity as small amorphous granules of indeterminate shape or with a fine globular form. The salts are soluble in weak mineral acid or acetic acid.

Ammonio-magnesium phosphate, Triple phosphate, is found as an abundant white precipitate in urines which have undergone alkaline fermentation. The crystals are in the form of large prisms with oblique ends, the "knife rest" or "coffin-lid" crystals. More rarely the salts deposit in quadripartite feathery forms like the frost flower. The deposit may occur in neutral or amphoteric urine, but is rare.

Neutral calcium phosphate is rare, but may occur in faintly acid or amphoteric urines as wedge-shaped crystals agglomerated in bundles or rosettes.

Crystalline tri-magnesium phosphate, a rare deposit, is found as large, smooth, highly refractile, rhombic crystals, in faintly acid or alkaline urines.

Calcium carbonate is a rare deposit found in the urine after a vegetable diet, the urine having a fixed alkaline reaction. The salt is amorphous or crystalline, in globules or masses of globules or dumb-bells. In human urine when present it is usually amorphous. On treatment with an acid it dissolves readily with the effervescence of carbonic

acid gas. It is a common constituent of the urine of herbivorous animals, such as the horse, when it is crystalline in spheres marked with radiating lines.

Calcium sulphate is found as long prisms or elongated tables, mostly cut off abruptly at the end, occurring either singly or in packets. It has no clinical significance.

Hippuric acid, though always present in the urine, rarely occurs as a deposit. If present as a crystalline salt it takes the form of long rhomboidal prisms.

Leucin and **tyrosin**, when present in the urine, are usually found together. If present in the urine in considerable proportion, the evaporation of a drop is sufficient to give evidence of their presence, but they are usually present in such small amount as to require separation by artificial means.

Leucin may be obtained by evaporating the urine to dryness and dissolving the residue in boiling alcohol. When cold the solution will deposit leucin in the form of a greasy mass. Examined under the microscope, leucin is found to be composed of spherical bodies, with concentric markings interrupting a radiated structure.

Tyrosin may be obtained from the urine by precipitating the colouring matters with basic acetate of lead, filtering the mixture, and decomposing the filtrate by means of sulphuretted hydrogen. On evaporation tyrosin separates out. It presents the appearance of long acicular prisms, sometimes separate, at other times combined as sheaves and spherical bodies.

The clinical significance of leucin and tyrosin is very precise. Their presence is characteristic of destruction of the hepatic cells, and they are most commonly to be found in the rapid disintegration of the liver of acute yellow atrophy, phosphorus poisoning, and some malignant forms of fever, as well as various hepatic disorders.

Cystin is an uncommon urinary deposit, and forms a rare constituent of urinary calculi. If it is present in urine it

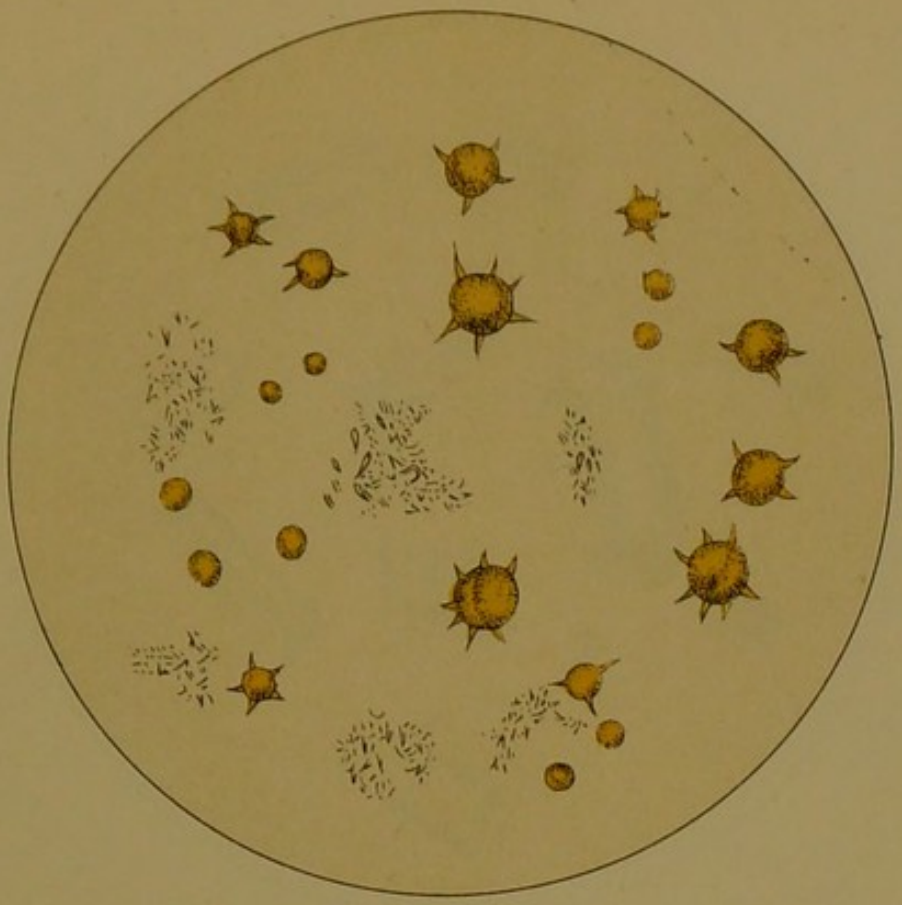


FIG. 107.—Urate of ammonium.

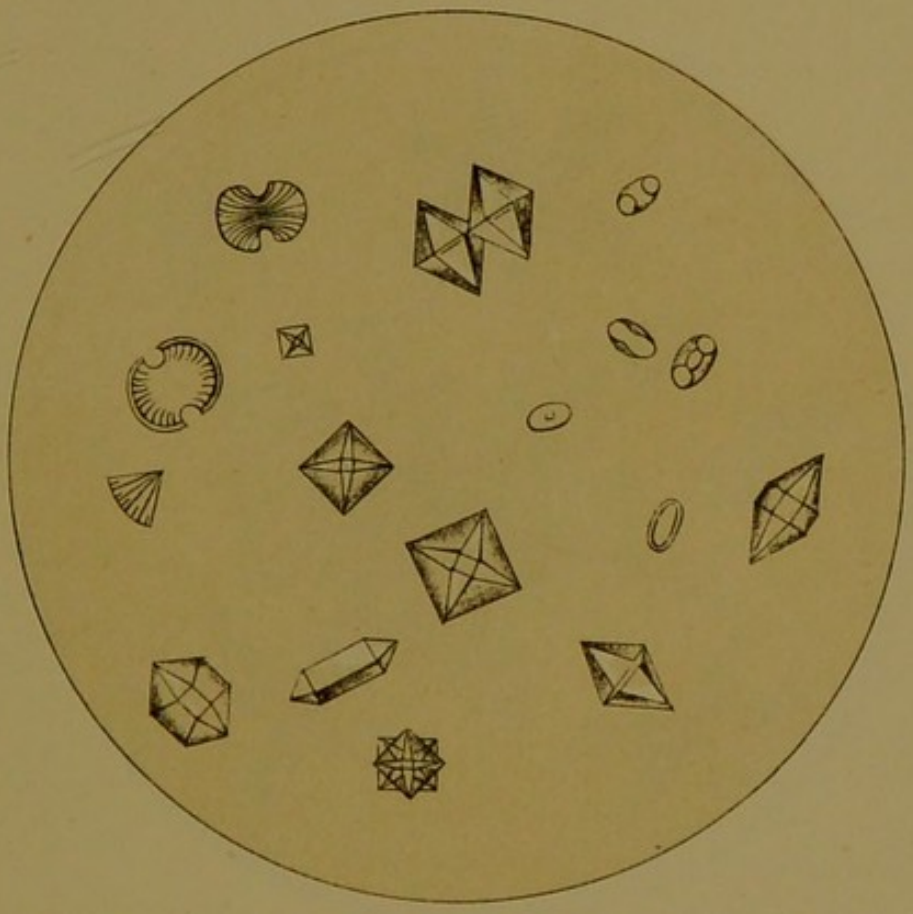


FIG. 108.—Calcium oxalate.

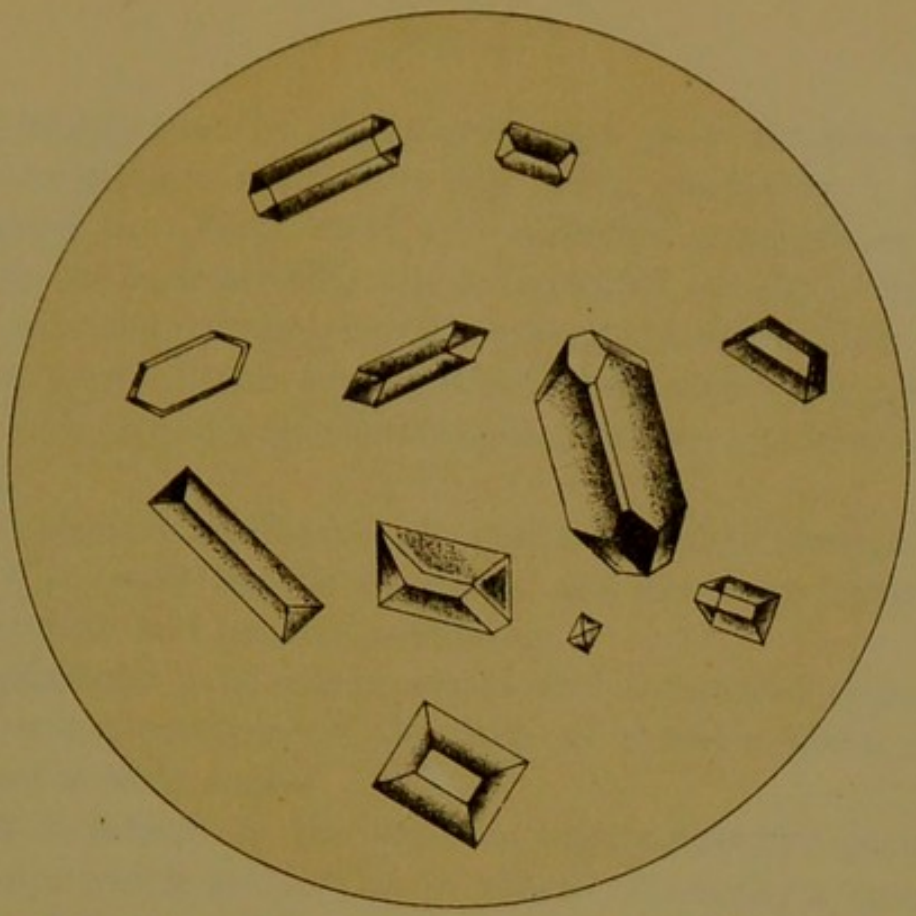


FIG. 109.—Ammonio-magnesium phosphate (prismatic form).

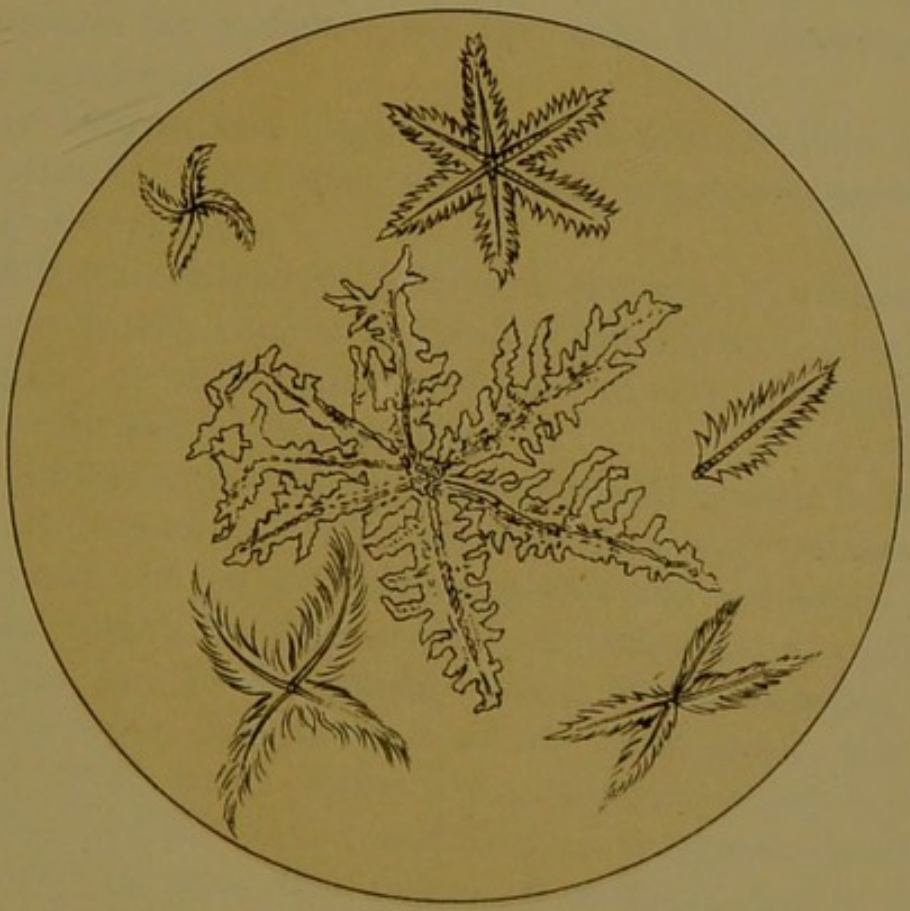


FIG. 110.—Ammonio-magnesium phosphate (feathery crystals).

may be separated by adding acetic acid in excess and allowing the urine to stand for a day. The precipitate is to be collected on a filter, dried, and dissolved in ammonia, from which cystin, if present, will be deposited on evaporation.

Under the microscope cystin appears usually in the form of hexagonal plates superimposed upon each other, but it is occasionally seen as rhombohedral prisms, scattered or in groups.

The clinical significance of cystin is rather obscure. It occurs in some hepatic disorders, and has been found in tuberculous and anæmic conditions.

Cholesterin is a monatomic alcohol sometimes deposited from urine, occurring in the form of rhombic plates with notches at their angles.

Fat is present in the urine as highly refractile globular droplets, which are soluble in ether. Normally a trace of fat can be separated from the urine. In lipuria it becomes a visible constituent, as in fatty degeneration of the kidneys. In chyluria fat globules give the urine the characteristic milky appearance.

The Organised Deposits of the urine include blood corpuscles, epithelial cells, tube casts, spermatozoa, parasites, and micro-organisms.

Red blood corpuscles are found in the urine in hæmaturia. They are usually swollen and pale, especially so if the urine is alkaline or very dilute; when the urine is concentrated, they shrink and become crenated.

Leucocytes in small numbers are normally present in urine, in the mucous cloud. They are present in large numbers as a constituent of pus in cystitis, pyelitis and urethritis. In acid or neutral urine they retain their shape for a considerable time, but in the alkaline urine of fermentation the ammonia causes them to swell up and disintegrate, forming a slimy mass, which under the microscope shows merely débris and nuclei. This transformation is taken advantage of as a test for pus. The deposit is removed, and to it is

added a piece of caustic potash. The corpuscles become disintegrated, and a tough, slimy mass forms. Pus corpuscles give the glycogenic reaction—that is, on treatment with a solution of iodine and iodide of potash they strike a mahogany-brown tinge. Urine that contains pus always contains albumin.

The **Epithelial cells** found in the mucus cloud present different characters according to their origin.

Mucus cells are small round or oval bodies, about the size of a white blood corpuscle. They swell up on the addition of water or dilute acetic acid, which render the nuclei distinct.

Squamous epithelial cells (Fig. 113) are derived from the bladder or vagina. They are large in size and irregularly circular in outline, presenting distinct nuclei. Cells from the vagina are larger than those thrown off by the bladder, but it is often very difficult to distinguish the one from the other.

Columnar epithelial cells have their origin in the urethra, ureter, or the pelvis of the kidney. They are irregular in shape, presenting cylindrical, pyramidal, or fusiform outlines. Their form gives no indication of their origin, which can only be ascertained by the accompanying symptoms (Fig. 114).

Round epithelial cells with distinct nuclei are of renal origin, and result from some change in the tubules of the kidney (Fig. 115). They will be referred to again in dealing with tube casts.

Renal tube casts are connected with the presence of albuminuria. Their presence does not of itself prove an anatomical lesion of the kidneys. Their value as a clinical sign depends upon the recognition of the varieties and their mode of formation. **Hyaline casts** and the so-called **cylindroids** are colourless, diaphanous, homogeneous, sometimes slightly striated and dotted, from the presence of detritus or fat. They are soft, elastic, narrow formations of varying length, which result from the coagulation of albumin which

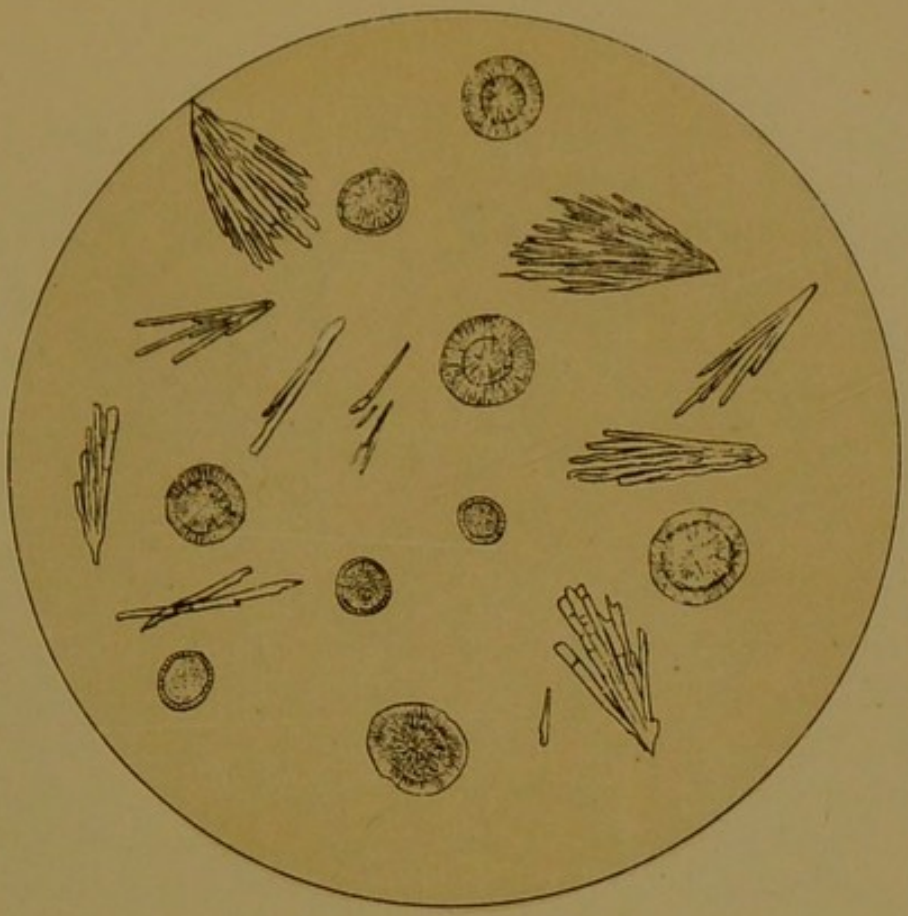


FIG. 111.—Leucin and tyrosin.

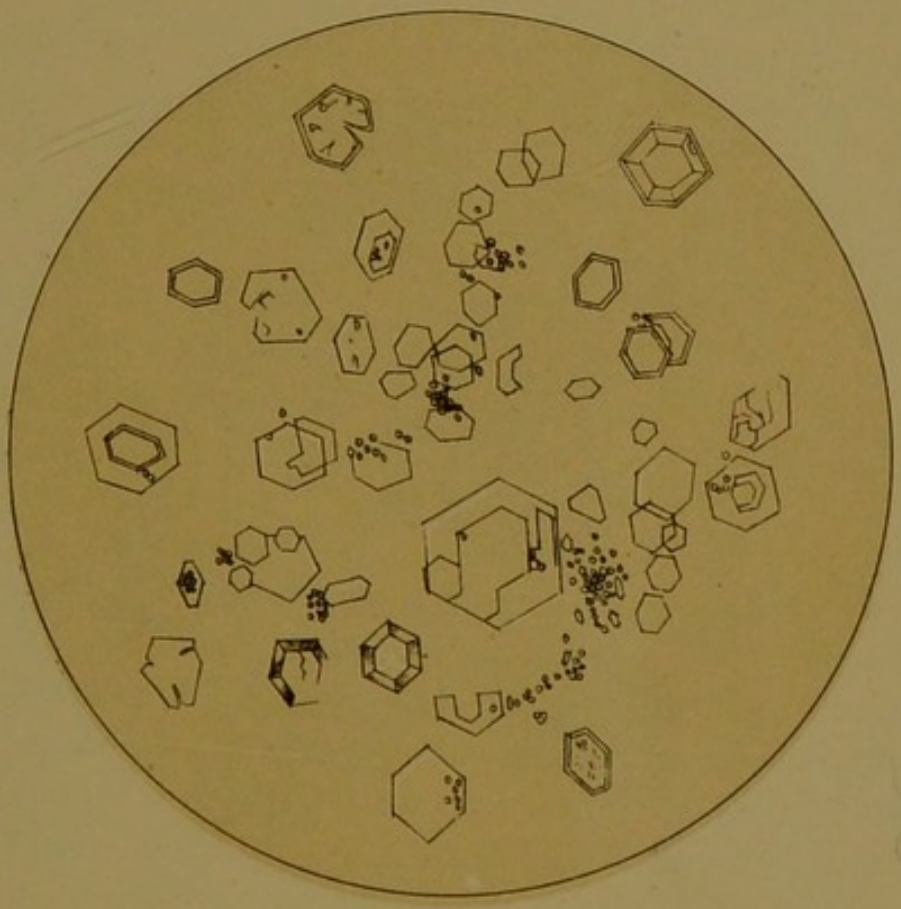


FIG. 112.—Cystin.

has passed out of the glomeruli into the urinary tubules. With bright illumination of the field of the microscope they are difficult to distinguish, but they can be easily stained, and are well brought out by bile pigments if the urine be icteric. **Epithelial casts** result from the disintegration of the epithelium of the urinary tubules. The cast consists of epithelial cells bound together with a basis of hyaline substance.

Granular casts are degenerated epithelial casts. In them epithelial cells are found in various stages of degeneration, from marked cloudy swelling to fatty degeneration and disintegration. The casts are thus dark, opaque, granular and often stained a brown colour, from the adherence of pigment. If a further metamorphosis of the epithelial cast takes place, it becomes the **hard hyaline** or **waxy hyaline cast**, a compact, highly refractive cast of a yellow colour, which is easily distinguished from the soft hyaline cast previously mentioned.

Blood casts result from hæmorrhage into the urinary tubules. They are formed of red blood corpuscles and leucocytes embedded in fibrin. The presence of hyaline casts in the urine is not necessarily indicative of nephritis. Epithelial casts show a desquamative inflammation of the tubules, and their state as regards degeneration, size, etc., will give information which has important bearing on the prognosis of a case of nephritis.

Spermatozoa are found at times in the urine. From their characteristic appearance their recognition is easy, on microscopic examination.

Urethral filaments occur in the form of small thread-like bodies visible to the naked eye. They are found in chronic prostatitis, a condition common after chronic gonorrhœa. Examined microscopically, they are seen to be very much larger than tube casts, and to consist of mucus and a few entangled leucocytes.

In tumour of the bladder, especially if the tumour be of a villous form, **fragments of the new growth** may be found in

the urine. The fragments consist of a ground-work of connective tissue, with vessels, and covered with epithelial cells if the growth be papillomatous, but if the growth be of a carcinomatous nature the débris is not so characteristic.

In ulceration of the bladder wall elastic fibres may be present in the urine.

Amongst the **parasites** which may be found in the urine are the embryo form of *Bilharzia hæmatobium*, *Filaria sanguinis hominis*, the fragments and hooklets of echinococci, and ascarides. These were described under the section on Parasites. Infusoria are found in the urine when it has been allowed to stand for some time, but they have no clinical significance. The micro-organisms of the urine will be considered under Clinical Bacteriology.

Accidental Constituents of the Urine.—Drugs, and the products of the administration of drugs, are found in the urine, and it is necessary, at times, to recognise the foreign substance.

The salts of **iodine** are rapidly excreted by the urine after exhibition. Very small quantities of iodine are recognisable in the urine. Add a few drops of starch solution to the urine, and float on to the surface of some concentrated yellowish nitric acid. In the presence of iodine a deep blue ring appears at the point of contact, which disappears on standing. A somewhat less delicate test is as follows. Treat the urine with a few drops of strong nitric acid, and then shake up gently with chloroform. The chloroform sinks to the bottom on standing, and in the presence of iodine shows a violet tinge.

Bromine, when exhibited in its salts of the alkalies, can be demonstrated in the urine. The urine is treated with chlorine water, and then shaken up with chloroform; in the presence of bromine a yellow colour develops.

Salicylates.—Salicylic acid and salol are freely excreted as salicyluric and salicylic acid. To recognise their presence, treat the urine with a few drops of ferric chloride solution,

FIG. 115.—Columnar epithelium from urethra.

FIG. 116.—Round epithelium from kidney.

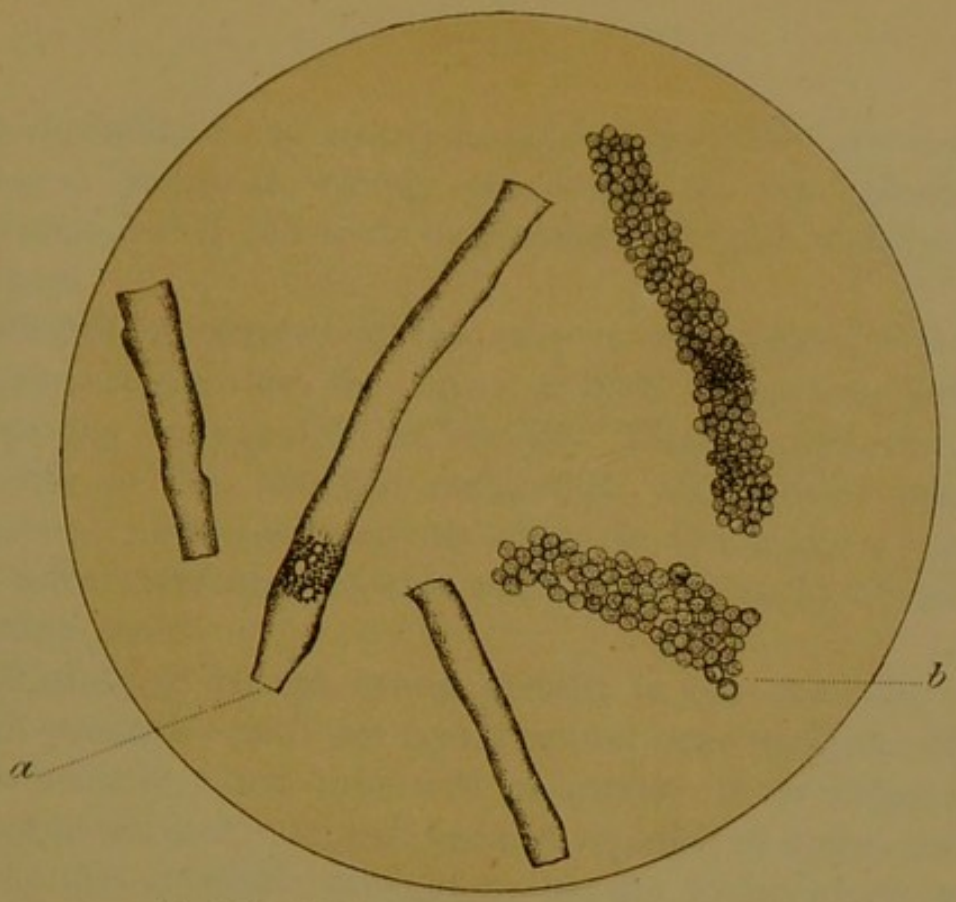


Fig. 117.—Hyaline (*a*) and blood (*b*) tube-casts.

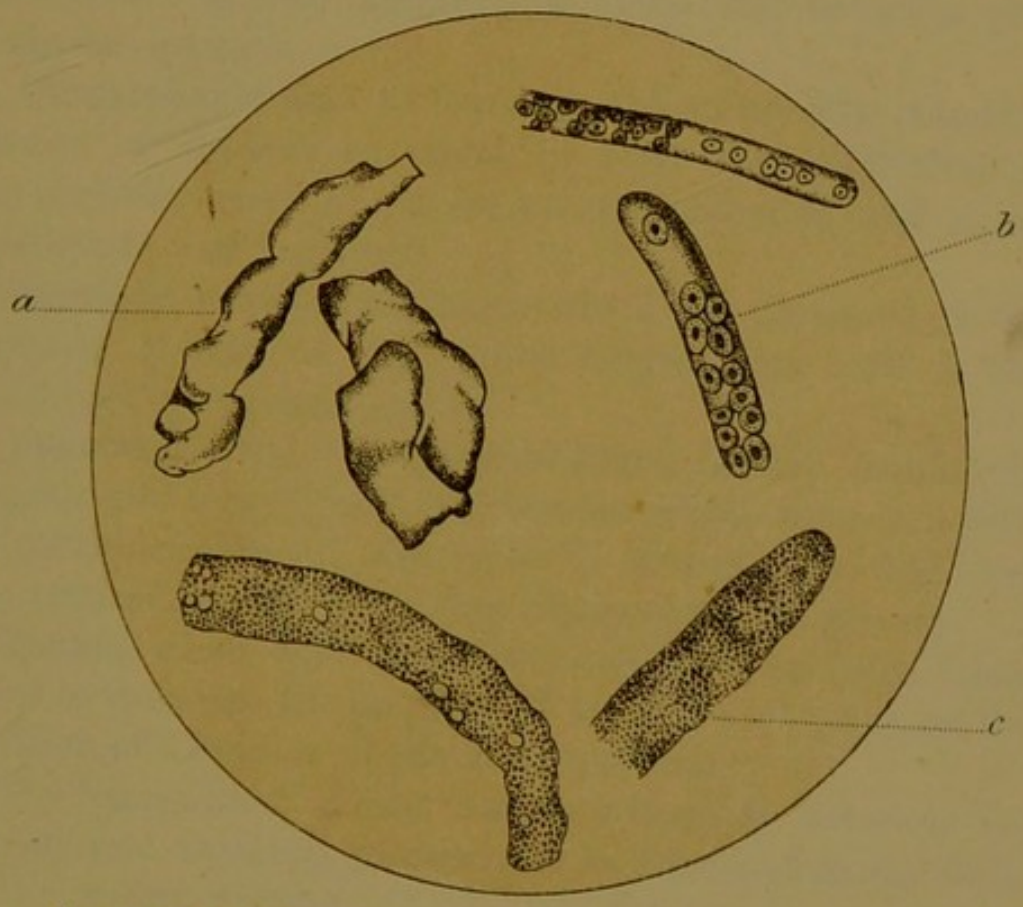


Fig. 118.—Hard hyaline (*a*), epithelial (*b*), and granular (*c*) tube-casts.

which should be as nearly neutral as possible. In normal urine a yellowish, cloudy precipitate of iron phosphate develops, but if the acids be present an intense violet colour appears.

Phenol is excreted in the urine in combination with sulphuric acid, giving the urine a dark olive-green colour, deepening on exposure to the air. Phenol gives the perchloride of iron test just mentioned. If Millon's reagent (mercuric nitrate solution) be added to the urine, a purple-red colour develops, but this reaction is given by a number of other bodies.

Chlorate of potash, when present in the urine in very small quantities (0·01 per cent.), can be demonstrated. Heat some urine in a test tube with a quarter of its volume of hydrochloric acid. It first becomes reddish or violet, owing to the decomposition of indican by the hydrochloric acid, and then, in the presence of chloric acid, yellow or quite colourless. It must, however, be noted that bromides give a similar reaction.

Chrysophanic acid, as found in the urine after administration of rhubarb or senna, or after the application of chrysarobin, gives a red colour with alkalies, and a red precipitate with lime salts.

Santonin yields a substance which also turns red on the addition of alkalies, but unlike chrysophanic acid is not destroyed by reducing agents.

Copaiba.—After the administration of the balsam of copaiba the urine yields a substance which reduces copper in alkaline solution. The urine is levo-rotatory, and does not reduce bismuth, being thus distinctive from glucose.

Tannin, when used as a medicinal agent in large doses, gives to the urine the property of turning dark green on the addition of a solution of perchloride of iron.

The recognition of the various **alkaloids** demands too complicated chemical processes to be discussed in the scope of the present volume.

CHAPTER X

THE NERVOUS SYSTEM

For obvious reasons, the investigation of the nervous system is concerned more with the observation of functions than with the examination of organs, and, as a consequence, the

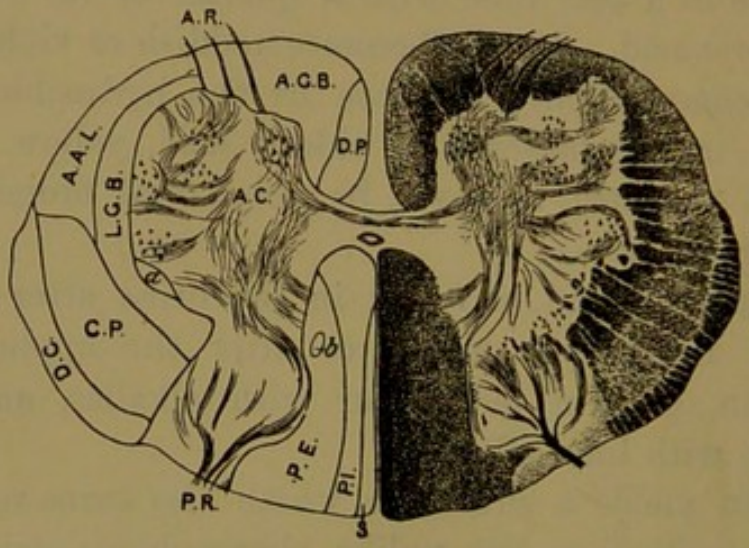


Fig. 119.—Transverse section of spinal cord. Cervical region.

A.R., Anterior root; D.P., direct pyramidal tract (Fürch); A.G.B., anterior ground bundle; A.A.L., ascending antero-lateral tract (Gowers); L.G.B., lateral ground bundle; C.P., crossed pyramidal tract; D.C., direct cerebellar tract; A.C., anterior cornu; P.R., posterior root; P.E., postero-external column, Funiculus cuneatus (Burdach); P.I., postero-internal column, Funiculus gracilis (Goll); S., septo-marginal tract (Bruce and Muir); *a*, descending fibres of fillet in lateral ground bundle; *b*, Schultze's descending tract in postero-external tract.

diagnosis of nervous disorders is beset with much greater difficulty than is the case with any other group of diseases. A thorough grasp of the subject can only be obtained by acquaintance with the facts of medical anatomy and physio-

logy, and their application to the symptoms in any given case. In the consideration, therefore, of the different symptoms and signs on which the investigation of the nervous system depends, it is necessary to sketch the anatomical and physiological facts upon which these phenomena depend.

I. SENSORY FUNCTIONS

Anatomy.—The path of conduction of the impulses of ordinary sensation, from their termination in the different sensory end-organs to the centres, is by the mixed nerves which enter the spinal cord by the posterior sensory roots. These roots, on their entrance into the cord, divide into two bundles, the smaller passing to the substance of Roland, and the larger to the ascending columns. Collaterals are given off to the cells of the grey matter on the same side, which subserve reflex action; and through the posterior commissure to the cells of the grey matter of the opposite side. Sensory impressions pass up the cord in the postero-median columns, the direct or posterior cerebellar tract and the antero-lateral ascending tract, or anterior-cerebellar tract. Two main courses are open for the impulses to the higher centres,—by the posterior column nuclei and tract of the fillet, and by way of the cerebellum.

In the first instance, the impulses pass up the posterior columns, to the cells of the nucleus gracilis and nucleus cuneatus in the medulla. These cells, acting as intermediary stations, pass on the impressions by the external arcuate fibres, which cross to the inter-olivary layer on the opposite side of the medulla forming the sensory decussation. The path is thence upwards in the pons, and by the crus as the fillet, lateral and median. The lateral fillet ends in the corpora quadrigemina; the median, reinforced by fibres from the sensory nuclei of the bulb, ends partly in the corpora quadrigemina, and for the most part in the optic thalamus.

From the thalamus the impressions are sent to the frontal and parietal cortex by way of the anterior end of the internal capsule, and through the posterior third of the hind-limb of the internal capsule to the temporal and occipital lobes.

The cerebellar path includes the fibres which pass by the anterior and the direct cerebellar tracts. These fibres pass up, to end in the superior vermis of the cerebellum. From thence the impressions pass upwards in the superior cerebellar peduncles to the corpora quadrigemina. There they decussate to pass to the cortical convolutions.

Investigation of the Sensory Functions

Ordinary Sensibility.—Many subjective sensations are associated with a disturbance of the nerves of ordinary sensibility. The most important is, perhaps, pain, which is, however, not simply to be regarded as an exaggeration of common sensation, but is probably a distinct sense. *Paræsthesia*, or perverted sensations, are frequent. There may be feelings of tightness, weight, sinking, heat, cold, numbness, itching, creeping, tingling, pinching, or throbbing. They vary greatly in degree, and several may be associated. Giddiness, or vertigo, is a complicated form of paræsthesia, which may arise from an infinite variety of causes, such as ear disease, cerebellar disease, dyspepsia, heart disease, or mental affection.

Of more importance than the subjective sensations are the results obtained by **testing the sensibility of the sensory nerves.** In testing this sensibility, it must always be remembered that the results obtained, while depending on the activity of the sensory nerves, must also, to a certain extent, be influenced by the intelligence and appreciation of the patient. All observations must be carried out with the patient's eyes covered, to eliminate the sense of vision; blank control experiments should be used to test the patient's statements.

The cutaneous sensibility includes the sense of pressure, the sense of locality, the sense of temperature, and the sense of pain.

By the **Tactile or Pressure Sense** an idea is obtained of the shape, consistency, and size of the body in contact with the skin. To obtain results, cover the patient's eyes and gently touch the skin with the point of the finger or with a feather. If the sensibility is normal the patient will

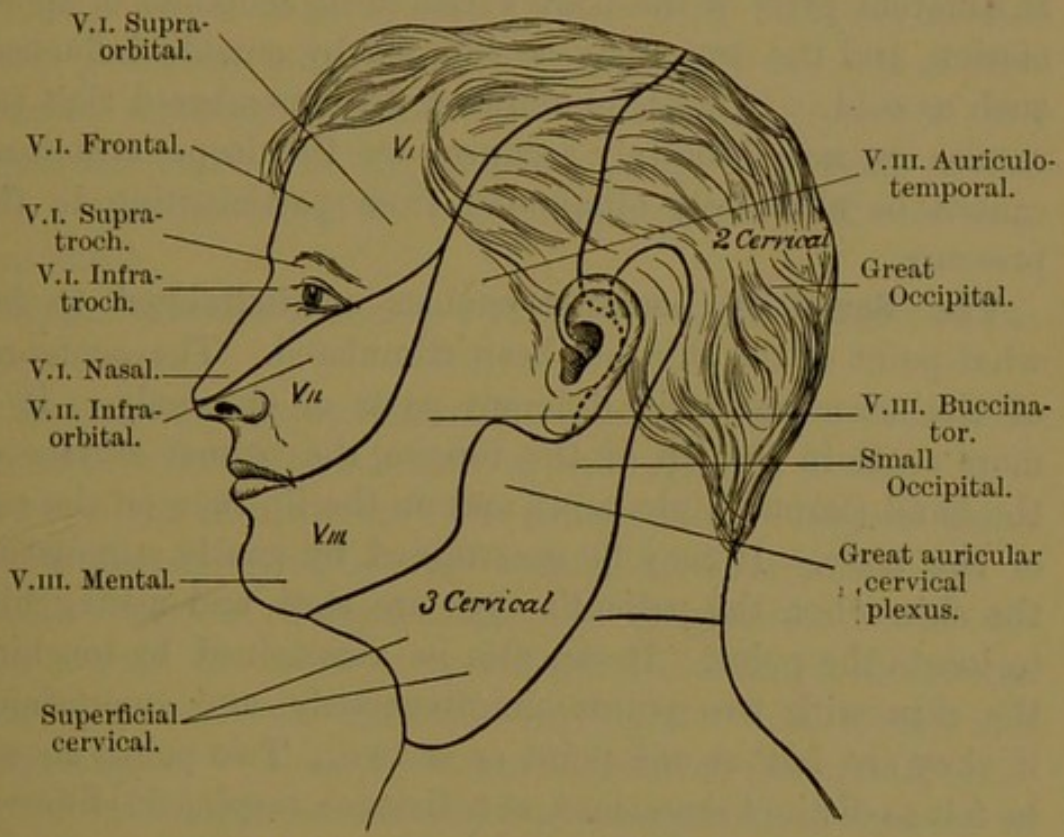


FIG. 120.—Cutaneous distribution of the sensory nerves of the head, the first, second, and third divisions of the fifth nerve, and the cervical plexus.

be able to tell when he feels the touch, and the character of the body which has come in contact with the skin. Control the experiments by blank observations. Next ascertain the patient's ability to appreciate different pressures. This may be done by placing on the same portion of skin, which it is desired to examine, discs of a similar size, but of different weights. In making the observation it is necessary to ex-

clude the muscular sense by supporting the limb on a firm surface; the influence of the temperature sense should also be excluded. Eulenberg has devised an instrument, the Baræsthesiometer, which meets these requirements, and by which the observation can be rapidly taken. Weber finds that the limit of discrimination for tactile stimuli from the volar skin of the ungual phalanx was reached, when the weights were related to each other as 29 to 30. The skin in different parts of the body varies in its acuteness of appreciation, and the sense may be blunted by outside influences such as cold. It must, in addition, be remembered that the results are not absolutely accurate, for the deeper structures cannot be absolutely eliminated from participating in the pressure.

The Sense of Locality enables an individual to tell what point of the skin has been stimulated. The acuteness of the sense varies in different parts of the body. It is more acute in the tip of the tongue, the palmar surface of the hand (terminal phalanx), and on the lip than on the rest of the body. It may be ascertained by gently stimulating the skin when the patient's eyes are shut, and asking him to locate the point. It can also be ascertained by touching the skin with two points simultaneously, and ascertaining if they are felt as one point or as two. Two points should be felt as distinct sensations, at a distance varying in different parts of the body. On an average, the distances in the different parts of the body are:—

Tip of tongue . . .	1 mm.
Palmar surface of hand, terminal phalanx	2 ,,
Lip	9 ,,
Front of forearm . . .	15 ,,
Forehead	23 ,,
Back of hand . . .	30 ,,
Neck, back, arm and thigh . .	50–70 ,,

The ordinary sensibility of the skin is altered in a variety of diseased conditions. Increased sensitiveness is spoken of as *hyperæsthesia*, diminished sensitiveness as *hypæsthesia*, while total loss of sensibility is distinguished as *anæsthesia*.

Hyperæsthesia is most frequently found in functional irritability of the nerves, as in neuralgia and neuritis. This hyperæsthesia is found especially at the point of emergence of the nerve from its bony canal; where the nerve enters a muscle; where a nerve lies upon a bony surface; at the point where an important branch is given off, and at the terminal ramification of a nerve. In organic diseases of the spinal cord a zone of hyperæsthesia may be present in the area of distribution of the spinal segment above the destructive lesion.

Hypæsthesia is found in a variety of partial lesions of the sensory tract.

Anæsthesia results from solutions of continuity of sensory nerves and destructive lesions of the cord and brain. It is also common in hysteria. The functional and organic forms can be distinguished by their anatomical distribution.

The Sense of Temperature, the recognition of an object as cold or warm, is specific, and distinct from tactual sensation. All over the skin are found "cold spots" and "warm spots." These spots are the seat of the specific end-organs of the nerves for the two senses. The number of these spots, and thus the acuteness of the sense, varies greatly over the surface of the body. Speaking generally, the parts of the body surface normally protected by clothes possess higher sensibility to cold than the parts habitually unclothed. Many thermo-æsthesiometers have been invented, but, clinically, it is found sufficient to gauge the acuteness of the temperature sense by the application to the skin of test tubes containing water at different temperatures, and ascertaining if the patient can appreciate the difference.

The temperature sense is frequently affected in diseased conditions of the nervous system. The sense may be

diminished, lost, or perverted. Cold may be felt, but not warmth, or *vice versâ*, or both cold and warm objects may be felt as distinctly warm or distinctly cold. The loss of the

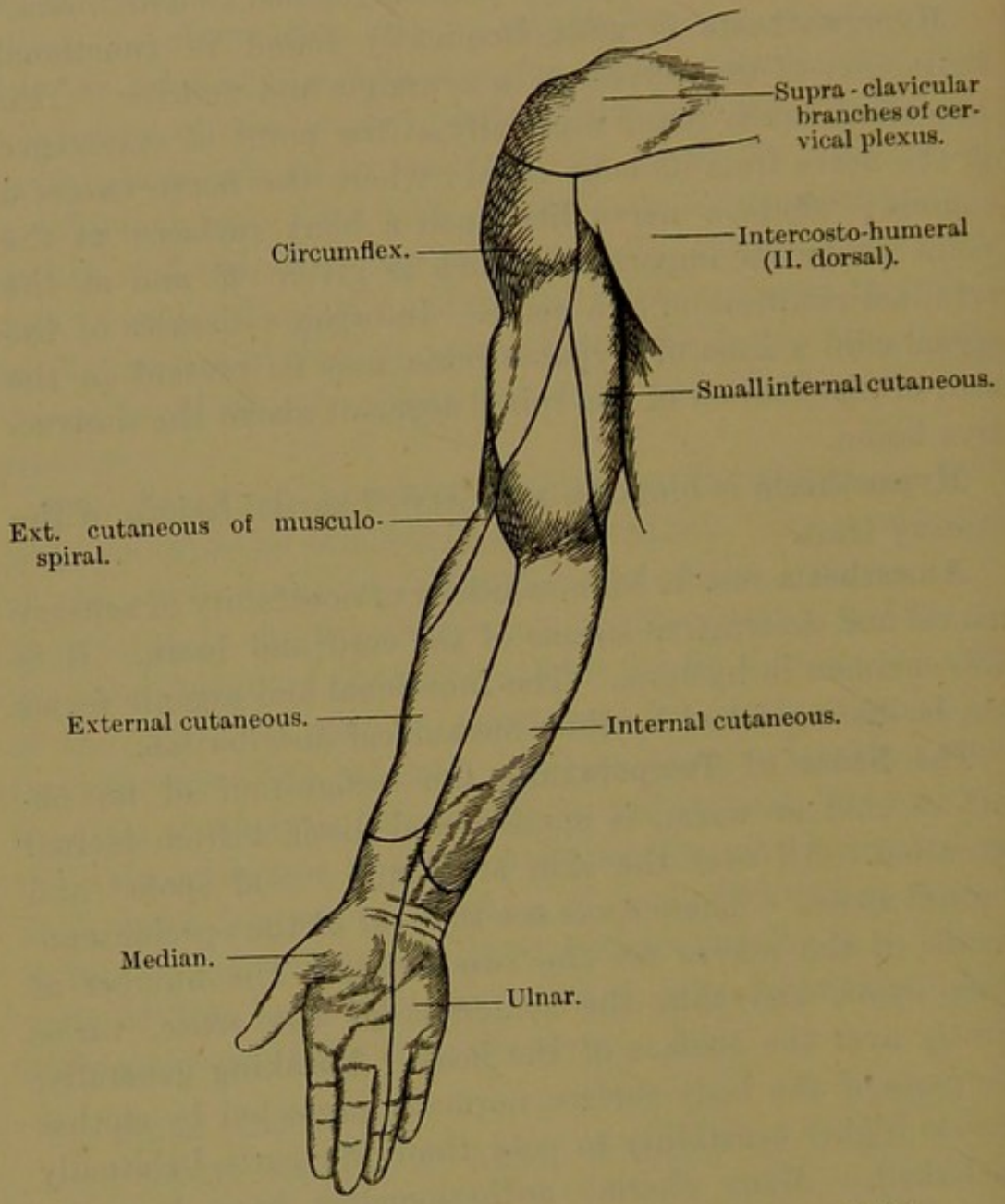


FIG. 121.—Cutaneous distribution of the sensory nerves of the anterior aspect of the arm.

temperature sense is a prominent symptom in syringomyelia, its perversion in lesions of the medulla and pons.

The Sense of Pain is at present recognised as a distinct sense, and not merely a hypertrophied tactile or temperature sensa-

tion, produced by over-stimulation of the nerves of the skin. Distinct pain spots can be mapped out on the skin, similar to

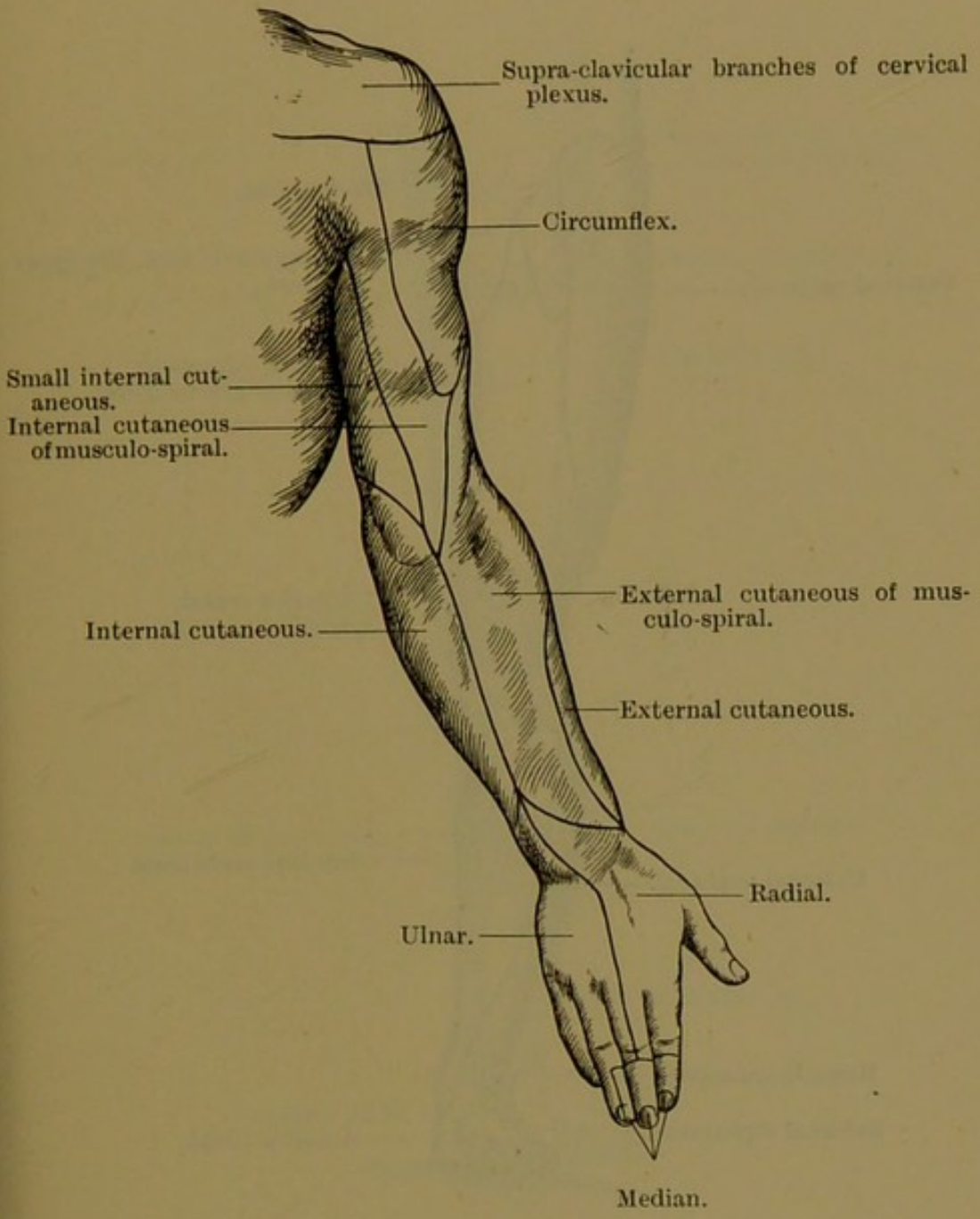


FIG. 122.—Cutaneous distribution of the sensory nerves of the posterior aspect of the arm.

the warm and cold spots. The sense of pain is usually investigated by pricking the skin with a fine needle. Deviations from the normal sensibility exist in many diseased conditions.

Hyperalgesia, or increased sensibility to painful impressions, is found under conditions similar in their nature to those which result in hyperæsthesia.

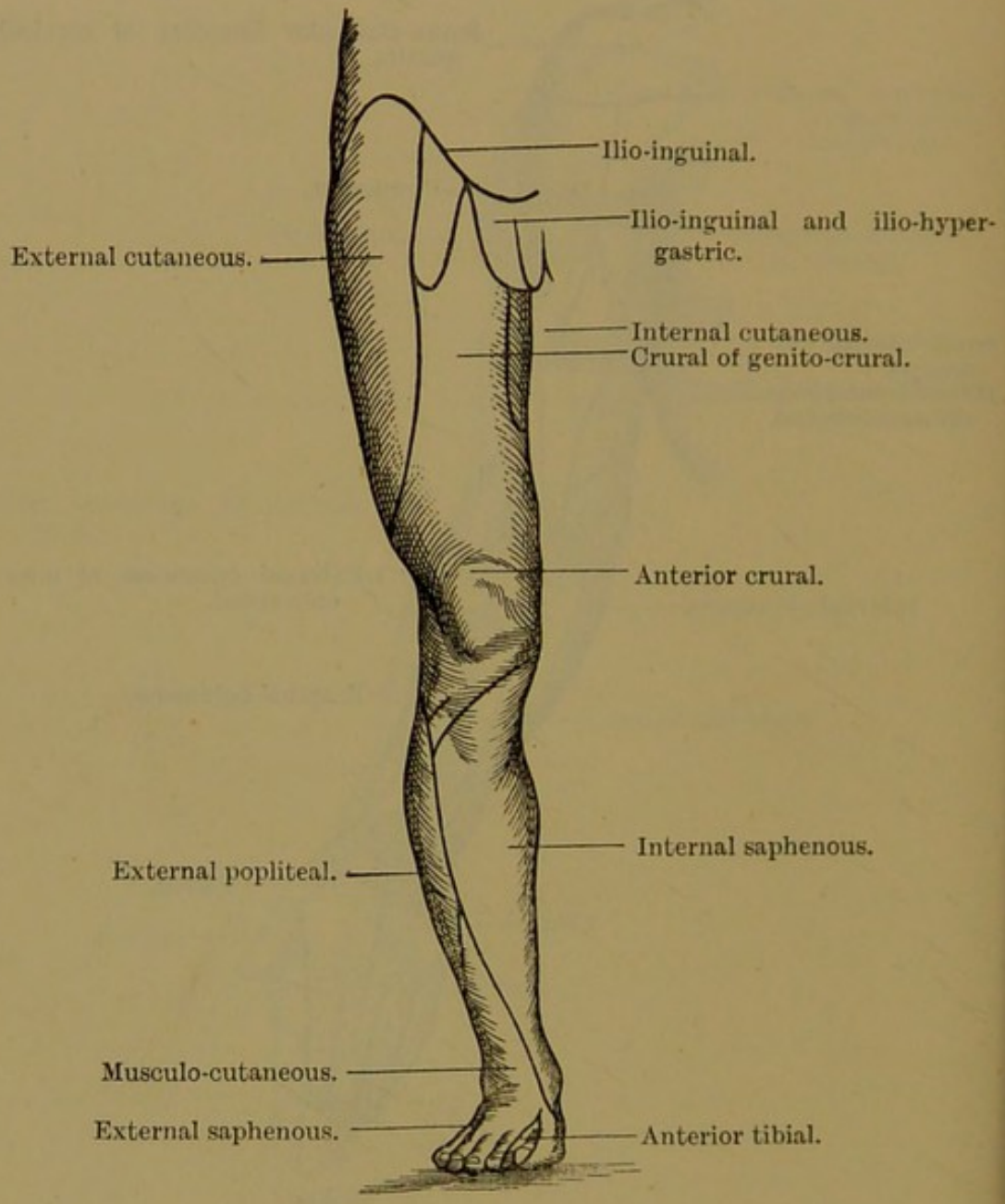


FIG. 123.—Cutaneous distribution of sensory nerves of the anterior aspect of the leg.

Analgesia, or loss of sensibility to painful impressions, results from total destruction of a sensory nerve, of the sensory tracts in the cord, or from a focal brain lesion,

particularly if situated in the parietal lobe or posterior limb of the internal capsule.

At times **perverted sensibility** may be present, the patient

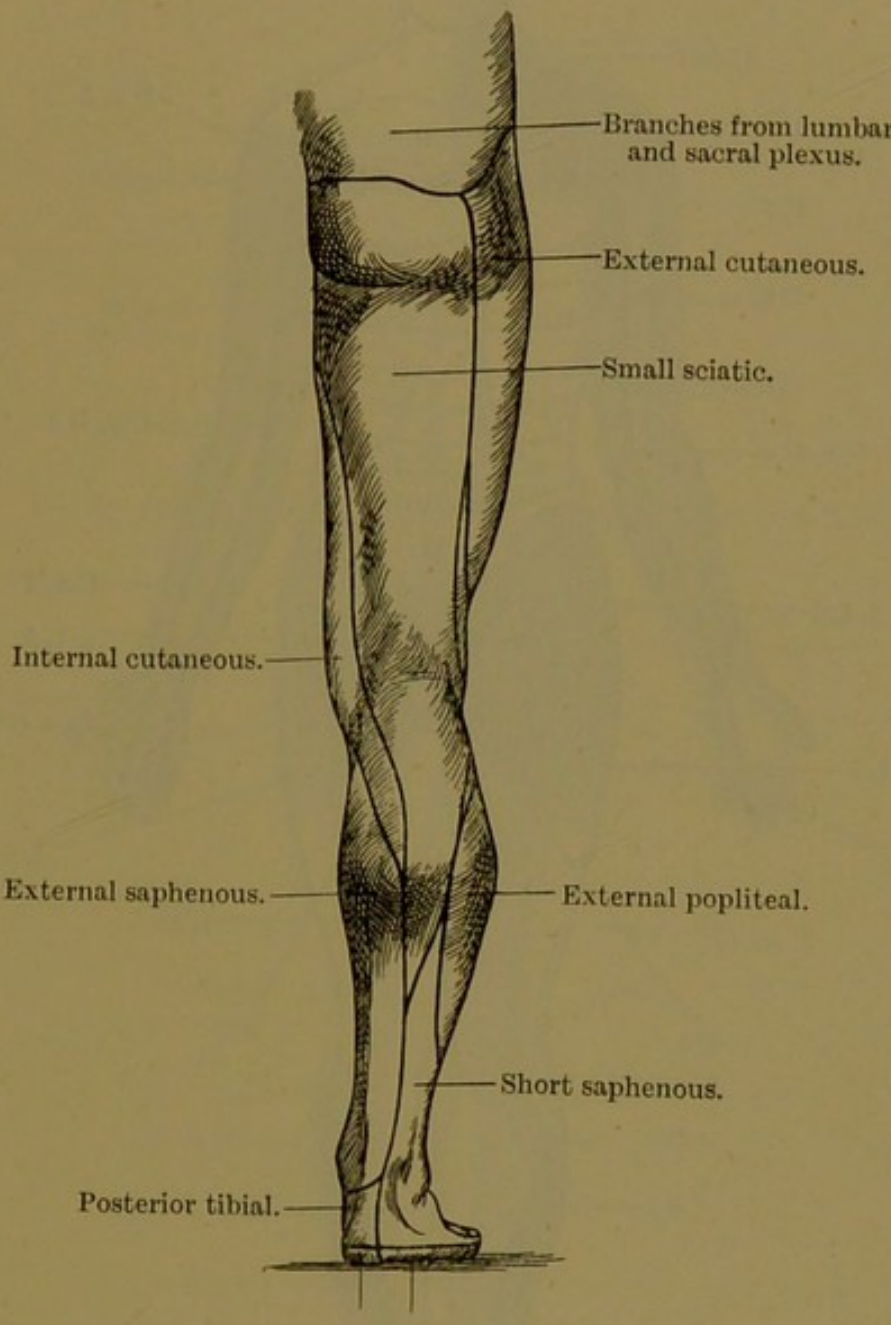


FIG. 124.—Cutaneous distribution of the sensory nerves of the posterior aspect of the leg.

feeling pain in the right leg when the left has been stimulated.

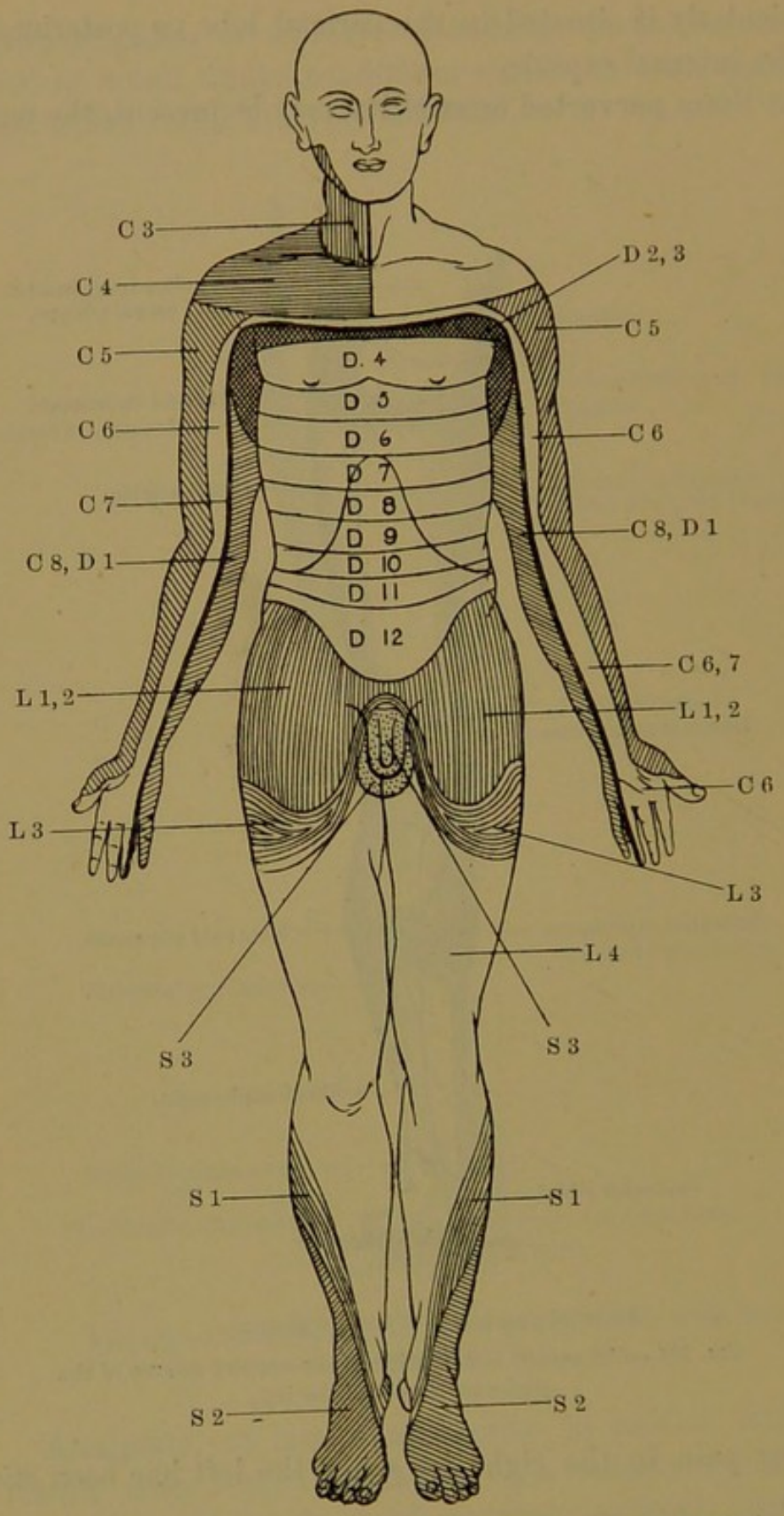


Fig. 125.—Area of peripheral distribution of spinal sensory roots, anterior aspect.—After Kocher.

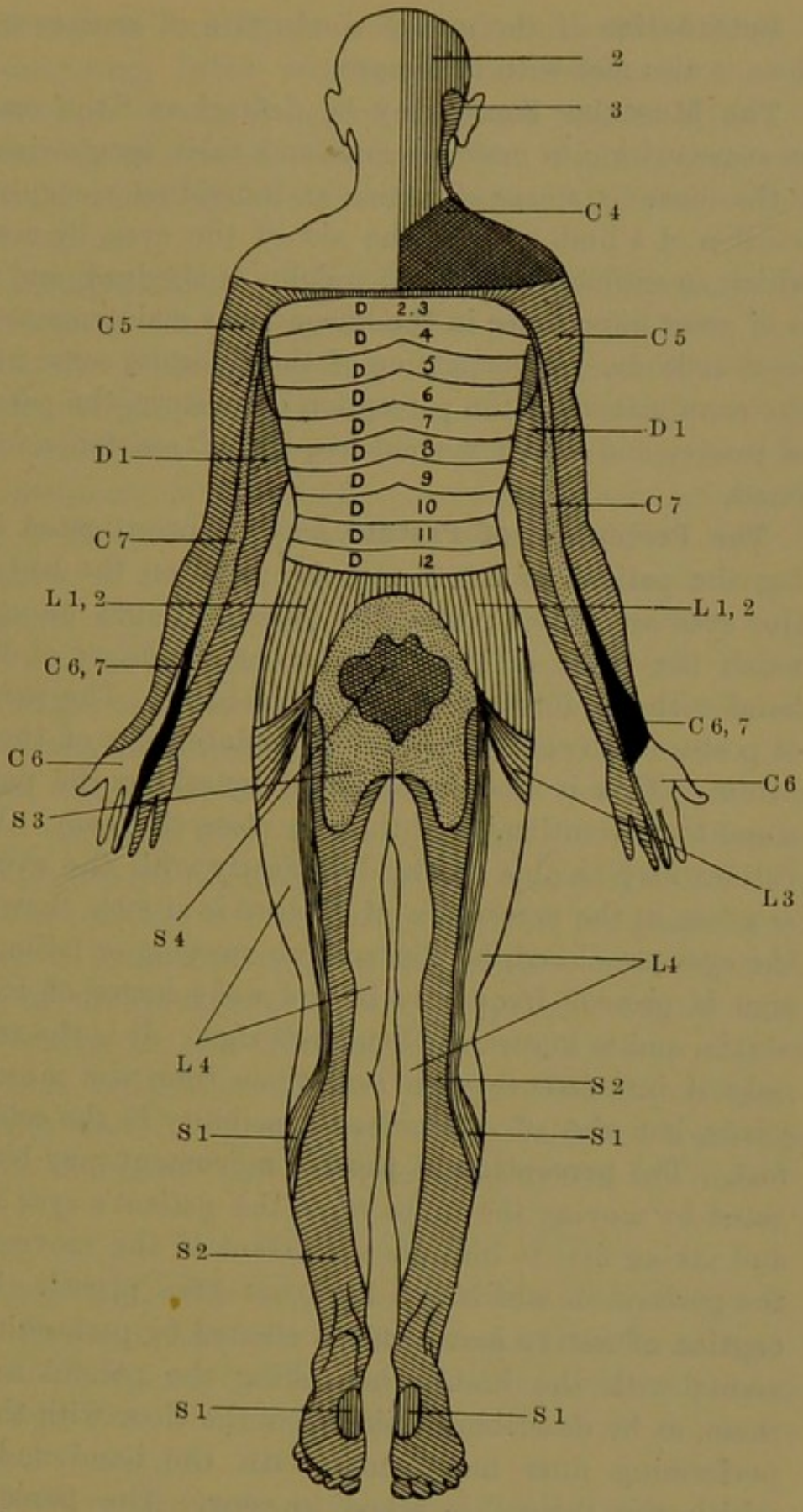


FIG. 126.—Area of peripheral distribution of spinal sensory roots, posterior aspect.—After Kocher.

Retardation of the rate of conduction of sensory impressions is also met with in disease.

The Muscular Sense may be defined as "the reactions on sense arising in motor organs and their accessories." It is the sense by means of which an individual recognises the position of a limb without the aid of the eyes, by means of which an estimation of lifted weights is obtained, and which is of great importance in the unconscious maintenance of the erect attitude. Investigation of the muscular sense includes the examination of the perception of posture, the perception of passive and active movements, and of resistance to movement.

The Perception of Posture may be investigated by asking the patient to touch a given point on the body when the eyes are closed; thus the patient should be asked to touch the point of the nose, or the forefinger of the left hand with the forefinger of the right hand. The perception of posture also contributes to the maintenance of the equilibrium. This is best investigated by asking the patient to stand to "attention," and then to close the eyes. While a patient may be able to stand perfectly with the eyes open, any loss in the perception of posture is at once shown when the eyes are closed, by the patient swaying or falling. The sign is present frequently in the early stages of locomotor ataxia, and is known as Romberg's sign. It is the result not only of interference with sensations from the muscles and joints, but also of diminished sensibility in the soles of the feet. The **perception of passive movement** may be investigated by moving the limbs when the patient's eyes are shut, and asking him to indicate the extent of the movement and the position in which the limb has been placed. The **perception of active movement** is elicited by performing movements with the limbs, and asking the patient to imitate them, as by describing a circle on the floor with the toe, or performing finer movements with the hand and fingers, which the patient is asked to copy. The **perception of**

resistance to movement is gauged by testing the power of estimating lifted weights. The weights are usually spherical, of a similar appearance, and covered with a non-conducting substance, such as leather, so that neither the sense of sight nor the thermal sense interferes with the observation.

Topographical Distribution of Sensory Disturbances.—In the investigation of disturbances of sensation, and in the localisation of the lesion, a knowledge of the area of peripheral distribution of the sensory nerves, and of the relation of the spinal roots and spinal segments with the periphery is necessary. The sensory area of distribution of the peripheral nerves is shown in Figs. 120–124. Affection of a single nerve or plexus is usually the result of traumatic affections, such as section or compression, seldom the result of a toxic or infective neuritis, with, however, the single exception of leprosy. In this disease the anæsthesia may be limited to the area of distribution of a single nerve. The peripheral relations of the posterior roots and spinal segments is shown in Figs. 125, 126. A comparison of the area of distribution of the peripheral nerves and the areas of the skin in connection with the posterior roots shows a marked contrast. The sensory areas of distribution of the peripheral nerves forms an irregular mosaic, the borders of the different areas overlapping. The skin areas, in relation with the posterior roots, are found in definite bands. These bands in the trunk run transversely, in the extremities parallel to the long axis of the limb.

II. MOTOR FUNCTIONS

Anatomy.—The motor impulses have their origin in the cells in the motor area of the cerebral cortex around the fissure of Rolando. The probable localisation of the cells, functional for the different parts of the body, is shown in Figs. 127 and 128.

The fibres arising from the cells of the different areas pass

along the corona radiata, and converge to form the genu and anterior two-thirds of the posterior limb of the internal cap-

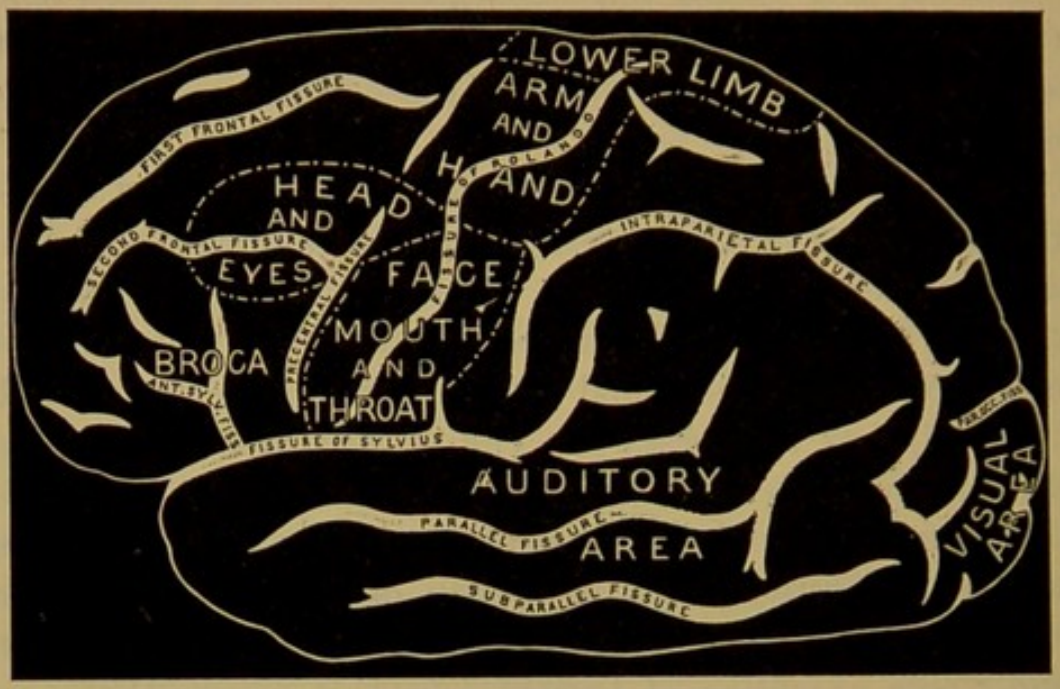


FIG. 127.—Diagram indicating the probable localisation of the chief motor and sensory areas in cerebral cortex—lateral surface.—Schäfer.

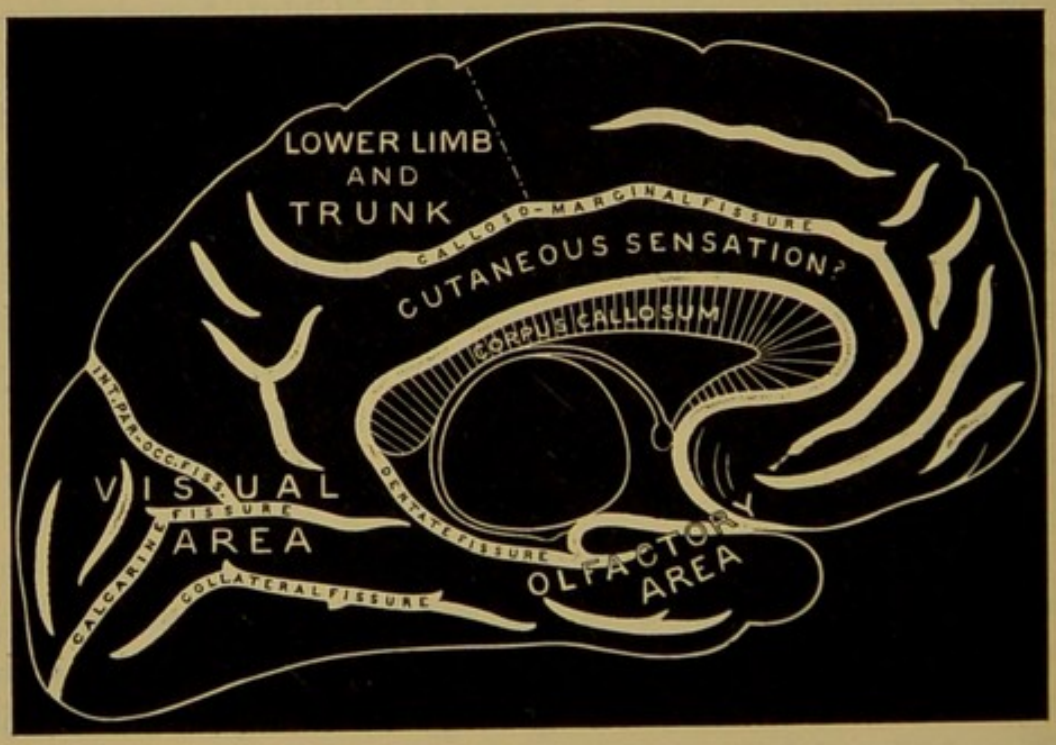


FIG. 128.—Diagram indicating the probable localisation of the chief motor and sensory areas on the mesial surface of the cerebral cortex.—Schäfer.

sule. The arrangement of the fibres in the internal capsule according to their function is indicated in Fig. 130.

Passing downwards in the crura cerebri, the fibres occupying the middle two-thirds of the crusta pass through the pons, and in the medulla occupy the anterior pyramid. At the lower part of the medulla the larger part of the fibres decussate, passing to the opposite side to form the crossed pyramidal tract of the lateral column. A certain proportion of the fibres do not cross, but pass down on the same side as the anterior or direct pyramidal tract. These fibres ultimately cross, to end in the cells of the anterior cornu of the opposite side. It should be mentioned that the decussation of the fibres from the motor centre for the facial muscles is in the pons, the fibres crossing there to the nucleus of the facial nerve of the opposite side.

The fibres of the motor tracts end in the large cells of the anterior cornu of the cord. From these cells fibres arise which pass out as the anterior root, to end in the motor end plates of the muscular fibres. Motor impulses are thus conveyed by two "*neurons*," the cells of the cortex with the axis cylinder process,—the pyramidal fibre, and the cell of the anterior cornu with the anterior root fibre ending in the muscle.

Investigation of the Motor Functions

In the investigation of the motor functions it is usual and convenient to begin with the functions of the voluntary muscles, then to pass to the visceral motor functions and the trophic functions.

Voluntary Motor Functions.—(*a*) **The tone of the muscles** is easily recognised, the flaccidity or rigidity being perceptible on grasping the muscle with the hand.

Abnormal **flaccidity** of a muscle or group of muscles follows the removal of motor impulses; it is best marked when there is disease of the motor cells of the anterior cornu.

Abnormal **rigidity** of the muscle is due to irritation of the motor neurons. It is well seen in sclerosis of the pyramidal tracts. Rigidity of the muscles is easily appreciated on squeezing the muscle with the palm, or on exciting passive movements with the limb, when the resistance will be found increased.

When rigidity is so pronounced and long continued as to lead to a more or less fixed position of the limb, it is spoken of as **contracture.** There are two forms of contracture—the active and the passive. In active contracture, muscular spasm has led to the fixation of the limb in the abnormal position. This is well seen in descending degeneration of the pyramidal tracts, as in cerebral lesions or transverse lesions of the cord. Passive contracture arises from injury, leading to more or less complete fixation of a joint, or to paralysis of one group of muscles. The limb assumes a permanent abnormal position, and, trophic changes taking place in the muscle, they become permanently shortened. These two forms of contracture can be distinguished clinically. In active contracture the limb can be extended without causing pain, and on relaxing the extending force the limb springs back into its abnormal position. The con-

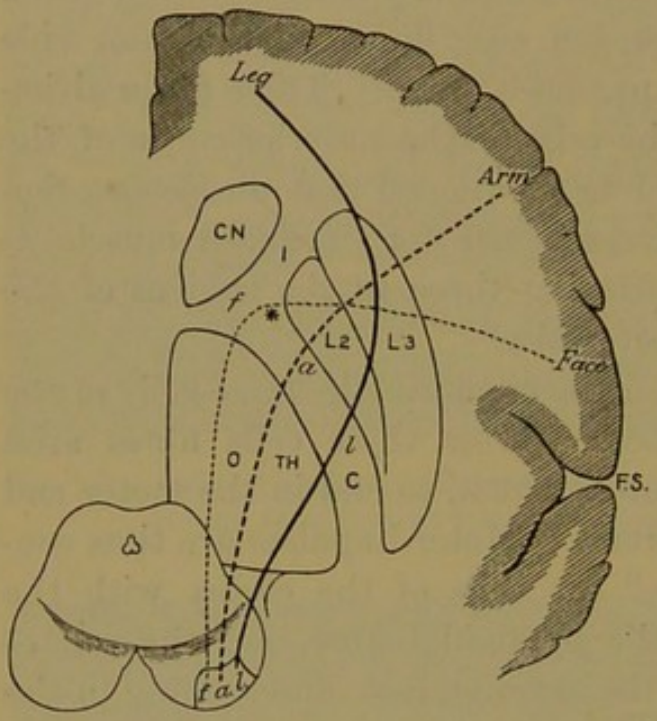


Fig. 129.—Diagram to illustrate the course and position of the fibres passing from the cortical motor areas to the crus cerebrum. The section through the convolutions and crus is in the vertical, that through the internal capsule I, C in the horizontal plane; CN, caudatic nucleus; O, TH, optic thalamus; L2, L3, middle and lower part of lenticular nucleus; *f*, *a*, *l*, face, arm, leg fibres.—After Gowers.

tracture is diminished in a warm bath, during sleep, and disappears under chloroform narcosis. It is increased on mechanical stimulation of the muscles. The reflexes of the limb are increased.

Passive contracture cannot be overcome without great pain, and the limb returns slowly to the abnormal position. It is unaffected by chloroform narcosis or by sleep.

(*b*) **The size of the muscles** may be estimated by the outline which they present, or may be more accurately ascertained by the measuring tape. Alterations in size may be produced by nervous diseases, or by other affections involving their lessened employment.

A real *increase* in the size of any muscle or group of muscles is only caused by continuous use. An apparent increase is seen in the condition known as pseudo-hypertrophic paralysis.

A *diminution* in the bulk of any muscle or group of muscles may be the result of disuse simply. The muscles of a limb, for example, which has been rendered immovable by ankylosis of a joint, invariably undergo a certain amount of atrophy.

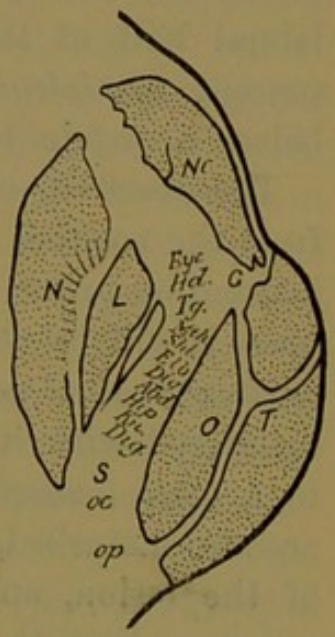


FIG. 130. — Diagram to illustrate the position of the motor and sensory fibres in the internal capsule. — After Sherrington.

Rapid wasting of muscles is in all cases produced by affections involving the loss of the influence of the anterior grey horns of the cord. It is found in disease of the motor nerves, or of the anterior nerve roots, or of the anterior cornua of the cord.

(*c*) **The movements of the muscles** may be modified in different degrees, from slight impairment to entire abolition. They may be tested by causing the patient to exert pressure against an obstacle, such as the hand of the observer. Various instruments—such as the dynamometer—have been invented

in order to test the strength of the muscles, but none of these are so satisfactory as the comparison of the power of different muscles by the hand itself.

When motor functions are lost the term *paralysis* is employed, slighter degrees of impairment being called *paresis.*

Certain terms are used to indicate the extent of the paralysis. In cases where only one group of muscles is paralysed, the term *monoplegia* is employed; where one lateral half of the body is paralysed, the case is said to present *hemiplegia*; and where both sides are paralysed below a certain level, the symptom is called *paraplegia.*

The exact site of a lesion causing paralysis is to be inferred from the position and extent of the effects, as will be further insisted on in the section dealing with reflex action. In this place it will be well to mention that in an affection of an *ordinary spinal nerve* both motor and sensory functions are interfered with in one region, and there is rapid wasting; in a *total transverse spinal lesion* both of these functions are also disturbed, on both sides of the body below the level of the lesion, and there is rapid wasting of a small area corresponding to the anterior cornual zone affected; in affections of the *anterior grey matter* of the cord, the paralysis is attended by rapid wasting; in disorders of the *motor tract*, the paralysis is associated with spasmodic symptoms, and if the motor tract be diseased in the medulla oblongata there is frequently some interference with such organic functions as respiration, or circulation, or deglutition; in diseases situated in the *anterior part of the pons cerebri*, there is paralysis of one side of the body and of the opposite side of the face, commonly termed "crossed paralysis"; if the *crus cerebri* is destroyed, there is paralysis of one side of the body, and of some of the ocular muscles of the other side; when there is a lesion in the *anterior part of the posterior limb of the internal capsule*, there is purely motor paralysis, often with contracture as a sequel, and if

the disease includes the *posterior part*, there is paralysis of both motility and sensibility.

(*d*) **Abnormal movements** may be observed in muscles. These are usually termed *spasms*. Spasms are of two kinds —*clonic*, in which there is alternate contraction and relaxation; and *tonic*, in which there is persistent contraction of a muscle or group of muscles.

Clonic spasms vary in extent and degree, from faint movements in muscular fasciculi, causing no change in the position of the limb, to general convulsions, involving the entire body. Minor degrees are termed *tremors*, and the slightest form is the **fibrillary contraction** of individual muscular bundles, causing a faint wave to travel along the surface of a muscle. On a larger scale they may be seen in the rhythmic tremors, which are present both during repose and action in paralysis agitans, in the irregular jerks seen in chorea during rest and activity, and in disseminated sclerosis during the action of the muscles. In the highest development they form the local or general convulsions characteristic of explosive nervous diseases. A special form of clonic movement is seen in *athetosis*, which usually occurs as a sequel to hemiplegia. In this symptom the fingers and toes maintain a series of continuous slow movements.

Tonic spasms likewise vary in extent and degree. In their slightest form they occur in cramp, affecting individual muscles, a group of muscles as in writer's cramp, in a more pronounced form as contracture, and in their highest possibility as tetanus.

(*e*) The **mechanical irritability** of a muscle is easily tested by the application of such an excitant as a moderately strong blow, which under ordinary circumstances produces a contraction of the part of the muscle struck. Changes in the degree of mechanical irritability are of small aid to diagnosis. In certain wasting diseases, especially phthisis, the irritability is greatly increased. Tapping over a motor nerve, where it lies superficially, may give rise to a contrac-

tion of the muscles supplied by the nerve. This mechanical irritability may be greatly increased; thus in tetany tapping over the facial nerve will at once give rise to sudden contraction of the facial muscles.

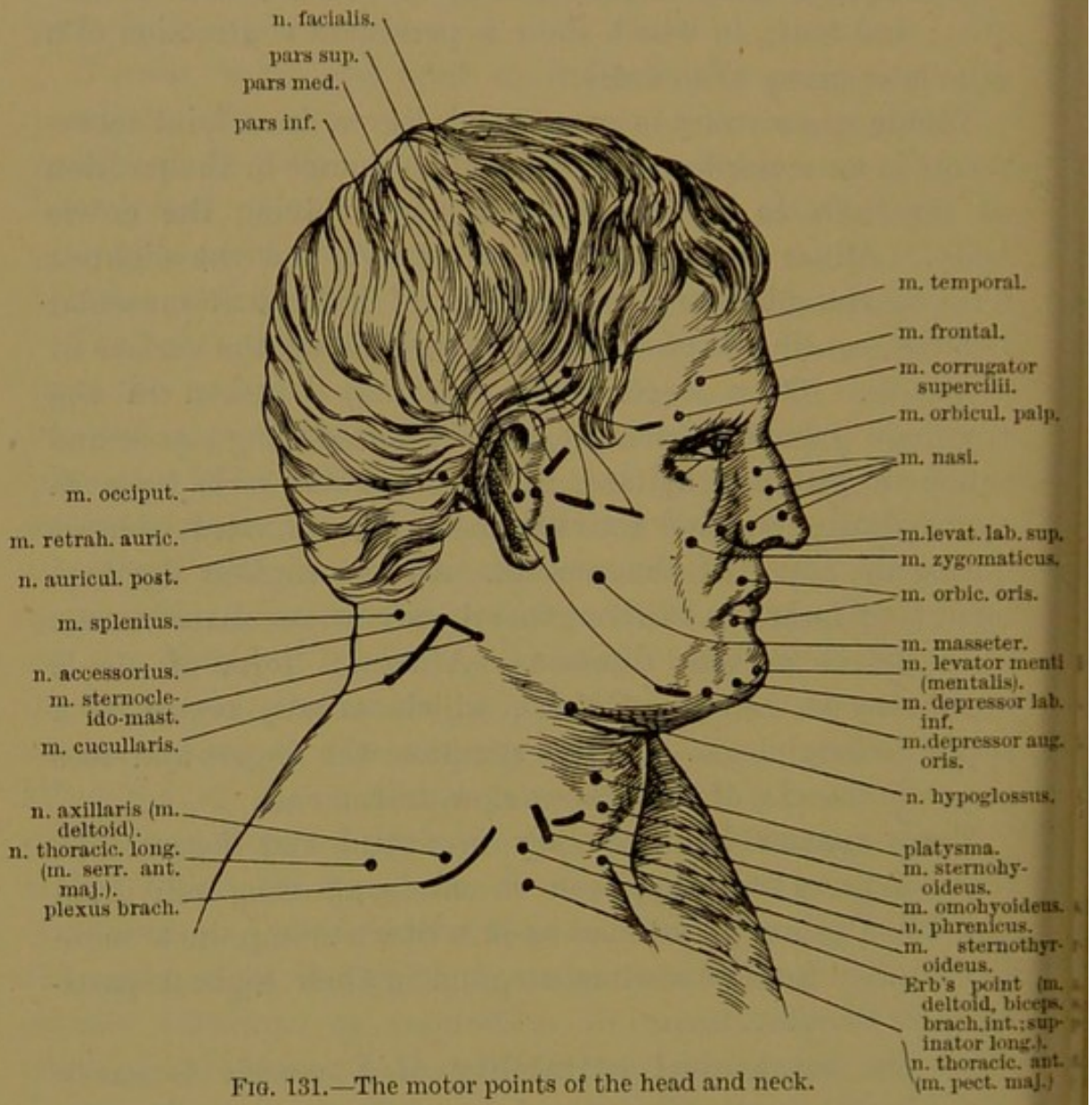


Fig. 131.—The motor points of the head and neck.

(*f*) The examination of the **electrical irritability** is of great value in the diagnosis of nervous diseases.

Electricity is employed for diagnostic purposes in clinical medicine in the form of the continuous current, the interrupted current, and as the X-rays described by Röntgen.

In using the continuous or the interrupted currents, the polar method is always employed for diagnostic purposes. The skin and electrodes having been well moistened, a large

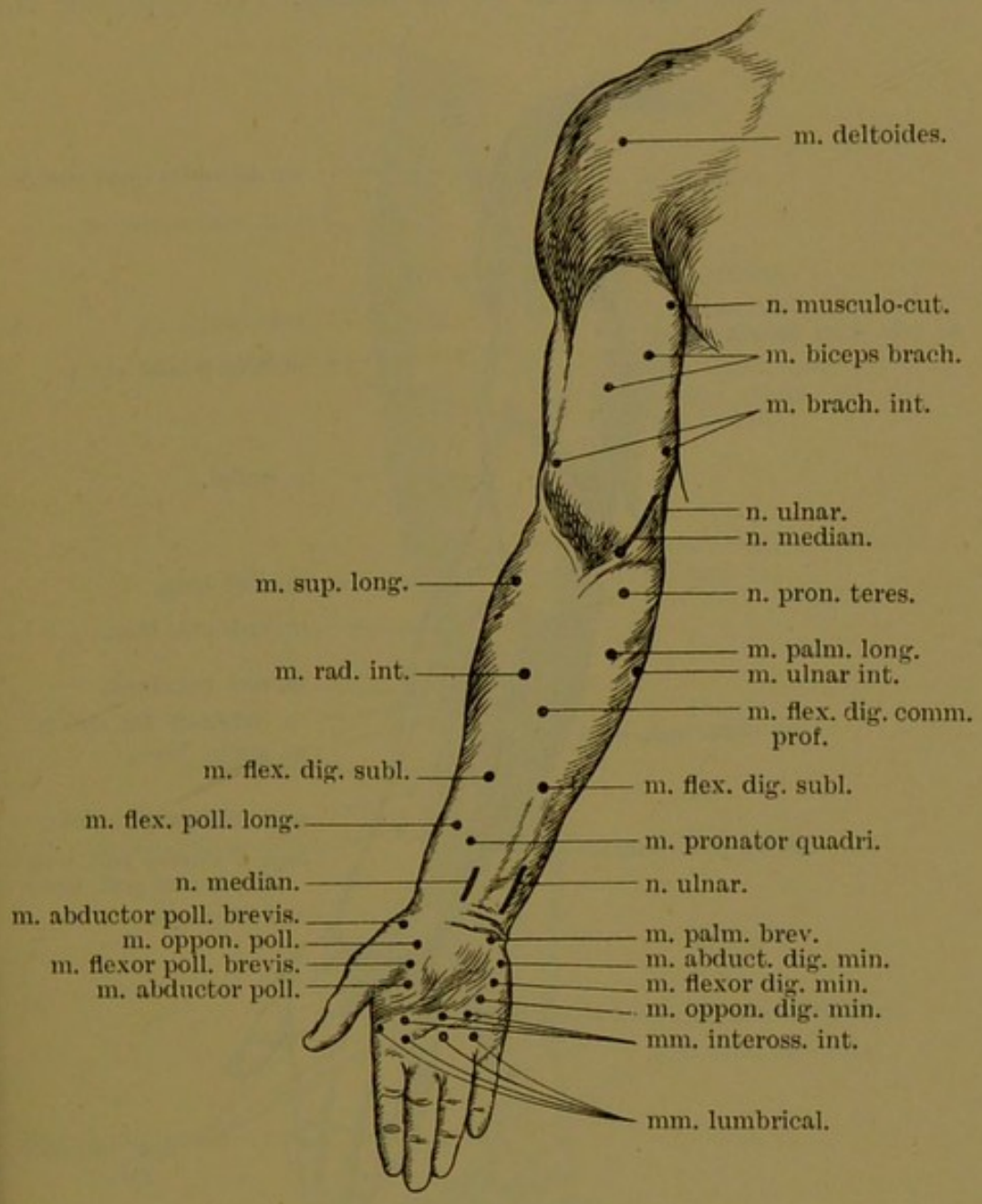


FIG. 132.—Motor points on the flexor surface of the arm.

flat electrode is applied to a central point on the body, while the second, a small electrode, is applied to a point on the skin over the nerve or muscle which it is desired to stimulate. Before applying the electrodes to the patient the

observer would do well to test the strength of the current on his own person. A weak current being used to begin with, the stimulus is applied to the trunk of the motor nerve or to the muscle to be examined. In applying the

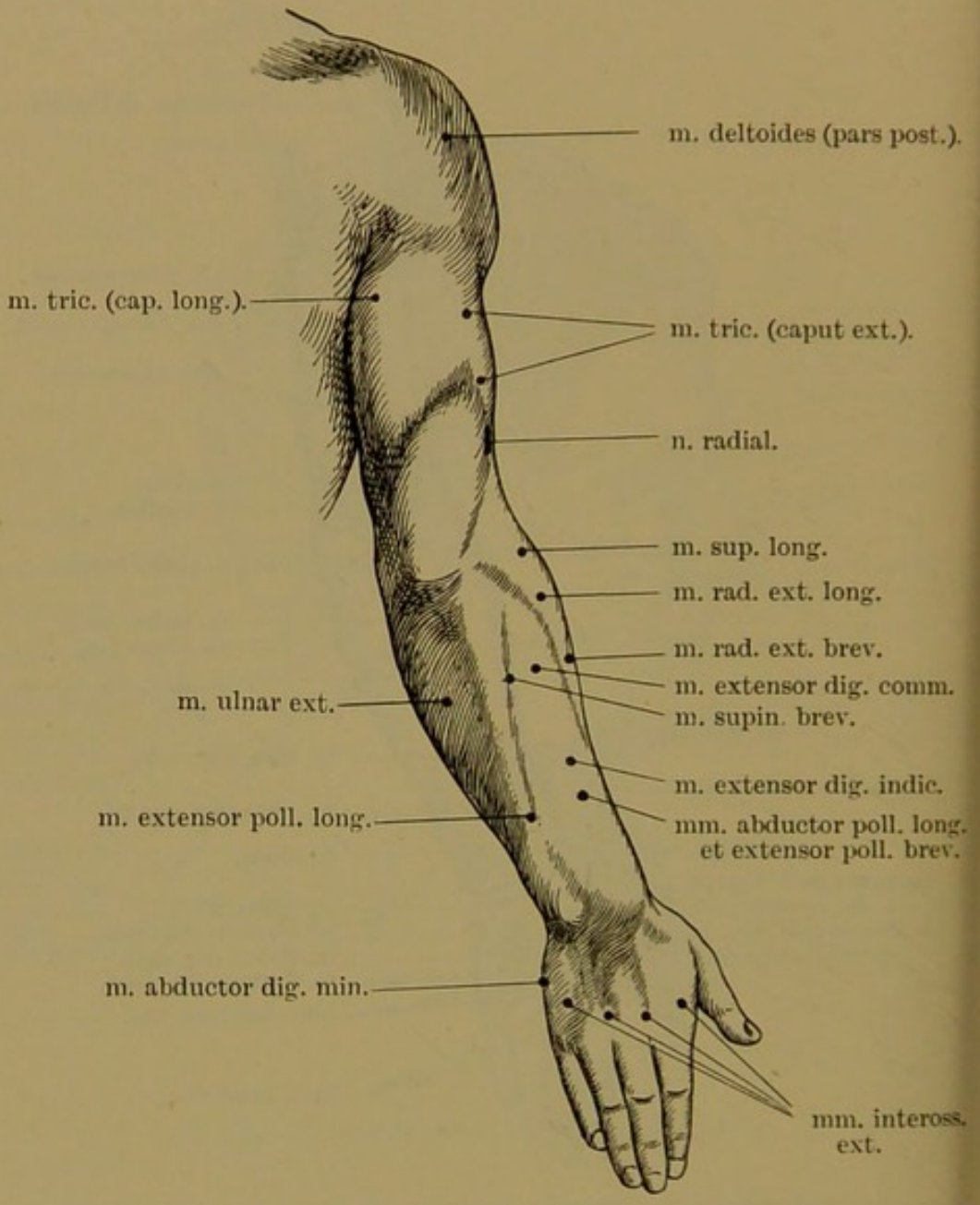


FIG. 133.—Motor points on extensor aspect of arm.

current to the muscle the electrode should be placed at the point of entrance of the motor nerve, the motor point of the muscle. The motor points for the different muscles are figured in Figs. 131–135.

It is well to begin the examination with the interrupted or faradic current, which excites muscular contraction through the motor nerve. The large electrode being applied

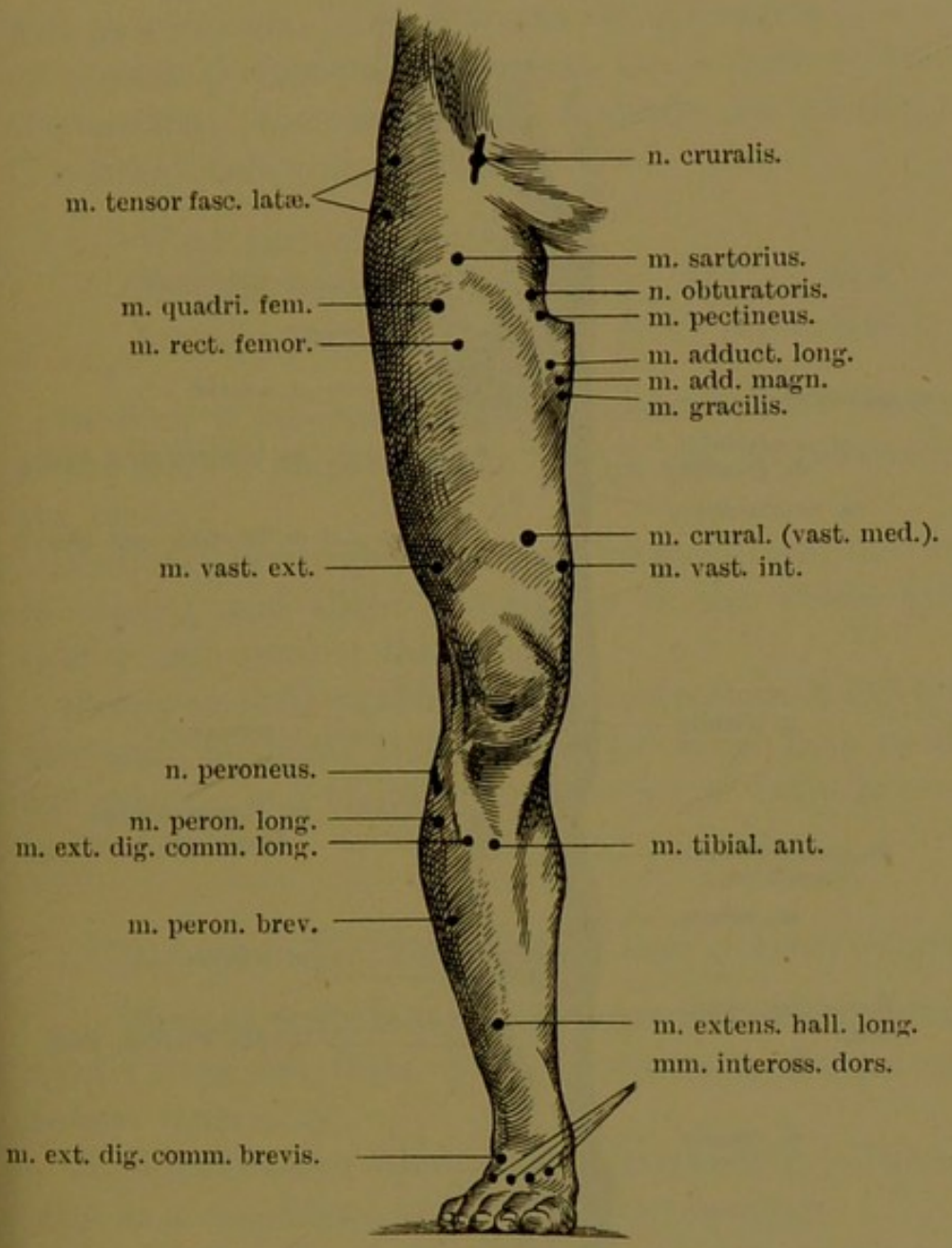


Fig. 134.—Motor points on anterior aspect of leg.

centrally, the small electrode is applied to the nerve trunk to be stimulated, and the observer notes the strength of current required to induce a contraction. If a unilateral lesion be suspected, the corresponding muscle or group of

muscles on the opposite side is next examined to give a standard of comparison. The law of normal faradic excitability is, that the *degree of contraction varies directly with the strength of the current employed.*

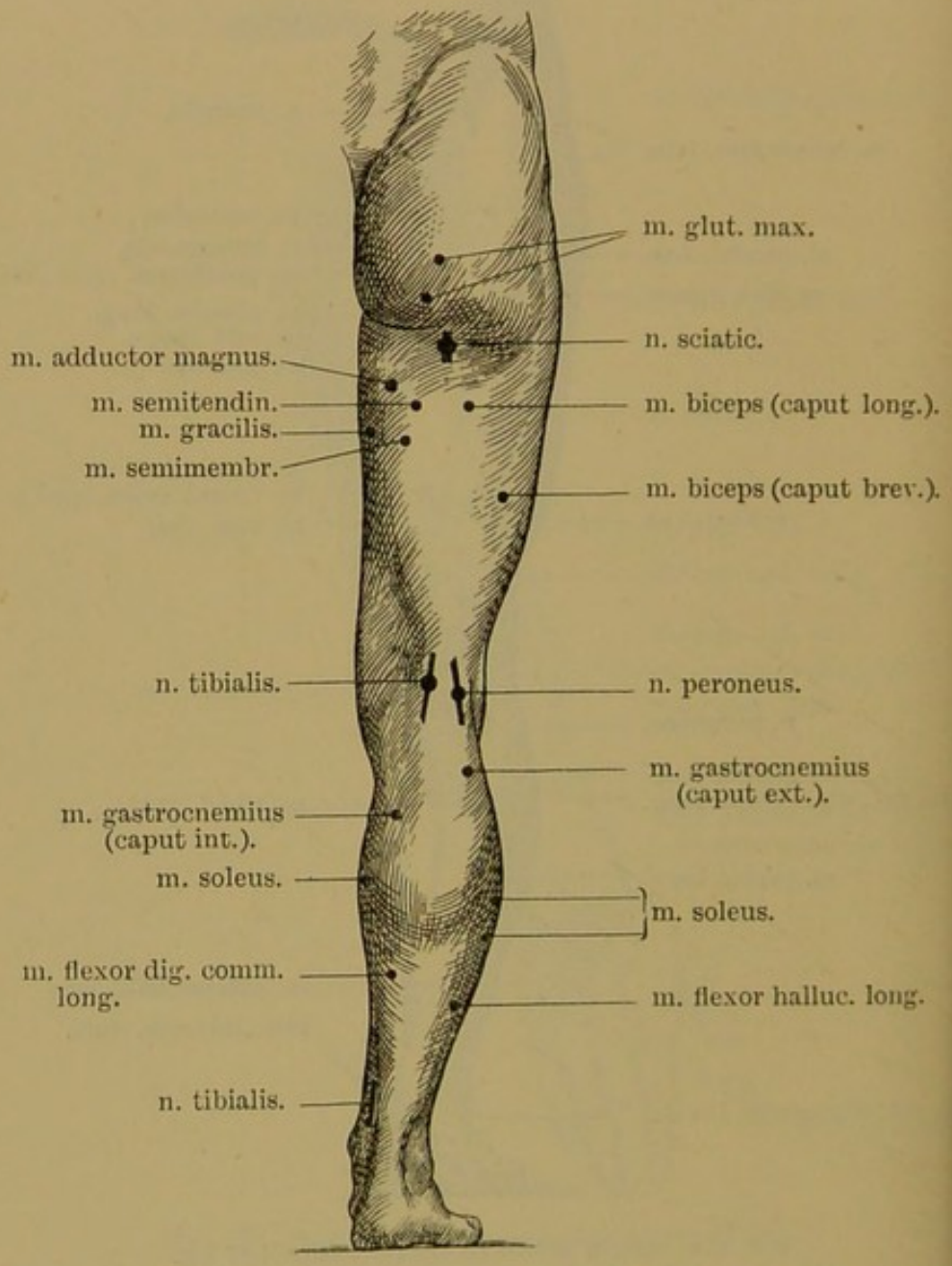


FIG. 135.—Motor points on posterior aspect of leg.

The **continuous current** produces no muscular contraction when flowing, but causes contraction when closed or opened. The cathode or negative pole chiefly produces

contraction when the current is closed; the anode or positive pole when the current is opened; and the cathode produces stronger effects than the anode.

The law of normal galvanic irritability may be stated here as a formula. Let K = cathode, A = anode, *C* = closure of current, *O* = opening of current, C – = weak contraction, C = moderate contraction, C + = strong contraction, and C^t = tetanic contraction; then—

Weak currents give K*C*C–;
Medium currents give K*C*C +, A*C*C –, A*O*C –
Strong currents give K*C*C^t, A*C*C, A*O*C, K*O*C –

In other words, weak currents produce slight contraction at the cathode on closing the circuit, but no contraction at the anode.

Medium currents produce strong contraction at the cathode on closing, and slight contraction at the anode both on closing and opening the circuit.

Strong currents produce tetanic contraction at the cathode on closing, moderate contraction at the anode both on closing and opening, and slight contraction at the cathode on opening the circuit.

Changes in Galvanic and Faradic Irritability

A. **Normal irritability.**—In hysterical and most cases of spastic paralyses there is no alteration in the galvanic or faradic irritability.

B. **Quantitative alterations of irritability.**—The change may be of the nature of an increase or decrease.

An *increase* of galvanic irritability is found in some cases of early hemiplegia, of spastic paralysis, and of locomotor ataxy. This causes some modifications from the conditions stated as the law of irritability, inasmuch as currents produce much more powerful effects than under normal circumstances.

A *decrease* occurs in the later stages of all paralyses leading to wasting of muscles, and it is found in all muscles which have undergone atrophy from disuse.

C. **Qualitative and quantitative alterations of irritability.**—These changes are characteristic of cases in which there is great disturbance of the nutrition of muscles. They are found in the various forms of peripheral paralysis from inflammatory changes in, or the effects of toxic agents on, nerve trunks. They are also present in diseases of the anterior cornua of the cord and the corresponding structures of the medulla. They are not met with in cases of cerebral paralysis, of spastic paralysis, of hysterical paralysis, or of paralysis from disuse of muscles. The great use of these alterations is the distinction given between lesions of the higher and lower neurons.

The alterations are commonly known by the designation of *the reaction of degeneration*, and they only occur in lesions of the lower neuron. As they do not follow a similar course in nerve and muscle, these must be taken up separately.

The reaction of *affected nerves* is marked by a progressive lessening of both galvanic and faradic irritability, without any alteration in their mutual relations to each other. This goes on in cases which are incurable to a permanent loss, but in curable cases the irritability to both currents returns simultaneously and gradually.

The reaction of the *affected muscles* to the different currents is marked by a change in their relationship. The galvanic irritability lessens for a few days after the commencement of the attack, and after ten or twelve days becomes greatly increased, so that the muscles respond to weaker currents than in health. This continues until about the fourth week, from which a gradual lessening goes on in curable cases to the normal, in incurable cases to a total loss. There is also a change in the relations of contraction at the cathode and anode, so that the anodal closing contraction is as great as, or greater than, the cathodal closing contraction,

and the cathodal opening contraction is as great as, or greater than, the anodal opening contraction. The faradic irritability is gradually lost, just as in the case of affected nerves, and gradually regained in curable cases.

The **clinical significance** of this "reaction of degeneration" is that it occurs in muscles when they are cut off from trophic nervous influences; when there is, in fact, disease of the lower neuron, consisting of the motor nerve, the anterior root, and the motor cells in the anterior cornua, or in the case of cranial nerves, the cells of the brain nuclei. It is thus typical of peripheral neuritis and of destructive lesions of the cells of the anterior cornua or brain nuclei. It is not found in paralysis of cerebral origin, in hysterical paralysis, or in paralysis due to muscular affections.

III. REFLEX FUNCTIONS

Reflex irritability affords useful aid in diagnosis. The explanation of the phenomena to be mentioned in this section cannot be said to be quite satisfactory, but it may be said that, for the performance of any reflex act, there must be unimpaired activity of the different structures entering into the reflex loop.

The reflex loop or arc consists of an afferent fibre, a centre, and an efferent fibre. By a knowledge of the anatomical position of the reflex centres connected with the various groups of muscles, and of the afferent and efferent paths, and by examination of the different reflexes, much diagnostic information can be obtained. At times even in health a reflex may be difficult to obtain, or may even be abolished through the attention of the patient being directed to it, involuntary inhibition being exerted. The reflex may then be brought out by distracting the patient's attention. Thus if the knee jerk is being examined, the patient is directed to make a voluntary effort with the upper limbs, such as hooking the fingers together and exerting traction. This is

termed **reinforcement** of the reflex. In discussing the reflexes, it is simplest to consider them from above downwards in relation to the anatomical position of the reflex centre.

The **jaw jerk** is a contraction elicited from the muscles attached to the jaw. To elicit the jerk the patient is directed to open the mouth slightly. The observer then places one finger upon the mental prominence under the lower lip, and strikes the finger sharply, as in percussion. In health a jerk is not always nor even usually elicited; but in conditions where there is increased reflex excitability, such as neurasthenia, hysteria, cerebro-spinal sclerosis, a marked jerk can be elicited. The reflex centre for the jaw jerk is the motor nucleus of the fifth nerve. Allied to this reflex is the phenomena known as **Chvostek's sign.** It is a sudden twitching of the muscles of the face, especially the muscles of the eyelids and elevators of the angle of the mouth. It can be elicited in tetany by percussing the skin of the face just in front of the ear.

The **biceps jerk** is obtained by the patient resting the relaxed arm upon some support, such as the arm of the observer. The tendon of the biceps is then struck with the percussion hammer or the end of a stethoscope. In most people a slight reflex contraction of the biceps can be obtained in this way. The **triceps jerk** is elicited by semi-flexing the forearm and striking just above the olecranon. The **supinator jerk** is got by striking the radius just above the styloid process when the arm is relaxed on the supporting arm of the observer. These reflexes cannot usually be obtained in health. Their absence is of no pathological significance. They are present and exaggerated in conditions where there is increased reflex irritability, as on the paralysed side in hemiplegia.

The **scapulo-humeral reflex** is obtained by tapping the spinal border of the scapula just above the angle, when a slight adduction and external rotation of the arm occurs in

normal conditions. Under pathological conditions, such as sclerosis of the pyramidal tracts above the cervical enlargement, the response may be greatly altered. The shoulder is lifted, the arm thrown from the side, the forearm flexed, and the fingers extended. The posterior fibres of the trapezius, the deltoid, the biceps, and the muscles of the forearm have all reacted to the stimulus. In addition, a crossed reflex may be present from a response in the muscles of the opposite shoulder.

Table to show Localisation of Functions in different Segments of the Cord. Compiled from Kocher, Thorburn, Ferrier, Bruce.

Segment.	Motor.	Sensory.	Reflex.
C. I. . .	Small rotators of head. Trapezius.	.. ..	.. ..
C. II.–III.	Trapezius. Sterno-hyoid. Omo-hyoid. Sterno-mastoid.	Back of head. Neck behind ear. Ear. Neck beneath chin.	.. ..
C. IV. . .	Muscles of shoulder. Diaphragm. Supinator longus. Rhomboids.	Upper part of chest. Shoulder. Back to level of middle of scapula.	Cilio-spinal reflex, C. IV.–VII.
C. V. . .	Muscles of shoulder. Diaphragm. Latissimus dorsi. Rhomboids.	Outer side of arm, and forearm as far down as thumb.	Biceps jerk. Triceps jerk. Scapulo-humeral reflex, C. V.–D. I.
C. VI. . .	Muscles of arm. Pectoralis major. Extensors of hand and fingers. Pronators.	Strip of arm and forearm. Portion of palm of hand with thumb and one and a half fingers.	.. Wrist jerk.
C. VII. .	Extensors of hand and fingers. Pronators and flexors of hand. Pectoralis major.	Strip of arm and forearm and hand, extending down middle and ring fingers.	..
C. VIII. .	Flexors of hand and fingers. Small muscles of hand.	Ulnar aspect of hand, little and half of ring finger.	..

Localisation of Functions—*contd.*

Segment.	Motor.	Sensory.	Reflex.
D. I. . .	Extensors of thumb. Muscles of thenar and hypothenar eminences.	Inner aspect of arm and forearm.	.. ..
D. II.–XII.	Muscles of back and abdomen.	Chest and abdomen.	Epigastric reflex, IV.–VIII. Abdominal reflex, VIII.–XII.
L. I. . .	Muscles of abdomen. Sartorius.	Pelvic region.	Cremaster.
L. II. . .	Psoas-iliacus.	Pelvic region.	..
L. III. . .	Psoas-iliacus. Sartorius. Adductors. Extensor cruris. Obturator externus.	Upper part of thigh. .. Thigh and inner surface of leg.	.. Patellar reflex, III.–IV.
L. IV. . .	Extensor cruris. Adductors. Gluteus medius and minimus.		Gluteal reflex, IV.–V.
L. V. . .	Hamstrings. External rotators of hip. Calf muscles. Extrinsic foot muscles.		
S. I. and II. . .	Hamstrings. External rotators of hip. Calf muscles. Extrinsic muscles of foot. Intrinsic muscles of foot.	.. Back of thigh. Outer surface and back of leg. Dorsum of foot. Area on sole of foot.	.. Plantar reflex. .. .. ..
S. III. . .	Calf muscles. Extrinsic muscles of foot. Intrinsic muscles of foot.	Gluteal region. ..	Ankle clonus. Achilles jerk, III.–V.
S. IV.–V. .	..	Penis and scrotum. Sacral region.	Bladder and rectal.

The **abdominal reflex** is a contraction of the abdominal muscles, obtained by gently stimulating the sensory fibres of the intercostal nerves, by stroking the skin of the abdomen with a blunt instrument, such as the end of a stethoscope.

The **cremasteric reflex** is elicited by stroking the inner side of the thigh, when the testicle on the same side is retracted. It is not always present in health.

The **knee jerk** can be produced under ordinary circumstances with comparative ease. If the knee is flexed and the leg allowed to hang freely, a tap upon the patellar tendon will be followed by a distinct jerk forward of the leg and foot, from sudden contraction of the quadriceps muscle. The same result may, in many cases, be obtained by tapping the attachments of the quadriceps to the patella, or even by striking the muscle itself. In order to study this phenomenon the patient should be placed in a sitting posture, and the observer should support the thigh of the patient, by passing his arm beneath it so as to rest his hand on the other knee of the patient. The stimulus should be applied by striking the patellar tendon with the inner edge of the extended hand, with the margin of the large end of a stethoscope, or with a percussion hammer. Care must be taken not to mistake mere swinging of the limb for the true knee jerk.

Ankle clonus may be produced in certain cases. To elicit it the limb is slightly flexed at the knee, and the foot is passively flexed by the observer pressing the sole upwards by the application of the hand; a series of jerks or clonic spasms is produced by alternate contraction and relaxation of the muscles of the leg, which continues as long as the pressure is maintained. In order to produce the phenomenon it is well that the knee should not be entirely extended, but kept in a semi-flexed position.

Knee clonus is a rhythmic contraction of the quadriceps, obtained by sudden extension of the tendon by pushing down the patella when the limb is in the extended position.

The **ankle jerk** can be elicited by tapping the tendo Achillis with the foot at right angles; extension of the foot results.

The **plantar reflex** consists in a flexion of the toes upon the sole of the foot with withdrawal of the foot when the sole is stroked. In anæsthesia of the foot the plantar reflex is absent.

Babinski's phenomenon is elicited by stroking the inner surface of the sole from the heel towards the toes. Normally the toes show plantar flexion. In cases of sclerosis of the pyramidal tracts of the cord the great toe is extended upon the foot; in some cases all the toes show the extensor movement.

Quantitative and qualitative alterations in the reflexes are of great importance in clinical work.

A **decrease** of reflex action is due to some interference with the reflex loop. This, as is shown by the diagram (Fig. 136), may be produced by disease of the nerve trunk—

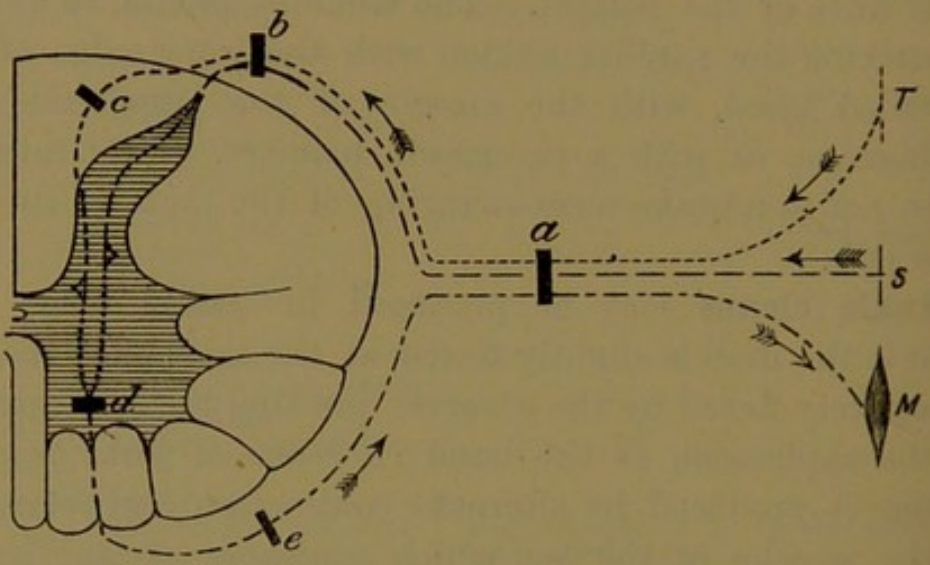


FIG. 136.—Diagram showing seats of interference with deep reflexes. *M*=muscle; *S*=skin; *T*=tendon.

(*a*) When motor and sensory functions are interfered with; by disease of the posterior root (*b*), when sensory functions only are altered; by disease of the posterior root zone (*c*), when no sensory or motor changes occur; or by disease of the anterior cornu (*d*), or anterior root (*e*), when motor functions are abolished and there is rapid wasting. It is most frequently diminished by disease of the posterior nerve roots, or of the posterior root zones, or of the anterior cornua. The position of the interruption must be determined by the associated symptoms. Loss of reflex action may occur as a transient symptom immediately after an

acute lesion of the cord, apparently from irritative inhibition of the centres. Cutaneous reflex action may be lessened permanently, in some cases of brain disease on the side of the motor palsy, even when muscle reflex action is increased. It is well to remind the student how difficult it is to be certain in some cases, whether the knee jerk is present or lost, owing to the readiness with which its occurrence may be prevented by the inability of the patient to relax the muscles.

Excess of reflex action implies of necessity the integrity of the reflex arc concerned. Each form of reflex action is often increased. In acute diseases, such as meningitis, the increase may be due to irritation of the centres. In most chronic affections the increase is the result of the loss of control of the higher neurons, and indicates disease between the reflex centre and the brain. All reflex action is controlled by the higher centres of the brain, and when this control is lost excessive reflex action results. This is well seen in degeneration of the pyramidal tracts of the cord (spastic paraplegia), and especially well seen if the terminal portion of the fibres within the grey matter be involved (secondary descending degeneration). The increase of reflex action is usually best manifested in the legs, in which reflex action is normally more active and more important than in the arms.

The **cranial reflexes.**—In addition to the skin and muscle reflexes there are what are termed the cranial reflexes. The principal of these are the sneezing produced by irritation of the nose, the conjunctival reflex to irritation of the conjunctiva, the reaction of the pupil to light, the muscle contractions produced by a loud noise, the contractions of the muscles of the palate and gullet produced by irritation of the fauces, and the coughing produced by irritation of the larynx or trachea. Of these reflexes the most important are the pupil reflexes.

The **pupil reflexes.**—In health the pupil reacts directly

and indirectly to the stimulus of light, to accommodation and to the stimulus of pain.

To observe the reactions to light, the patient is placed facing the light of a window, with one eye carefully excluded by the observer's hand. The patient is then directed to look into the distance, and the size of the pupil noted. The eye under observation is then shaded from the light, and if normal the pupil will be seen to enlarge; if the shading be next removed, the pupil will contract to its former size. This is known as the **direct reflex action of the pupil.** If during the observation the obscured eye be noted, the pupil will be found acting in unison with its fellow—**the indirect reflex action of the pupil.**

The **convergence reaction** of the pupil is tested by requiring the patient to look into the distance and then to quickly direct his eyes to a near object, such as the point of a pencil held close to the eyes: the pupil is found to contract under the influence of accommodation and convergence,—the sphincter of the iris contracts in association with the ciliary muscle and internal recti.

The **skin reflex** of the pupil consists in a dilatation which occurs on stimulation of a cutaneous nerve. It is best elicited by pinching the skin of the neck. The pupil-dilating fibres, originating in the grey matter about the floor of the aqueduct of Sylvius, pass to the lower cervical and upper dorsal region of the cord (cilio-spinal centre), emerge along with the two first dorsal nerves, join the sympathetic in the neck, and pass upwards to the cavernus plexus, and by the Gasserian ganglion and the ophthalmic division of the fifth, reach the nasal branch, and by it the ciliary ganglion, where they are joined by more branches from the cavernus plexus, and from thence reach the eye by the short ciliary nerve.

Lesions affecting the reflex contraction of the pupil may occur—1. In the centripetal part of the reflex arc, which includes the optic nerve, the chiasma, tracts and connecting

fibres to the cortex. If the lesion be on the left side, stimulation of the *left* eye by light fails to elicit either the direct or indirect reflex, while stimulation of the *right* eye produces a direct reflex in that eye and an indirect reflex in the *left*.

2. In the part of the reflex ring which carries the light impulses from the corpora quadrigemina to the oculo-motor nuclei (Meynert's fibres). There is then loss of reaction to light, but a normal reaction to accommodation and convergence (Argyll Robertson pupil).

3. The centripetal part of the ring, which includes the nucleus of the sphincter of the iris (the third nerve) and its terminations in the iris. If the lesion be in the left nucleus, both the direct and indirect light reflex action of the left pupil is lost. The pupils may possibly react to accommodation and convergence, and the indirect light reflex is present in the *right* eye on light stimulus being applied to the *left*.

If the trunk of the third nerve or its terminations be the seat of the lesion, in addition to loss of direct and

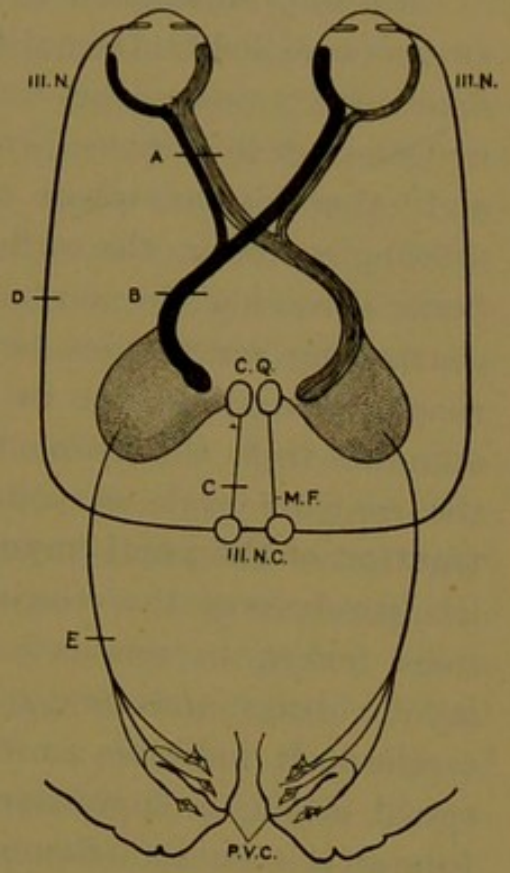


FIG. 137.—To illustrate the influence of different lesions on the pupil reflexes. A lesion at A in the left optic tract will cause loss of both the direct and indirect pupil reflex. A lesion at B will produce homonymous hemianopsia, and on stimulating the blind half of the retina there may be an absence of pupil reaction (Wernicke's sign). A lesion at E or in the primary visual centre P.V.C. will give homonymous hemianopsia without interference with the pupil reflex. A lesion in C (Meynert's fibres) will give loss of reaction to light without interference with the reaction to accommodation and convergence. A lesion at D will produce loss of direct and indirect reaction to light, and loss of reaction to accommodation and convergence.

indirect light reflex there is loss of the reaction to accommodation and convergence.

Clinically, **dilatation of the pupil (mydriasis)** is found in various so-called functional diseases—as neurasthenia, nervous depression; under the influence of mydriatic drugs; in irritation of the cervical sympathetic; in spinal meningitis, and other lesions where there is irritation of the pupil-dilating centre in the cord; in affections at the base of the brain involving the centre for the third nerve (the pupil-contracting centre), or its conducting fibres, as in hæmorrhage, tumour, etc.; or in failure in the conduction of the stimulus from the retina to the centre. Hæmorrhage into the centrum ovale or peduncles produces mydriasis. **Contraction of the pupil (myosis)** is found in congestion of the iris, paralysis of the sympathetic and of the fifth nerve, in some fevers, in venous congestion, under the influence of myotic drugs, and in cerebral disease. In affection of the cerebrum it indicates an irritative stage of the lesion; if of spinal origin, a depression or paralysis of the part. The former is seen in inflammatory affections of the brain and meninges, the latter in lesions of the cord above the dorsal vertebræ, as in tabes dorsalis. The pupil then shows the *Argyll Robertson phenomenon*; it reacts to accommodation and convergence, but not to light. The lesion is probably then in the fibres which pass from the proximal end of the optic nerve to the oculo-motor nuclei.

IV. CO-ORDINATING FUNCTIONS

The co-ordination of muscular movements is closely associated with the condition of the muscular sense, and is at the same time dependent on the integrity of the reflexes. The sensory and motor paths provide for impulses to and from the centres in the cord, which are probably under the influence of a higher centre, or of higher centres, in the ganglia at the base of the brain. The integrity of these

structures provides for the perfect co-ordination of all muscular acts, and disease results in inco-ordination.

The power of co-ordination is *to be tested* by making the patient perform different movements, and observing the manner of their performance. In regard to the lower extremities, the patient may be directed to stand with his feet close together and his eyes shut, to walk along a line with his eyes shut as well as open, and to walk backwards.

Impairment of co-ordination is specially characteristic of locomotor ataxy, in which it is due to disease of the posterior root zone of the cord.

Closely allied in some respects to disorders of co-ordination are *disturbances of the balancing power*, caused by interference with the centres concerned in the maintenance of the erect posture. In such cases the gait is staggering, and the patient cannot stand with his feet close together. This symptom differs from that above mentioned, in the fact that while the patient is in bed he can perform all kinds of movements without apparent loss of the power of co-ordinating muscular movements; and further, in the fact that there is no alteration at any time in the movements of the arms. Disturbances of the balancing power are found especially in disease of the middle lobe of the cerebellum.

V. VISCERAL FUNCTIONS

The most important visceral functions which may be disordered in affections of the nervous system are those connected with the circulation and the respiration, whose centres are situated in the medulla oblongata, as well as those concerned in defæcation, micturition and generation, which have their centres in the lumbar enlargement. All these functions are frequently classed under the term *organic reflexes.*

The **Circulatory functions** show alteration, for the most part, in changes in the rate and rhythm of the heart's action.

As a result of cerebral hæmorrhage, for instance, the pulse may be frequent and is often associated with a high temperature, while in consequence of a tumour or other intracranial affection the pulse may be very considerably retarded. Various forms of irregularity of the pulse are found in cerebral disorders, such as meningitis. All these symptoms are most marked when the disease is situated at the base of the brain near the medulla oblongata.

The **Respiratory functions** undergo modifications in rate and rhythm in consequence of cerebral disease. In exaltation of the cerebral functions the respirations may be extremely frequent, while the rate may be much lessened by lowering of these processes. The rhythm is often modified in cerebral affections. The respirations may become simply irregular in rhythm as well as depth, or the peculiar type of periodic breathing, already described as "Cheyne-Stokes respiration," may be present. Simple irregularity may occur in exalted or depressed conditions of the cerebral functions, but the "Cheyne-Stokes" rhythm only occurs when there is depression.

If there be paralysis of the diaphragm from disease affecting the phrenic nerve phrenic dyspnœa results. The dyspnœa is marked on exertion. Any straining effort, as in defæcation, becomes difficult. The voice is weak and there is difficulty in coughing and sneezing, as a full inspiration cannot be taken. On inspiration, instead of the natural expansion of the lower chest, the epigastric and hypochondriac regions are drawn in. During expiration they are pushed forward. The thoracic movements are thus reversed.

The **Functions of micturition and defæcation** may be disturbed in various lesions of the nervous system.

Micturition in the young child is a purely reflex action, depending on the tension of the fluid within the bladder. With advancing age the individual acquires more or less control over the reflex act. In the adult the process of

retention and evacuation of urine is modified and controlled by voluntary effort. There are two muscular mechanisms in opposition to each other,—the detrusor, the longitudinal muscular coat of the bladder, by contraction of which the viscus is emptied; and the sphincter, a muscular arrangement, by which the outflow of urine is prevented. The afferent or sensory path of impulses passes from the mucous membrane of the bladder to the centre in the fourth and fifth sacral segments of the cord. The centres in the cord for the sphincter and detrusor mechanism are controlled by the higher centres in the cerebral cortex.

When urine accumulates in the bladder, and the pressure rises, a stimulus travels from the mucous membrane to the centre in the cord, and from the centre in the cord to the brain, informing the individual that the bladder is full. If circumstances be favourable for micturition a message is despatched to the centres in the cord, which causes the sphincter to relax and the detrusor to contract, and the bladder is emptied, the flow of urine being accelerated by the voluntary contraction of the abdominal muscles. If, when the tension in the bladder is rising, the circumstances be not favourable for micturition, a stimulus passes to the sphincter centre to increase its action and so retain the urine in the bladder. In diseased conditions such a complicated mechanism may easily be thrown out of co-ordinate action.

Increased sensitiveness of the bladder mucous membrane, as seen in cystitis, will lead to frequency of micturition, a relatively small amount of urine being sufficient to stimulate the centres. Destruction of the peripheral nerves, as from neuritis, will lead to an opposite result, the bladder becoming overfilled without the individual's knowledge. If the cortical centres be dulled, as in coma, the individual no longer perceives when the bladder is full. A reflex act *may* result in evacuation as in the child, but more commonly the bladder becomes over-distended. When the pressure has

risen sufficiently high in the viscus to overcome the action of the sphincter, the urine dribbles away and incontinence results.

In lesions of the upper part of the cord, such as transverse myelitis, the path between the higher centres and the reflex centres is destroyed, and the patient no longer knows when the bladder is full, nor is conscious micturition possible. The act of micturition may be entirely reflex, the bladder regularly filling and emptying without the knowledge of the individual. More commonly this automatic action does not take place, the bladder becomes distended, the pressure within the viscus rising till the sphincter can no longer control the outflow; the urine then dribbles away until the pressure is relieved, then the sphincter regains control, the bladder thus remaining more or less distended. When the centres in the cord are themselves implicated in the lesion, both the detrusor and sphincter are paralysed; the urine dribbles away as soon as it reaches the bladder, the bladder remaining nearly empty.

The functions of defæcation may be affected in lesions of the nervous system in a similar manner to the functions of micturition.

The **Sexual functions** depend on the integrity of their centre in the lumbar enlargement. If the lower part of the cord is not under the control of the higher centres, the sexual reflexes are exalted, and *priapism* is the result. If the lower part of the cord is diseased and the sexual centre is involved, the sexual functions are *depressed or abolished*.

VI. THE CRANIAL NERVES

The olfactory, optic and auditory nerves will be considered in treating of the special senses.

The **third nerve,** or **oculo-motor,** arises in a nucleus situated in the anterior part of the grey matter surrounding the aqueduct of Sylvius, and immediately dorsal to the

posterior longitudinal fasciculus. The root fibres pass forwards to emerge in the oculo-motor sulcus on the inner side of the crus cerebri. The nucleus of the third nerve is capable of subdivision into a number of nuclei. Of these the median seems to subserve convergence; the postero-internal, accommodation and pupil contraction; the postero-lateral and external, the elevation of the globe and upper lids; while the anterior group sends fibres to the internal and to the inferior rectus.

Paralysis of the third nerve produces **ptosis**, or drooping of the eyelid, the pupil is semi-dilated and immovable, the power of accommodation is lost, the eyeball is often slightly protruded, owing to the backward traction of the recti being lost. Motion inwards exists, but to a slight degree; motion downwards is effected only by the aid of the superior oblique, and it is accompanied by a marked inward wheel-motion. If the paralysis be of some little standing, the external rectus rotates the eyeball permanently outwards. Paralyses of the third nerve are frequently partial; when only some of the fibres of the nerve are affected the diagnosis is at times extremely complicated.

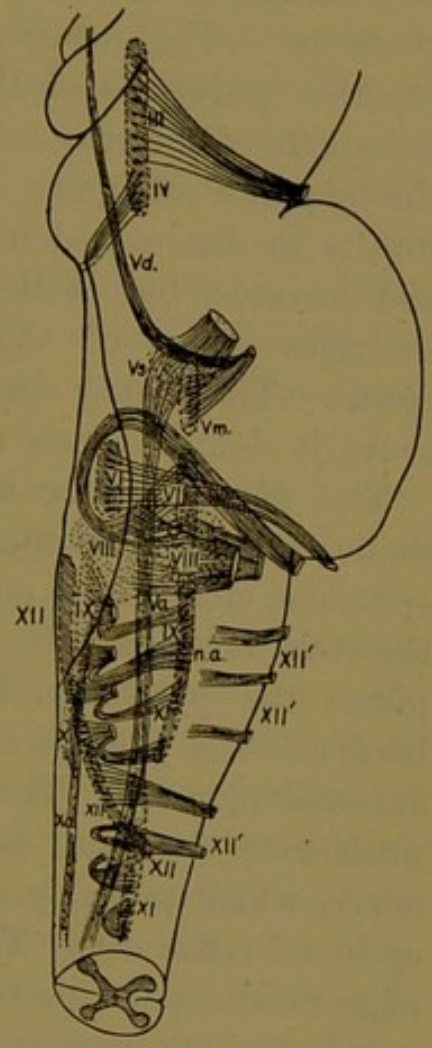


FIG. 138.—Diagram to show the situation of the chief nerve nuclei in the medulla oblongata and pons near the floor of the fourth ventricle.

The **fourth nerve** arises in a nucleus in the posterior aspect of the posterior longitudinal fasciculus, almost continuous with the lower end of the nucleus of the third

nerve. The root fibres pass first outwards as far as the inner aspect of the upper root of the fifth nerve, they then bend downwards, and below the posterior corpora quadrigeminum in the valve of Vieussens they decussate with the roots of the nucleus of the opposite side, and after decussation emerge on the anterior part of the roof of the fourth ventricle. The nerve then passes outwards across the superior peduncles of the cerebellum, forwards round the outer side of the crus cerebri, passing to the orbit to supply the superior oblique. Paralysis of the superior oblique results in diminished movement of the eyeball downwards and inwards, but at times is not easy to detect owing to vicarious action of the inferior rectus and the internal rectus. There may be upward and inward squint, and there is double vision on looking downwards.

The **sixth nerve** arises from a round nucleus which underlies the eminentia teres on the floor of the fourth ventricle, close to the median groove and immediately above the striæ acusticæ. Its fibres pass nearly directly forwards, pierce the pyramidal fibres and emerge below the lower border of the pons in the groove in the outer aspect of the anterior pyramid. The nucleus has connections with the nucleus of the third nerve and the nuclei of the auditory nerve, which probably subserve conjugate deviation of the eyes and reflex acts. The nerve, after emerging at the lower edge of the pons, passes through the sphenoidal fissure to supply the external rectus muscle. In paralysis of the external rectus there is inability to move the eyeball outwards, and there is double vision. There may be internal squint.

Clinically the recognition of paralysis of the ocular muscles depends upon—

1. **Deviation.**—When the motion of the eyeball is limited by paralysis of a particular muscle the condition is known as **primary deviation.** It is present on the affected side. **Secondary deviation** occurs from excessive contraction of the corresponding muscle on the healthy side.

2. **Diplopia,** or **double vision.**—The affected eye being deviated from its correct position, and being more or less incapable of associated motions with the other eye, the image of the object looked at is not formed on identical spots of the retina in each eye, and hence the object seems doubled.

3. **Indistinct vision.**—If the paralysis be slight, actual diplopia may not be present, but the double images overlapping each other will produce dimness or confusion of sight.

4. **Vertigo,** due partly to the diplopia and partly to faulty projection of the object.

5. **False orientation.**—The deviation results in a false idea of the position of surrounding objects, and if the paralysed eye be used in walking, a zigzag course may result.

6. **Altered carriage of the head.**—The patient carries the head in such a position that the diplopia is reduced to a minimum.

Of these signs diplopia is the most important, and unless the limitation of the ocular movements is so marked as to leave no doubt as to which is the affected muscle, it is necessary to test the diplopia by means of the **method of double images.** The patient is seated in a dark room with a red glass placed over one of the eyes in order to facilitate the identification of each image by its colour, and a lighted candle is held on a level with the head about 15 ft. off. Two images are seen. The candle is now moved upwards, downwards, to the right and to the left, and the relation which the two images bear to each other in the different meridians of the field is noted. The first point to decide in making the examination is, which is the true and which is the false image? It will be found that the diplopia is most manifest when the enfeebled muscle is most called upon to exert itself; the diplopia will, in fact, be most manifest when the object is at one of the meridians of the field—upwards, downwards, inwards, or outwards. As the demand

upon the enfeebled muscle increases, and it cannot meet the demand, the separation of the two images becomes wider. If, then, the diplopia be most manifest at one meridian of the field, say the lower, the image which is farthest in the direction in which double vision exists, in this case the lower image, is the image given by the affected eye—the false image.

Which muscle is affected? *The false image is deviated exactly in the direction of action of the paralysed muscle.* A knowledge of the action of the ocular muscles is naturally necessary in determining which is affected. The false image may be deviated in the vertical plane—it is then an elevator or depressor muscle which is at fault. It may be deviated laterally—it is then an inward or outward mover which is paretic. It may be rotated, the muscle at fault is then a rotator of the eyeball.

Paralysis of the third, fourth and sixth nerves may be either peripheral or central. Paralysis from peripheral causes embraces local affections of the orbit. Rheumatism, gout, diphtheria, influenza and other toxic processes may produce a neuritis with paralysis. Affections of the nerves in their course, by the pressure of tumours and by meningeal inflammation, may occur. The third nerve, having a longer and more exposed course, is more liable to such interferences.

Affections of the nuclei of the nerves, resulting in paralysis, is known as **Ophthalmoplegia.** It may be acute, when it is analogous to the acute poliomyelitis anterior of the spinal cord. It is seen as a chronic lesion in locomotor ataxia, general paralysis of the insane, chronic bulbar paralysis, multiple cerebro-spinal sclerosis, progressive muscular atrophy and syphilis. Paralysis of ocular muscles is at times cortical in origin.

Conjugate deviation of the eyes to one side or other is normally presided over by the nucleus of the sixth nerve on one side, which by its connections influences the centre of the third nerve on the opposite side, which centre in turn

governs the internal rectus muscle. The whole mechanism is under control of the higher centres in the cortex. Say that under ordinary circumstances a sound is heard, an impulse goes to the motor centres in the cortex, from thence to the centre of the sixth nerve, which influences the centre of the third nerve on the opposite side—contraction of the external rectus on one side, and of the internal rectus on the other, follows, and the eyes are turned to the side. If a lesion interrupts this arc, the eyes in a destructive lesion are turned away from the paralysed side; for example, in cerebral hæmorrhage resulting in *left* hemiplegia, if there be conjugate deviation, the eyes would be turned to the *right*, the right external rectus and the left internal rectus overcoming the corresponding paralysed muscles. If the lesion be, on the other hand, an irritative one, the eyes will be turned towards the affected side.

In lesions of the pons resulting in conjugate deviation the direction of the eyes is the converse of that seen in cerebral lesions,—in paralytic lesions towards the paralysed side, in irritative lesions towards the sound side.

The **fifth nerve** has a wide origin in the medulla and pons, and has two roots, a motor and a sensory. The motor root consists of the superior root and the motor root proper. The superior root arises in the grey matter around the aqueduct of Sylvius, the fibres pass downwards to emerge with the motor root, which latter arises from a group of cells placed a little above the root of the seventh nerve, near the lateral angle of the fourth ventricle, and emerges from the lateral aspect of the pons about the middle of its vertical extent. The sensory root has a very extensive origin, which begins below, at the level of the second cervical root, and passes upwards through the cervical cord and medulla to the level of the motor nucleus of the nerve, where it bends outwards and emerges as a broad band of fibres external to the motor root. The two roots pass forwards to the middle fossa at the base of the skull,

over the summit of the petrous portion of the temporal bone, where the sensory root breaks up into a plexus to form the Gasserian ganglion. From the ganglion pass the three sensory trunks—the ophthalmic, superior maxillary and inferior maxillary. The first two are from the ganglion alone and are purely sensory, the third is joined by the motor fibres and is thus both sensory and motor.

The distribution of the nerve is **sensory** to the face, the fore-part of the head, the eye, the nose, the ear, the tongue, the gums, the teeth, the soft palate, and the upper part of the pharynx. It also supplies the salivary glands.

The **motor** distribution is to the muscles of mastication and to the tensor tympani. Lesions of the nerve may be irritative or destructive. Irritative lesions will result in the form of cramp, usually known as **trismus**, due as a rule to central disease. When the sensory fibres and Gasserian ganglion are implicated **tic douloureux** results.

Destructive lesions give loss of sensation over the area supplied, various trophic lesions, particularly ulceration of the cornea, diminution in the secretion of saliva and motor paralysis of the muscles of mastication and of the tensor tympani. Motor paralysis can be ascertained by placing the fingers upon the temporal and masseter muscles while the patient clenches the teeth. The muscles should stand out with equal prominence on both sides; when paralysis is present, there is loss of prominence on the affected side. When the patient opens the mouth the jaw is deviated towards the *paralysed* side, being pushed over by the sound external pterygoid muscle. Paralysis of the tensor tympani cannot be recognised, as was at one time supposed, by difficulty in hearing notes of a particular pitch. In suspected cases of paralysis of the fifth, the sense of taste should always be investigated, as most, if not all, the taste fibres reach the brain by way of the fifth nerve.

The **seventh nerve**.—The facial nerve arises in a nucleus in the anterior part of the tegmentum in the lower part of

the pons, near its junction with the medulla; the root fibres pass backwards and inwards round the nucleus of the sixth nerve, and then forwards, to emerge at the lower border of the pons immediately internal to the auditory nerve. The nerve passes outwards, in company with the auditory nerve, to the internal auditory meatus to enter the aqueduct of Fallopius and pass through the temporal bone to issue by the sterno-mastoid foramen. In its passage through the aqueduct it gives off a branch to supply the stapedius muscle, and is joined by the chorda tympani, which contains taste fibres from the anterior two-thirds of the tongue. As the nerve is enclosed in a bony canal—the aqueduct—it may be exposed to the effects of compression in this part of its course. After emerging at the mastoid foramen the nerve passes through the parotid gland, and a little behind the ramus of the lower jaw terminates by dividing into two parts, from which numerous branches pass to supply the muscles of the head. It is the principal motor nerve of the head, supplying, as it does, the muscles of the scalp, external ear, nose, mouth, and eyelids (with the exception of the levator), and the cutaneous muscle of the neck. It also supplies the muscles of the tympanum with the exception of the tensors, and in the neck the stylo-hyoid and posterior belly of the digastric.

The nerve is purely motor in function. Destructive lesions produce paralysis of the muscles supplied. The folds disappear from the affected side of the face, the angle of the mouth droops, and there is inability to close the eye, and the eye is more widely open. The patient cannot whistle, and, on smiling, the sound side of the mouth is drawn upwards. Food is apt to collect between the teeth and the gums.

The nerve may be affected by lesions in different parts of its course. In lesions above the nucleus of the nerve, in **facial paralysis of cerebral origin**, the lower part of the face is most affected. The orbicularis palpebrarum muscles

are so constantly acting in unison that probably each is supplied from both sides of the brain, and thus a unilateral lesion only partially cuts off the nerve impulses to one side.

Infranuclear paralysis may result from lesions of the nerve trunk before it enters the aqueduct, in the aqueduct, or in its peripheral course. A lesion of the nerve, before it enters the aqueduct, involves the branch to the stapedius, causing paralysis of the stapedius with **hyperacusis**, that is, excessive sensitiveness to loud sounds. A lesion in the aqueduct will, unless it be at the outer end, involve the fibres of the chorda tympani nerve, giving loss of the sense of taste in the anterior two-thirds of the tongue on the affected side. A peripheral lesion causes facial paralysis without hyperacusis or loss of the sense of taste.

The **ninth (glossopharyngeal)**, the **tenth (vagus)**, and **eleventh (spinal accessory) nerves** are arranged on the same plan, and arise along the whole length of the medulla. There are three roots and three common nuclei: (1) A motor root arising from the nucleus ambiguus in the middle of the formatio reticularis; (2) a dorsal root arising from a nucleus of small cells near the hypoglossal nucleus; (3) a sensory root which forms a rounded bundle, external to the dorsal nucleus in the upper medulla and behind it in the lower.

The **glossopharyngeal nerve** is sensory for the posterior third of the tongue and for the mucous membrane of the pharynx. It is motor for the middle constrictor of the pharynx and for the stylo-pharyngeus. In part of its course it contains the taste fibres for the posterior third of the tongue. The nerve is rarely paralysed alone. In paralysis the pharyngeal reflex is abolished. Taste in the posterior third of the tongue may be lost if the lesion is in the course of the nerve, but is not affected in nuclear lesions.

The **vagus** is the motor nerve of the palate, pharynx and

larynx. It is also the sensory and motor nerve of respiration, for the heart and through the sympathetic ganglia, for most of the abdominal viscera. Lesions of the palatine branches result in paralysis of all the muscles of the palate.

There is then insufficient elevation of the soft palate during swallowing, and fluids are apt to regurgitate through the nose on attempting to swallow. At the same time, the pronunciation of certain words which demand the closure of the naso-pharynx becomes altered. The speech takes a nasal snuffle. On examining the naso-pharynx the soft palate is seen to be motionless when the patient attempts to say "Ah."

In unilateral paralysis, regurgitation of fluids is not present, nor is the voice altered. On phonating, the palate is deflected by the action of the sound muscles.

The **spinal accessory** consists of two parts, the one joining the vagus and sending its fibres to the pharyngeal and superior laryngeal nerves, the other passing to supply the sterno-mastoid and trapezius. Paralysis of the sterno-mastoid causes difficulty in rotating the head towards the unaffected side. Paralysis of the trapezius is shown by inability to shrug the shoulder.

The **twelfth or hypoglossal** arises in a nucleus in the lower part of the floor of the fourth ventricle, and emerges by a series of roots in the groove separating the anterior pyramid from the olivary body. The nerve supplies, either alone or in union with other branches of the spinal nerves, all the muscles connected with the hyoid bone, including those of the tongue, but with the exception of the digastric, stylo-hyoid, mylo-hyoid and the middle constrictor of the pharynx. It also supplies the sterno-thyroid.

The hypoglossal nerve is motor for the tongue. If there be unilateral paralysis the tongue, on being protruded, is pushed to the paralysed side by the healthy muscles. If

the lesion which causes the paralysis be nuclear or infra-nuclear, the tongue may show wasting on the paralysed side.

VII. VASOMOTOR FUNCTIONS

Disturbances of vasomotor functions manifest themselves in a variety of different ways.

Changes in the **temperature** occur in many cases, the departure from the normal being most commonly a rise, but not infrequently a fall. These alterations are local or general, according to the cause.

Modifications in the **colour of the skin** are also frequent —flushing and pallor being seen over larger or smaller regions of the surface.

Alterations in the **amount of perspiration** may also be present in the form of excess or deficiency. These changes may be local or general. It is open to question whether in such symptoms there is not also a disturbance of the secretory nerves.

In addition to these external symptoms of vasomotor disturbance, there may be others connected with secretory apparatus, in which, however, it is possible that there is, along with the vascular change, some interference with the nerves of secretion.

Excessive or modified secretion from the **lachrymal glands** may be found in certain affections.

Excessive or modified secretion from the **salivary glands** may be observed in some cases.

Changes in the **amount of the urine** are common—*oliguria*, or a deficient flow, being observed, as well as *polyuria*, or an excessive flow.

There may be alterations in the **character of the urine**, the most common of these being *albuminuria*, dependent on some transitory affection of the vessels and epithelium of the kidney, resulting from vasomotor disturbance, and *glycosuria*, produced by some interference with the vascular

supply to the liver, which is usually brought about by affections involving the hepatic vasomotor centre in the medulla oblongata.

These vasomotor changes are extremely frequent in cases of hysteria, but, as above mentioned, they are also found as the result of structural lesions.

VIII. TROPHIC FUNCTIONS

Interference with trophic functions is frequent in nervous disease.

Changes in the **nutrition of the skin** may result from functional or organic disease. Various skin rashes, as well as atrophies and hypertrophies of skin appendages, may be found without any definite organic disease of the nervous system, but they are often, on the other hand, associated with them.

Sloughing of the skin is frequent in such grave structural changes as myelitis.

Atrophy of the hair and nails may be observed in some cases where there is some local or general disturbance of the nervous system.

Wasting of the muscles occurring rapidly, as already referred to, shows some disease of the multipolar cells of the anterior cornua of the cord.

Deformities of bones and joints are frequently found in connection with locomotor ataxy.

IX. MENTAL FUNCTIONS

In considering disturbances of the mental processes we have to pay attention to sleep, consciousness and speech.

Sleep.—The states of sleeping and waking may be modified to excess in each of these two directions, the tendency to excess of sleep being termed *somnolence*, while the opposite is *insomnia*. Such departures from the normal may be only

a slight exaggeration of an individual idiosyncrasy, or may result from nerve-exhaustion, or may mark the presence of some definite lesion: the cause of such changes must be determined by a consideration of all the associated symptoms.

Consciousness.—The condition known as consciousness, which we may for clinical purposes assume to be the outward manifestation of perceptive processes, undergoes more or less entire abolition or perversion in many cases. When the patient gives no evidence of perception spontaneously, or in response to external stimuli, he is said to be *unconscious*. There are degrees of unconsciousness: when partial it is termed *stupor*, when profound it is called *coma*. When the patient lies in a state of deep unconsciousness, with the eyes open, the condition is termed *coma-vigil*. In stupor the reflexes are not entirely abolished; the iris contracts in response to a bright light, and a painful stimulus will induce movement. In coma, on the other hand, all superficial reflex functions are abolished. These states are frequently accompanied by some of the alterations of the organic reflexes, which have already been described, such as changes in the circulation and respiration.

Sometimes, from the symptoms present and their mode of onset, it is easy to diagnose the cause of the unconsciousness; at other times it is extremely difficult. The loss of consciousness may be the result of some organic lesion of the cerebrum, such as hæmorrhage or inflammation; of some explosive affection, such as epilepsy; of some stoppage of the circulation, such as syncope as a general or embolism as a local cause; or of some toxæmic condition, as in fevers, uræmia or narcosis. For a full discussion of the means by which these different causes may be diagnosed, special works must be consulted. The diagnosis must turn on the entire group of symptoms, not on one alone, and the mode of invasion is of high importance.

The perversions of consciousness presented in disease may be grouped together under the term *delirium*. This term is

usually restricted to those symptoms which result from some of the causes which have just been referred to in the preceding paragraph, and it corresponds to the condition known in psychological medicine as *insanity*. Delirium is more common in such conditions as fever and narcosis than in cerebral disease.

There are three well-marked classes of mental conditions common in delirium. The impressions made upon the senses may arouse false sensory perceptions, which are termed *illusions*; or sensory images may arise without any impressions upon the senses, which are termed *hallucinations*; or false ideas may be present, which are termed *delusions*.

Speech.—Disturbances of speech, using this term in its widest sense, present one of the most difficult problems which have to be dealt with by the physician. A patient may be unable to speak on account of disorders of the mental processes, or from affections of the muscles concerned in articulation. With such alterations of the faculty of conveying ideas we have nothing to do in this place. We have to confine ourselves to disorders of speech unconnected with mental disturbances, and independent of muscular affections.

In order to grasp the bearings of this subject, attention must be bestowed upon the perception as well as the production of spoken and written language, *i.e.*, the employment of conventional symbols as a basis for the communication of ideas. There are receptive as well as emissive functions connected with language.

Disorders of the faculty of communicating thoughts, arising from defect of the cerebral speech processes, are classed under the term *aphasia*. Four cortical centres in the brain, with connecting tracts, are recognised as being instrumental in the understanding and production of speech. Two are sensory and two are motor. All four are situated in the left hemisphere in right-handed persons; in the right hemisphere in

left-handed persons. The positions of these centres is shown in Fig. 139.

A. The posterior half or three-fourths of the first left temporal convolution is the **auditory speech centre,** in which are stored the auditory speech images. Here memories of the sound of words are stored, and here language when heard is interpreted and understood.

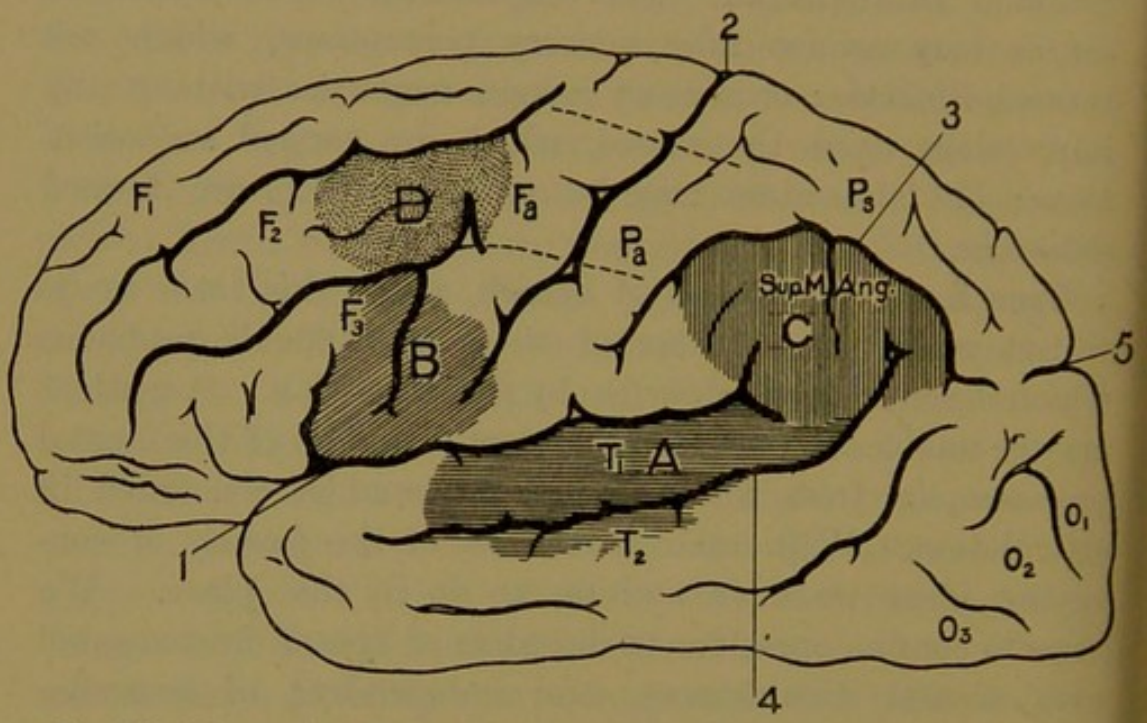


FIG. 139.—The cortical centres for the understanding and production of speech.

A. Auditory speech centre.
B. Broca's convolution.
C. Centre for visual images of written or printed speech.
D. Graphic motor centre.
F_1, F_2, F_3, Fa. Frontal convolutions.
Pa. Ascending parietal.
Ps. Superior parietal.
T. Temporal.
O. Occipital.
1. Fissure of Sylvius.
2. Fissure of Rolando.
3. Interparietal fissure.
4. Parallel fissure.
5. Parieto-occipital fissure.

B. Broca's convolution is the centre for the **motor memories of speech**: in it the motor impulses of speech originate.

C. The angular and supra-marginal convolution, the centre for the **visual images of written or printed speech,** where the faculty of understanding and interpreting written language is located.

D. The posterior extremity of the second frontal convolution is the **graphic motor centre,** where are stored the motor memories which guide the right hand in the act of writing.

Aphasia then results from a lesion of one or more of these centres or their communicating tracts.

Four principal forms of aphasia are recognised.

1. **Auditory aphasia,** due to the destruction or disablement of centre A. The memories of word sounds are lost; the sounds of spoken language no longer convey a meaning: they are sounds and nothing more. It is doubtful if the patient in whom destruction of centre A has taken place can read even though C be intact, for C appears to require the aid of A to grasp the meaning of words seen. Further, destruction of centre A has an effect on the production of speech. Words can no longer be revived as auditory word images for the expression of ideas, and the power of expression is damaged. There is failure of the power to recollect words—**Amnesia verbalis,** and this is especially noticeable in the case of nouns. Destruction of A also results in "**Paraphasia,**" the patient uses a word which he did not intend to employ, and being "word-deaf" is unable to recognise and rectify the error. "**Articulative amnesia**" shows itself by the word being remembered in a general way, but the articulative details are forgotten, so that the word as produced is barely recognisable. Again, writing is a mere representation of internal speech, and the same amnesia verbalis, paraphasia and articulative amnesia appear in the patient's writing as in his speech. This is usually termed **Paragraphia.** There is in addition, in destruction of centre A, loss of power of repeating words and copying written words.

2. **Motor aphasia,** frequently called **Aphemia,** results from destruction of centre B. The power of calling up motor images and of originating word utterances is destroyed. The patient is thrown for verbal expression on the scanty resources of the uneducated centre in the opposite cortex

which cannot rise above "yes and no," or, it may be, emotional utterances, such as oaths. The patient can understand what is said to him, but he cannot express his thoughts in writing, nor can he understand written language, for he cannot call up the motor images of words. Written or printed language can be copied: if the right hand, as frequently happens, is paralysed, the left with a little practice becomes efficient in copying. The connecting fibres between the centres A and B run under the fissure of Sylvius (Fig. 140), and if destroyed in their course the lesion gives rise to a peculiar form of aphasia called "**Conduction aphasia.**" The centres are intact, but their communicating fibres are destroyed and paraphasia is produced; the patient in expressing himself uses the wrong word.

3. **Visual aphasia** is due to the effects of a lesion in centre C, that is, of the supra-marginal and angular gyri. There is destruction of the visual memories of words, and thus "word-blindness" and loss of the power of reading (**Alexia**). There is also loss of the power of writing, for the power of calling up the visual images of letters and words is destroyed. There may, however, be word-blindness without agraphia—the patient can write correctly, but he cannot read even that which he himself has written. The lesion then is not in the centre C, but in the connecting fibres between it and the primary optic centres in the occipital lobe (*vide* Fig. 140). A lesion of the left primary optic centre, or in the radiation of Gratiolet, will produce right homonymous hemianopsia (*vide* p. 397). If, in addition, the lesion divide not only the fibres connecting the left primary optic centre about the calcarine fissure, but also the fibres connecting the right primary optic centre with the centre C, then there will be complete word-blindness as well as homonymous hemianopsia. The centre C is still intact, and therefore the patient will be able to call up the visual memories of words, and will be able to write.

In some rare cases of word-blindness there is, in addition,

"mind-blindness,"—the patient not only fails to recognise words when he sees them, but also fails to recognise familiar

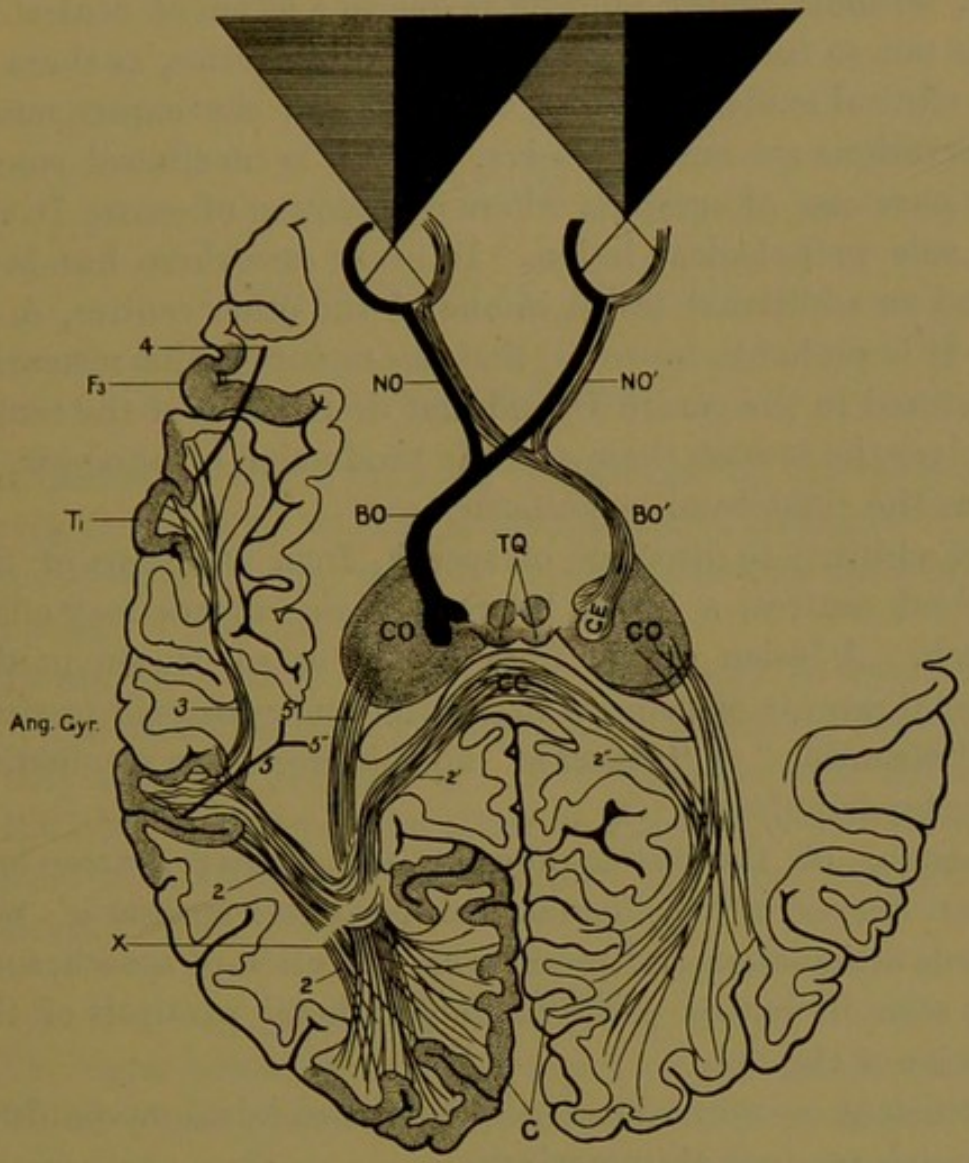


FIG. 140.

NO, NO'. Optic nerves.
BO, BO'. Optic tract.
TQ. Corpora quadrigemina.
CC. Corpus callosum.
C. Cuneus.
F_3. Broca's convolution.
T_1. Superior temporo-sphenoidal (auditory speech centre).
1. Optic radiation.
2. Fibres connecting angular gyrus with left primary visual centre about calcarine fissure, and through 2' and 2'' with right primary visual centre through corpus callosum. A lesion at X cuts the connection of the left primary visual centre with the angular gyrus and the optic radiation, and thus causes right lateral homonymous hemianopsia, with word-blindness but no agraphia.
3. Fibres connecting the first temporal convolution with the angular gyrus.
4. Connection between the first temporal and Broca's convolution.
5. Connections of the angular gyrus by 5' with the motor region of the left cortex, by 5'' with the motor region of the right cortex.

objects. He cannot recognise the face of a familiar friend or the most familiar object.

4. **Graphic motor aphasia** is due to a lesion of centre D. It is not so fully understood as the other forms, as there is less clinical evidence bearing upon it, and the experimental observations are not conclusive. There is no clinical record of a pure case of agraphia where destruction of centre D was the sole pathological lesion. In every case there has been found an additional lesion in one of the other centres, A or B. It is probable, however, that the motor graphic memories are stored in the centre D, and that destruction of the centre involves the loss of these and the production of agraphia, as far as the right hand is concerned.

In addition to disorders of speech, from affections of the cerebral centres, a lesion between these centres may affect speech. A lesion of the fibres of the upper neuron in the internal capsule or pons, as in multiple sclerosis, will produce the "**scanning**" or "**staccato**" speech, from slow conduction of motor impulses.

Lesions of the lower neuron, the bulbar centres and motor nerves of oro-lingual and laryngeal apparatus, will impede articulation and produce an alteration of speech, such as is seen in bulbar paralysis or peripheral paralysis of the muscles of the lips.

Deficient co-ordination of the vocal and labial mechanisms of speech produce **stammering.**

Slurring speech is seen as the result of alcoholic intoxication and in general paralysis of the insane.

Lalling or **baby speech** and **syllable stumbling** are also at times met with in disorders of the speech mechanism.

CHAPTER XI

THE SPECIAL SENSES

Sense of Smell

The sense of smell, entirely subserved by the olfactory nerves, is liable to *subjective* modifications, pleasant or unpleasant odours being present in some cases as auræ, or as symptoms of cerebral disease.

To test the sense of smell, substances should be applied to each nostril, taking care that no pungent bodies are employed, as they affect the fifth nerve. If there be no local affection of the nose, changes of the sense of smell may be found in organic disease of the brain, but they are more common in hysteria.

Sense of Sight

The optic sense, or sense of sight, is the most complex of all the sensory functions which we have to consider. Its investigation in disease is rendered still more complex by the necessity for distinguishing between the defects caused by morbid conditions in the external organs of vision (the eyes), and those arising peripherally or centrally in the optic nerve or tract.

The examination of the sense of sight should therefore be conducted objectively and subjectively, and the particular character of each case will largely determine which of these methods should be first pursued. As a general rule it is always desirable to commence with a thorough objective examination.

Objective Examination of the Eye

The objective examination of the eye is necessarily limited to those parts which we can handle or see, so that the methods at our service are Palpation and Inspection.

The former is too often neglected by the physician, whether because, as compared with the latter, it appears to yield little information, or because its performance occupies so little time that it does not impress his mind as a student, we shall not stay to discuss; but we give it the first place here, lest by relegating it to the close of the optical examination we help to perpetuate that neglect through which too many eyes are now sacrificed.

Palpation.—The chief application of this method is found in the determination of the state of the intraocular tension. The fluid contents of the eyeball are constantly being changed by secretion of aqueous humour from the ciliary processes and back of the iris, and by the escape of this fluid into lymph spaces at the periphery of the anterior chamber, and in slight amount at the optic disc.

In health the inflow and outflow are so balanced that the contents of the globe practically maintain a constant quantity, and, exerting a uniform pressure upon the coats of the eye, offer an elastic sense of resistance to compression. This resistance is called the *tension* of the eye, and it increases or diminishes directly as the contents of the globe. It corresponds to what surgeons term "fluctuation."

To elicit it, direct the patient to look downwards without lowering his head, and to close his eyelids gently.

The observer steadies the globe against the floor of the orbit by lightly resting the pulp of one index finger on the upper lid; with the other he makes gentle intermittent pressure upon the eye, and mentally compares the sense of resistance with that offered by a normal eye.

The patient's two eyes should be contrasted with one

another, and if in doubt, with those of a healthy person. In some persons the globes feel much firmer than in others; allowance must also be made for the thickness and softness of the lid.

Following Bowman's suggestion, it is convenient to express the results obtained thus :—

T.n. = tension normal.
T + (?) = ,, increased probably.
T + 1 = ,, ,, certainly.
T + 2 = ,, ,, but globe can be dimpled.
T + 3 = stony hardness.
T − (?) = tension diminished probably.
T − 1 = ,, ,, certainly.
T − 2 = ,, much diminished.
T − 3 = globe flaccid.

Increased tension is intimately related to that morbid condition of the eye known as glaucoma, and is a sign of the gravest import, since it is usually associated with marked deterioration of vision, and is capable of inducing blindness with great rapidity. The tension undoubtedly changes in some persons at short intervals. In the case of adult persons, with or without external signs of inflammation of the eyes, complaining of failure of vision, and especially of paroxysmal pain, and particularly when mydriatics are being employed, the tension should be carefully examined *at each visit*. For want of this precaution too many eyes are treated for supposed neuralgia, or iritis, or cataract, and the surgeon's aid is not invoked until too late.

Diminished tension usually indicates an unnaturally fluid state of the vitreous, following escape or shrinking of that body, and this again is frequently associated with detachment of the retina.

Various mechanical tonometers (*i.e.* tension measurers) have been devised, but their practical utility is scarcely yet established. Probably the educated sense of touch will

always be the most accurate, as it certainly is the most ready, method of tonometry.

Palpation with one or more fingers is also of value in revealing increased or diminished sensitiveness of the globe to pressure, and often throws light upon the seat, nature and extent of new growths or inflammatory changes affecting the position of the eyeball, or modifying its shape.

In retrobulbar neuritis some sense of pain may often be elicited by pressing the eyeball backwards into the orbit. This may be best done by steadying the back of the patient's head with one hand, and gently pressing upon the closed lids with the pulp of the thumb of the other hand, held with the palm against the patient's temple. The same means may be employed to determine whether proptosis of the eyeball is due to a solid new growth in the back of the orbit, or some more compressible and less serious tissue alteration, such as vascular engorgement.

Inspection.—We have now to determine objectively the state of the refractive media—cornea, aqueous, crystalline lens, and vitreous—first as to—

(*a*) Transparency,
(*b*) Refractive power;

and secondly, the condition of the coats of the eye viewed from within.

(*a*) **Transparency.—Simple inspection.**—For the examination of the *anterior segment* of the eye the patient should be placed facing a window or other good source of light.

The illumination can be heightened by focussing the light upon the eye by means of a lens.

Bright sunlight should of course be avoided, since the heat rays are focussed as well as those of light. If the room be darkened, a bright steady gas, oil, or candle flame may be used, and should be placed at a distance of at least 18 in.

from the side of the patient's head, and slightly in front of the plane of his face, so that the light falls obliquely upon it.

Oblique focal illumination.—The observer, steadying his hand by resting the little finger on the patient's cheek or temple, should support a lens of 2 or 3 in. focus between his thumb and forefinger, and with it concentrate the light upon any desired spot. By moving the lens nearer to or farther from the eye, the light can be brought to bear on any part of the anterior segment of the globe, as deep as the posterior aspect of the crystalline lens and anterior layers of the vitreous.

Whether employing simple or oblique focal illumination, the observer may aid his sight by holding another lens as a magnifying glass between his own eye and that of the patient. Opacities in the cornea, aqueous humour, and lens should be carefully sought for.

To explore the *posterior segment* of the eye, the ophthalmoscope is required.

The Ophthalmoscope.—Every pencil of light which enters the eye through the pupil is bent by the refractive media towards a focal point on or near the retina. In like manner the rays which escape from the interior of the eye are, by the same relation of the media to the outer air, caused to return almost along the directions by which they originally came. It is therefore impossible for the eye of an observer to receive the returning rays without at the same time intercepting those which are passing to the observed eye, or, in common phraseology, "putting his head in the light."

Hence the pupil appears black, or so few rays reach the observer's eye that at the most only a faint reddish glow, and no satisfactory picture of the interior, can be obtained.

By placing an artificial light behind the plane of the patient's face, and using a mirror to reflect rays into his eye, the mirror becomes the apparent source of light, and the

returning rays tend to be restored towards it. A small aperture at the centre of the mirror allows some of them to enter an observer's eye if placed immediately behind it.

What will now be seen will depend on the size of the pupil, the transparency of the media, their refractive power, and the distance of the observer from the eye observed.

In a normal eye, at 18 in. distance, a beautiful rosy-red glow will appear through the pupil, and is due to the light being reflected from the very vascular choroid and retina. The tint will vary greatly with the complexion of the individual and the amount of pigment present in the hexagonal epithelial layer of the retina and the connective tissue stroma of the choroid.

If the pupil be small it may be dilated by instilling a drop of a watery solution of hydrobromate of homatropine (4 gr.—1 oz.) twenty minutes before the examination. The mydriatic effect of this substance asserts itself rather more quickly than a corresponding application of atropine, and passes off more rapidly. In many persons a few drops of a solution of the hydrochlorate of cocaine (4 per cent.), applied at intervals of a few minutes, is an efficient mydriatic, and has the advantage of not impairing the power of accommodation.

The patient should now be requested to look successively upwards, downwards, to right and left, and finally straight forward, while the position of his head remains unaltered.

In this way any *fixed* opacity in the vitreous (as well as any corneal nebula or striæ in the lens) will probably be brought into view, especially if the observer take care to examine the peripheral parts by moving his own head in a contrary direction to that taken by the patient's eye.

If the same movements be rapidly repeated by the patient any *free* opacities which have sunk to the bottom of the vitreous will be thrown upwards, and be seen floating or falling as dark dots, threads, or cloudy masses. Brilliantly

glittering crystals of cholesterin are sometimes seen. All such opacities indicate an unhealthy state of the vitreous.

By placing a lens of suitable strength behind the sight-hole of the mirror and approaching within two inches of the patient's eye, we can examine the vitreous at any plane, scrutinise in detail any fixed opacity, and tell its distance from the retina.

(*b*) **Refractive power of the ocular media—Refraction.**—If a ray of light travelling in one medium (*e.g.* air) enters another of different density (*e.g.* glass or aqueous humour) obliquely, it is bent from its original direction, or undergoes what is termed "refraction."

The refractive power of a medium depends on its density and the relation of its surface to the adjacent medium. In physiological optics the media with which we have to deal are air, glass and the transparent tissues of the eyeball; the last two being in the form of lenses.

The strength of a lens is expressed in terms of its refractive power on a standard pencil of light (namely, one composed of parallel rays) travelling in air.

A Dioptre.—In physiological optics *a lens which can bend parallel rays to a focus at a distance of one metre from its centre* is taken as the unit of lens measurement, and is called a lens of 1 *dioptre*. As the meaning of this term is somewhat perplexing, we may explain its origin. In the older works on optics, matters relating to the *reflection* of light were discussed under the heading "Catoptrics" (κάτοπτρον = a mirror), while those relating to *refraction* of light were termed "Dioptrics" (διόπτομαι = to look through). Hence the unit of dioptric (refractive) measurement has been called a dioptre.

If a lens has only half the refractive power of the unit lens its focal point will be at twice the distance, namely, 2 metres. If it be twice as strong as the unit lens, the focal point will be at half the distance; if thrice as strong, at a third of the distance, and so on.

Only convex lenses can produce *positive, i.e.* real foci. A concave lens causes divergence of rays, but its refractive power can be expressed in terms of its *negative* focus, *i.e.* the point from which it makes parallel rays appear to come.

Lenses are accordingly numbered thus :—

Convex—

+0·25 D.	Focal distance,	4 metres,	*or*	(roughly) 160 in.
+0·5 D.	,,	2 ,,	,,	80 ,,
+0·75 D.	,,	1·5 ,,	,,	60 ,,
+1 D.	,,	1 metre,	,,	40 ,,
+2 D.	,,	0·5 ,, or 50 cms.	,,	20 ,,

And so on.

Concave—

−0·25 D. −0·5 D. −0·75 D. −1 D. and so on.

These have the same *negative* focal distances as the *convex* lenses of corresponding strength.

Spherical lenses are those whose surfaces are arcs of spheres, and have the same refractive power in every meridian.

Cylindrical lenses are curved about one axis. Rays which enter such a lens in planes parallel to the axis of the cylinder pass through unchanged. Those in planes at right angles to the axis meet with curved surfaces and undergo refraction. Cylindrical lenses may be convex or concave, and are numbered like sphericals. In writing we indicate the difference thus (say), +1 D sph. or +1 D cyl., −1 D sph. or −1 D cyl.

The refractive media of the eye form a combined system of lenses whose principal focal distance in a normal eye is 22·231 mm. (equal to a single lens of about +45 D sph.). The cones of the retina occupy the focal plane at the fovea lutea.

Under the subjective examination are described the refractive errors which may be met with, and their correction by subjective means.

There are various objective methods of greater or less accuracy whereby they may also be detected, such as—(1)

the shadow test (retinoscopy); (2) the apparent direction of movement of the optic disc or blood vessels when seen with the ophthalmoscopic mirror alone at some distance from the patient's eye; (3) the lens required to give the clearest image by the direct method of ophthalmoscopy; and (4) the alteration of the size of the optic disc when the object lens is withdrawn or advanced slightly in the indirect method of examination.

The Shadow Test, *Retinoscopy*, *Pupilloscopy*, *Keratoscopy*, or the *Flash Test* are identical terms. If a beam of light be thrown with an ophthalmoscopic mirror into a healthy eye *from a distance of* 4 *feet*, it will be more or less accurately focussed on the retina, and the reddish glow seen through the pupil will be brightest when the eye is emmetropic, and less bright in proportion as the eye is hypermetropic or myopic.

If the mirror now be tilted so as to make the light move over the eye from side to side, or up and down, the spot of light on the retina will also move, and with it that area of shadow which always surrounds focussed light.

The following phenomena may be observed, and what is true of the glow is true of the shadow.

(1) With a **concave** mirror of 8 or 9 in. focus the retinal glow moves in the *opposite* direction to (technically called "*against*") the mirror in emmetropia, hypermetropia, and in myopia of less than 1 dioptre.

If the myopia exceed 1 dioptre the retinal glow appears to move in the *same* direction as the mirror is tilted (technically called "*with*" the mirror).

(2) With a **plane** mirror in emmetropia, hypermetropia, and low myopia the glow moves "*with*," in higher myopia it appears to move "*against*" the mirror. It is then essential for the observer to note first the kind of mirror he is employing.

The *brilliance* of the glow and the *direction* of its movement are alone sufficient to indicate the presence, nature,

and to some extent the degree of a refractive error; but the amount of error may be ascertained with very great accuracy thus:—

By successively placing convex lenses of increasing strength close up to and immediately in front of an eye which we have determined by the shadow test to be emmetropic, hypermetropic, or feebly myopic, we can alter the direction of the rays as they leave the patient's eye and make them appear to come from an eye which has 1 dioptre of myopia.

If this effect is produced by a lens less than +1 D sph., the observed eye is *feebly myopic.*

If +1 D sph. is required the eye is *emmetropic.*

If a stronger convex glass is needed the eye is *hypermetropic.* From the *weakest* convex lens which makes the movement of the glow appear to be myopic, *subtract* 1 *dioptre*; the remainder expresses the amount of hypermetropia present.

On the other hand, if the original movement reveals myopia, concave lenses of increasing strength should be employed. The *weakest* which *reverses the direction of movement of the glow* may be taken as approximately the measure of the *myopia.*

Since any exercise of accommodation by the patient will increase the refractive power of his media, he should be directed to gaze at a distant object at the lowest possible level over the observer's head, in order that the test may be applied to the macular region.

For absolute accuracy a mydriatic and cycloplegic should be employed. The pupil is then large, the patient can look straight at the mirror and cannot accommodate.

This test is of the greatest value for the detection of astigmatism, since the movement in any meridian can be made to reveal the refractive state of that meridian.

When the ocular refraction is not absolutely spherical (and it seldom is), the meridians of greatest and least curvature are either respectively vertical and horizontal, or, if

oblique, are always at right angles to one another (except in irregular astigmatism).

While investigating the refraction, as above explained, the observer's attention should be concentrated first on the correction of that meridian in which the movement of the glow is most easily seen, and then on that at right angles to it. The difference between the correcting glasses required is due to astigmatism, and is the measure of it.

The first meridian may be corrected with a spherical lens, and the second more conveniently with a cylinder.

The apparent movement of objects on the Fundus.—If with an ophthalmoscopic mirror *alone* an observer seated *at a distance of* 30 *in.* from a patient perceives not merely a rosy glow, but a distinct image of part of a blood vessel on the optic disc, he should move his head from side to side, keeping the light on the patient's eye and the mirror before his own.

If now the retinal object appears to move in the *same* direction as the observer's head, the eye is *hypermetropic*; but if in the *opposite* direction, there is myopia of at least 2 dioptres present, and there may be much more.

The Examination of the Fundus Oculi.—There are two methods whereby we may view the inner surface of the globe.

(1) **The direct method** is that in which the observer, approaching with his ophthalmoscope to within 1 or 2 in., reflects the light into the patient's eye, and obtains an erect image of a small portion of the fundus enlarged about twenty times (in emmetropia). In this case the patient's media act as a simple magnifying glass.

To get the full advantage of this method, the observer should employ a compound ophthalmoscope, and must not exert his accommodation. If he has himself any refractive error, he must commence the examination with that lens behind the aperture of his mirror which corrects his vision for distant objects (*i.e.* for parallel rays).

If the patient's eye be emmetropic, the fundus will now be readily seen, since the rays emerging from an emmetropic eye are disposed in parallel pencils. But if the patient be hypermetropic, a convex lens, or, if myopic, a concave lens, will be required to render the rays parallel. This lens will give the most distinct image, and, provided the observation be made in relation to the patient's macula, *will be at the same time the measure of his refractive error.*

If the observer be emmetropic, he will readily select the required lens, if any, by bringing plus or minus lenses of increasing strength behind the mirror aperture while gazing at the fundus. If himself ametropic, and therefore employing a lens to correct his own refraction, he must increase or diminish its strength until the clearest image is obtained. The difference between the lens, if any, with which, as above directed, he commenced and that with which he concluded the observation is the measure in dioptres of the patient's error.

The source of illumination should be placed behind and to the same side of the patient's head as the eye which is to be examined. The observer should use his right eye to examine the right eye of the patient, and his left eye for the left.

(2) **The indirect method** is that in which the observer, holding the mirror in one hand before his eye, reflects light into the eye of the patient from a distance not less than 18 in. In his other hand he supports a convex lens of 3 in. focus at its focal distance from the patient's eye, resting his little finger on the patient's brow or cheek. By means of this lens the beam of light reflected from the mirror is not only concentrated so that nearly the whole of it enters the patient's pupil, but the returning rays reflected from the fundus, after emerging from the eye, are bent to foci in the air, and form an inverted image of a portion of the fundus (enlarged in emmetropia about five times) at or about the focal distance of the lens on the side nearest to the observer. This image, being situated 10 or

12 in. in front of the observer's eye, will be seen by such an effort of accommodation, or the use of such a lens behind the mirror aperture, as is required by the observer in order to read small print at that distance.

The source of illumination should be so placed that the patient's face is in shadow, and the hand holding the lens does not intercept its light on the way to the mirror. The observer should hold the mirror in his right hand when using his right eye, and in his left when using his left eye.

A definite method of conducting the exploration of the fundus is essential, and an examination of parts in the following order is recommended :—

The **optic disc** should be first sought for. The optic nerve pierces the globe about 3·5 mm. to the inner side of the posterior pole. To bring the disc into the observer's line of view, the patient must turn his eye slightly inwards. For one eye the tip of the extended little finger of the hand holding the mirror, for the other the ear on the opposite side of the observer's head, gives a convenient indication of the direction in which the examined eye must be turned.

The apparent *size and shape* of the disc depend chiefly on the refractive condition under which it is viewed.

The normal shape is circular or very slightly oval, with the long axis vertical.

In hypermetropia it appears small by the indirect method, in myopia large; and as the lens is withdrawn it becomes *smaller* in the former case, *larger* in the latter. Consequently in astigmatism the disc may appear decidedly oval, the more so as the lens is withdrawn, and the shortest axis corresponds to the meridian of least, the longest to that of highest, refractive power.

The normal *colour* of the disc is roseate from its capillary blood supply, but it is considerably paler in tint than the surrounding fundus.

Its *edge* is usually well defined, and often exhibits a fine

white circle—"sclerotic ring"—bounded by a dark border—"choroidal ring"—the free edge of the choroid. More commonly the latter ring of pigment only is seen.

The *surface* of the disc usually presents a physiological pit at or near the centre. This is the whitest part of the disc, being nearest the sclerotic, and is produced by the centrifugal disposition of the nerve fibres as they expand on to the retina.

The nerve cylinders usually lose their white sheaths before piercing the lamina cribrosa of the sclerotic. Occasionally some persist for a variable distance beyond the disc margin, producing pure white patches with striated edges which radiate from the disc. This innocent congenital condition is termed *opaque nerve fibres*, and is only of importance when mistaken for some serious lesion.

The **macula** will be found two discs' breadth to the *real* outer side of the disc. It should be seen when the patient looks directly towards the mirror. It is an area devoid of large blood vessels, sometimes indicated by a deeper pigmentation of the fundus, frequently bounded by an oval silvery halo of light reflected from its shelving margin, and often presenting a bright yellowish point at its centre—the fovea centralis.

The **retinal vessels** generally pierce the disc within the "pit." The arteria centralis retinæ, before or immediately after its emergence, bifurcates into an ascending and descending trunk, each of these again divides on the disc into one or more main vessels, which pursue sinuous arched courses inwards or outwards, giving off numerous branches to supply the retina generally. Corresponding veins converge to the disc. The main arteries are paler in tint, and have about two-thirds the diameter of the veins.

Pulsation of one or more of the veins on the disc is of frequent occurrence.

Pulsation of the arteries may be seen in glaucoma, aortic incompetence, and in association with some retrobulbar

tumours, and may be induced by artificial pressure on the globe.

By following the retinal vessels in their distribution, the general surface of the retina can be explored with the greatest accuracy and completeness. It must never be forgotten that the image obtained by the indirect method is inverted and reversed.

Some of the more important morbid appearances of the *disc* and *retina* may be briefly described.

Anæmia may accompany profound general anæmia, and small linear or irregular hæmorrhages are frequently found in severe and pernicious cases.

Hyperæmia of the disc produces an approximation of its colour to that of the fundus, and makes its margin less defined. It is not infrequent in hypermetropia.

Inflammation of the optic nerve can only be diagnosed ophthalmoscopically when the optic disc is involved. The general term for the former is *optic neuritis*; for the latter *papillitis* is a better term.

Papillitis may occur in one or both eyes independently of general inflammation of the retina—*retinitis*, or in association with it—*neuro-retinitis*, or in slighter degrees may participate in *choroiditis*.

The signs of papillitis are:—Increased redness or fluffy reddish-greyness, blurring of the edges by exudation, tumefaction, expansion and striation of the surface, distention of retinal veins, narrowing of arteries and obscuration of many of those vessels on the disc.

Its presence in both eyes affords a most important confirmation of the diagnosis, when other symptoms point to cerebral tumour; but among other causes are meningitis (tuberculous or syphilitic), cerebral abscess, renal disease, lead poisoning and cold.

Hyperæmia of the retina, owing to the natural vascularity of the part, cannot be easily diagnosed until tortuosity and distention of the veins, with commencing cloudiness of the

membrane from exudation, especially along the lines of the vessels and at the disc margin, indicate that *retinitis* is actually present.

Moderate congestion, with a steamy appearance of the fundus, characterises *serous retinitis*, and is probably syphilitic, especially if dust-like opacities occupy the vitreous humour. More marked congestion, with flame-like or linear hæmorrhages scattered over the fundus, may justify the term *hæmorrhagic retinitis*.

In **albuminuric retinitis** the intensity of the signs varies greatly. The most characteristic changes are found about the macula in the form of glistening white dots radiating in rows from the fovea centralis. Some of these may coalesce into patches of various shape and size. Small spots of hæmorrhage are frequent. In any case an absolute diagnosis should not be given until the urine has been examined, since similar appearances are sometimes found in diabetes and other less understood conditions of systemic poisoning. Inflammation of the disc and retina may resolve or pass into atrophy.

In **optic atrophy** there is pallor of the disc from fibrosis, and loss of capillary blood supply. If the retina is involved there is also shrinking of the retinal vessels, and frequently white lines along their edges from fibrosis of their walls.

Mere pallor of the disc does not imply atrophy unless associated with restriction of the field of vision.

Embolism of the central artery causes sudden blindness. In thrombosis the onset is less sudden. In both, marked pallor of the fundus, great constriction of arteries, narrowing of the veins on the disc, with some increase in size towards the periphery, are the earliest signs: within forty-eight hours foggy œdema of the retina about the disc, and especially at the macula, with a plum-coloured spot at the fovea. Absorption of the œdema and atrophy result in a few weeks. If only one branch is plugged, the appearances are limited to the area supplied. Cardiac or arterial disease should be looked for.

Retinitis pigmentosa is a slowly progressive non-inflammatory degeneration of the retina, characterised by the disposition of pigment in long lines upon the coats of the retinal vessels, or in branching spider-like spots, forming together a lace-like-pattern disposed in a zone intermediate between the equator and posterior pole of the eye. Shrinking of the vessels, and hazy waxy pallor of the disc, are further indications of the atrophic sclerosing process.

Detachment of the retina may be traumatic or idiopathic.

If recent, slight and transparent, the diagnosis must depend on the relatively hypermetropic refraction of the detached portion, as compared with the rest of the fundus, on examination by the direct method. By the indirect method, if the lens be moved from side to side, a detached portion of the retina (being focussed nearer to the observer) appears to move more freely than the attached membrane,—just as when one eye is closed and two objects in the same line are viewed, the nearer appears to move over the more distant when the observer sways from side to side. This is termed "parallactic movement." If the detached retina has any folds or rucks, as is commonly the case when atrophied or extensive, silvery grey reflections of light occur from their surfaces, and may be readily detected with the mirror alone at a distance of 8 or 10 in. A floating billowy grey membrane, with dark or bloodless vessels coursing over the crests, is typical. If it does not quiver as the eye is moved, an intraocular tumour should be suspected.

The Choroid is only visible in proportion as the hexagonal pigment layer of the retina is deficient, and then presents numerous dull or brick-red vessels disposed in leashes, or a network with pigmented connective tissue between. The distribution of the choroidal vessels can be still more easily seen in very fair subjects — especially albinos — the red vessels standing out clearly against the white sclerotic.

Acute choroiditis is commonly accompanied by exudation in the vitreous, which further screens the process.

The commoner appearances are the results of previous disease which have led to thinning or atrophy of the membrane and pigmentation. A crescentic defect in the choroid at one (usually the outer) side of the disc is so frequent in myopia as to be called a *myopic crescent.* When larger, it may surround the whole disc and extend irregularly—*staphyloma posticum.* Its colour is white, because the sclerotic forms the background. It may be pigmented, especially at its margin. The retinal vessels pass distinctly superficial to it. The macula is a frequent seat of choroidal atrophy and pigmentation—*central choroiditis.* There is a senile form, which presents numerous small yellowish-white spots, which tend to coalesce, and may undergo atrophy and pigmentation. This should be looked for when sight fails in old persons.

In **disseminated choroiditis** white patches of atrophy, with or without pigment, varying in number and size, are scattered irregularly over the fundus. When small and well defined, enclosing a spot or bounded by a ring of pigment, syphilis is strongly suggested.

Hæmorrhages and pigmentation of the choroid tend to assume coarser proportions than in the retina, and their deeper situation can often be appreciated more readily if a retinal vessel can be traced passing superficially to them.

Tubercles in the choroid are rare, but may occur in general tuberculosis, usually at a late stage. They usually present isolated prominent yellowish rounded spots situated about the posterior pole of the eye, behind the plane of the retinal vessels, and free from atrophy or pigmentation.

The term **glaucoma** is applied to a group of affections accompanied by increased intraocular tension.

In *acute glaucoma* there is usually a dull or steamy condition of the media, which may entirely obscure the state of the fundus. In severe cases retinal hæmorrhages may be visible, or extensive hæmorrhage may complete the shroud. Pulsation in the retinal *arteries* on the disc is not uncommon.

Complete cupping or retrocession of the whole surface of the disc, from long-continued pressure, is characteristic of the more *chronic* forms of the disease. A bluish pallor within the cup, and a yellowish halo round its margin, indicate the atrophy by which it is usually accompanied. Abrupt turning of the vessels over the edge of the disc, apparent loss of continuity in their course, and parallactic movement of the edge of the disc in relation to its floor, are further proofs of deep cupping. These, however, are but the final appearances of this insidious malady. The state of the intraocular tension and the subjective symptoms are the more essential factors in the diagnosis of the disease at the time when it is remediable.

The Subjective Examination

In ordinary parlance the term "sense of sight" includes both simple visual sensations and visual judgments.

The simple or direct visual sensations are those of *light*, *colour* and outline or plane *form*.

Combinations of these with motor impulses sent to the ocular muscles, aided by psychical processes of memory and comparison, give us conceptions of size, distance and solid form. These are visual judgments.

To determine the functional power of the optic nerves and visual paths, it is sufficient to direct our attention to the state of the simple sensations.

In health, and under suitable stimulation, these may be called forth at any part of the retinal surface, except the optic disc ("the blind spot") and near the ciliary processes.

The retinal sensibility, under ordinary illumination, is by no means uniform throughout. It is most acute at the fovea centralis, where the cones are most abundant, and fades away towards the periphery of the fundus.

Direct vision.—If we wish to see an object distinctly we direct our eye towards it in such a way that its image falls upon the fovea centralis. We call this act *direct vision*.

But rays of light from a considerable area around the object looked at enter the eye through the pupil at the same time, and stimulate sensitive retinal elements.

Field of Vision for Light.—This area is termed the *field of vision* for light. The point on which our gaze is fixed is the *point of fixation*, and any objects visible in the field not occupying the point of fixation are said to be seen by *indirect vision*, their images being applied to parts of the retina which are excentric or peripheral in relation to the fovea centralis.

A complete subjective examination should therefore determine the sensibility to light, colour, and form at every part of the fundus of the eye which is normally sensitive. In the great majority of cases, however, there is no necessity for such exhaustive inquiry, and for clinical purposes it will be found convenient to conduct the examination as follows, taking care *to test each eye separately*, first in direct and then in indirect vision.

I. **The direct Visual Acuity for form should be tested for distant objects,** first *without* and then *with* the aid of suitable glasses, if necessary.

It has been found experimentally that, provided an object is sufficiently well illuminated and stands in sufficient contrast to its surroundings, it will be clearly seen if its retinal image subtends an angle of one minute at the nodal point of the eye.

No one object can fulfil this condition if placed at different distances from the eye, since the farther it is removed the smaller becomes its retinal image.

Snellen's Test Type.—Starting from this basis, Snellen has prepared a series of black test types in horizontal rows on a white surface, of such dimensions that when the card is placed at 6 metres (about 20 ft.) from a normal eye, each of the smallest letters subtends an angle of five minutes within

the eye, and the breadth of each of its component limbs an angle of one minute.

The letters in the next row are uniformly larger, so that each would subtend the same angles as above, though removed to a distance of 9 metres (about 30 ft.).

Similarly the other rows contain letters which when seen at 12, 18, 24, 36 and 60 metres respectively subtend the same angles. The letters have otherwise no special relation to one another, they do not combine to form words, and thus do not easily become familiar.

The individual to be tested should stand with his back to a bright window, the types being hung on a level with his head on the opposite wall *at* 6 *metres distance.*

In a badly lighted room daylight may be excluded and artificial light employed. In this case the light should be placed near the test types, and shaded on the side nearest the observer. A steady lamp or gas flame should be used, and a note taken of its approximate brilliance, so that, if required, the examination may be subsequently repeated under conditions as nearly similar as possible.

Having covered one eye lightly with the palm of the hand —or, preferably, a disc of some opaque material, as cardboard or dull glass, which does not prevent free evaporation from the excluded eye—the patient is requested to read the letters aloud, commencing with the largest.

How to express Visual Acuity.—If at this distance he reads the smallest of the series successfully, his sense of form (visual acuity) *at the macula* is normal, and may be conveniently expressed as a fraction thus—V (vision) $= \frac{6}{6}$ or unity; the numerator expressing the distance at which he is standing, the denominator the distance at which the type should be seen by a normal eye.

Suppose that at this distance (6 metres) he is unable to read letters smaller than those which should be seen by a normal eye at 12 metres distance, his $V = \frac{6}{12}$, *i.e.* only half of the normal. The visual acuity may be similarly expressed

throughout the whole scale from $\frac{6}{6}$ to $\frac{6}{60}$, the denominator in each case corresponding to the row of letters at which distinct vision ceases.

Some test cards for distance bear a row of smaller letters which should be read at 5 metres by a normal eye. Many persons having keen vision can distinguish these at 6 metres. In such a case $V=\frac{6}{5}$, the fraction showing that the acuity is above the average. If the size of a room necessitates the application of the test at a less distance than 6 metres, the actual distance employed must be stated in the numerator, and proportionately smaller test type must be used to denominate normal vision.

Thus at 5 metres if $V=\frac{5}{5}$ it is normal, but it may be defective, as $\frac{5}{6}$, or $\frac{5}{18}$, or $\frac{5}{60}$. If at 4 metres $V=\frac{4}{4}$, it is probably normal, but, strictly speaking, the full distance of 6 metres should always be employed by preference, for reasons which we must now consider.

The Refraction of the Eye.—Thus far we have said nothing of the influence of the refraction of the eye on the visual acuity. It is evident that if there is some error in the refractive media, the patient's optic nerve may be quite healthy, and yet his vision will appear defective because an indistinct image is received on the rods and cones.

The advantage of testing a patient at 6 metres is that *rays coming from any one point at that distance are practically parallel when they enter the pupil*, and thus afford a convenient standard for estimating the ocular refraction.

Emmetropia.—In an *emmetropic* (*i.e.* normal) eye the relation between the length of the eye and the refractive power of the media is so fitly proportioned, that parallel rays are brought to a focus on the fovea retinæ, if only the eye be directed towards the point from which the rays are coming.

Ametropia.—If the eye be *ametropic*, parallel rays will not be thus passively focussed. The defect may arise from hypermetropia, myopia, or astigmatism.

Hypermetropia.—In *hypermetropia* (badly termed "long sight") the refractive media at rest are incapable of bending the rays sufficiently, so that they impinge on the retina before they have come to foci. To get a sharper image the patient has to use his accommodation (*i.e.* to increase the refractive power of his lens by the action of his ciliary muscle), or requires the aid of such *convex* spherical glasses as will, by rendering the rays sufficiently convergent before they enter the eye, provide compensation for the defective refractive power of the natural media.

Myopia.—In *myopia* ("near sight") the refractive media are proportionately too strong, so that parallel rays are brought to foci before they impinge on the retina. Rays from a less distance, being divergent, will have their foci thrown farther back. Hence for clear vision a myope requires to get nearer an object than an emmetrope, or has to employ *concave* spherical glasses, which cause rays passing through them to diverge as if coming from a nearer point.

Astigmatism.—By *astigmatism* we mean that the rays from any one point are not equally focussed within the eye.

Simple Hypermetropic or **Simple Myopic Astigmatism.**—Thus in one meridian the refractive power of the media may be emmetropic, but in another, and usually that one at right angles to the first, it may be hypermetropic or myopic.

Compound Hypermetropic Astigmatism. — Sometimes hypermetropia is present in every meridian, but most marked in one, and least in that at right angles to it.

Compound Myopic Astigmatism.—Similarly an eye may be myopic in every meridian, but more markedly in one than in another at right angles to the first. In astigmatism the patient can only obtain clear vision with the aid of cylindrical lenses.

Irregular Astigmatism.—Sometimes the refraction varies so greatly from meridian to meridian that it cannot be definitely estimated, nor can the vision be satisfactorily improved by lenses.

In any case, then, in which *V* appears defective we must ascertain the state of the refraction, with a view to determining whether the failure is optical or nervous.

Subjective Examination of the Refraction.—In proceeding to make this examination much time may be saved by a few judicious questions as to the nature of the patient's difficulty in seeing. Thus one who asserts that he sees well at a distance but less well near at hand, and especially in artificial light, who complains of letters running together, of painful sense of straining of the eyes, or headache after continued near work, is probably *hypermetropic* if young, or *presbyopic*, or both, if over 45 years of age.

Presbyopia is that condition in which there is inability to *accommodate* the eyes for near objects of a certain degree of fineness, on account of loss of elasticity of the lens. It commonly makes itself felt about the age of 45, and increases with advancing years. Distant vision is unaffected. Convex spherical glasses of strength suited to the age of the patient remove the disability for near work. Emmetropes and hypermetropes become conscious of the defect sooner than myopes. A myope (*e.g.* of 50 or more) may never experience any symptoms of presbyopia, because for ordinary near work, such as reading, he has little or no occasion to exert accommodation.

One who sees badly at a distance, but reads the smallest print if held near, is generally *myopic*, and the nearness required is a fairly accurate measure of his myopia.

Regular Astigmatism may be suspected if there is a complaint of seeing imperfectly both far and near, and especially if lines which run in one direction are more evident than those at right angles to the former.

Having ascertained the last row of letters which the patient can see with either eye separately, keep one covered and place before the other a weak *convex* glass, such as +0·5 D sph.

If *V* was previously $\frac{6}{6}$ with the naked eye, but is made

less distinct with this lens, the refraction is practically *emmetropic*, and need not concern us further.

If this lens causes no diminution of the visual power, or actually improves V, replace it by a slightly stronger convex glass. Inquire again what type can be read, and so long as *V* is improved, gradually increase the strength of the lens.

Suppose that with the naked eye a patient has $V = \frac{6}{6}$, and that when + 2 D sph. is placed before it he has still $\frac{6}{6}$ (the letters appearing larger and better defined), we conclude that he is *hypermetropic*, that he obtained the first result by exerting his accommodation, and the second by relaxing it when the artificial lens gave the necessary degree of convergence to the rays before they entered his eye, and made an alteration of the curvature of his own lens unnecessary.

All the hypermetropia which we can reveal in this way is called "*manifest hypermetropia.*" If the patient is young it is probable that he has some more hypermetropia "*latent*," that is, concealed and corrected by an habitual accommodative action of his ciliary muscle, which he is incapable of altogether relaxing. By the use of a mydriatic which paralyses the ciliary muscle the latent hypermetropia may be made manifest. It will be found that a stronger convex glass is now needed to give the most distinct distant vision, and this lens is the measure of the "*total*" hypermetropia. In practice it is usually sufficient to order glasses which correct the manifest hypermetropia, unless there be convergent strabismus which it is desired to counteract by relieving the associated accommodative action.

In adults of 45 years and upwards, owing to the tendency to failure of accommodative power, all the hypermetropia is usually *manifest*, and there is no need, and possibly some risk, in using a mydriatic, since it may cause an increase of the intraocular tension. In such a person the examination may show $V = \frac{6}{18}$ with the naked eye, but with + 4 D sph. $= \frac{6}{6}$. Here it is evident that the refractive media, even aided by such accommodative power as remains, are unable

to bring parallel rays to a focus upon the retina without the above artificial assistance, and we declare that this patient has 4 dioptres of manifest hypermetropia.

By commencing this subjective inquiry with *convex spherical* glasses, the best inducement is given to the patient to relax his ciliary muscle. If concave glasses be first presented to a hypermetrope they are apt to excite, especially in the young, a strong ciliary contraction to overcome their dispersive action, and this muscular exertion may prevent the true state of the refraction being determined by this method of examination.

If *V* be improved by convex spherical glasses, but be less than $\frac{6}{6}$, the convex spherical glasses which give the best vision should be retained in front of the eye, and a weak *convex cylindrical* lens added to it, and rotated slowly through two right angles, to determine in what axis, if any, its assistance is required. If benefit results, the case is one of *compound hypermetropic astigmatism.* The position of the axis should be noted, and convex cylinders of increasing strength successively inserted, preserving the same axial position so long as *V* is improved. If convex cylinders give no aid, concave cylinders should next be similarly tried.

The patient has *compound hypermetropic astigmatism*, *simple hypermetropic astigmatism*, or *mixed astigmatism*, according as the concave cylinder lens, if any, which gives the best vision has a numerical strength *less than*, *equal to*, or *greater than* the convex spherical glass with which it is combined.

If, however, a weak convex spherical glass impairs rather than improves visual acuity, it should be removed, and concave spherical glasses should be employed, commencing with the weakest, and gradually increasing in strength. If *V* is improved thereby the patient is evidently **myopic,** and the feeblest glass with which the greatest improvement in vision is obtained is the measure of the patient's spherical error.

Here, again, if V be less than $\frac{6}{6}$, a cylinder should be placed in front of the spherical glass, commencing with a weak concave; the best position for the axis of the cylinder should be sought, and its strength gradually increased. If improved vision results, the patient has *compound myopic astigmatism.* If concave cylinders produce no benefit, try convex. The patient has *compound myopic astigmatism*, *simple myopic astigmatism*, or *mixed astigmatism*, according as this convex cylinder, if any, which gives the best vision has a numerical strength *less than*, *equal to*, or *greater than* the concave spherical glass with which it is combined.

In any cases where spherical glasses alone have been rejected, *cylinders alone* should be tried. Indeed, simple hypermetropic or simple myopic astigmatism is usually discovered in this way. But it sometimes happens, and especially in patients whose vision is not very acute, that the examiner is misled by the patient into believing that the eye requires the aid of a spherical glass, when in reality a simple cylinder is all that is needed, and this is ascertained by the patient admitting that he sees best when a cylinder of equal numerical strength, but opposite sign, has been combined with the spherical glass first adopted, and has neutralised its effect upon rays passing at right angles to the axis of the cylinder.

If the examination is made with the object of ordering glasses, it is well, after correcting each eye separately, to make a final test with both eyes open, in order to see that no diplopia or sloping of objects results from unequal focussing in the two eyes.

Now, though these details of the method of estimating the visual acuity have occupied some time to describe, with a little experience on the part of the observer, and a moderate degree of intelligence on the part of the patient, they can be very rapidly performed in practice.

The great advantage of commencing the investigation in this way is, that if no subjective visual sensations are com-

plained of, and $V = \frac{6}{6}$ has been obtained in each eye with or without glasses, we may *generally* conclude that the sense of sight is normal, and further examination is needless, unless to exclude the possibility of congenital colour blindness or some ophthalmoscopic change being overlooked. The ophthalmoscopic examination should never be omitted, since evidence of disease may be found in the fundus, though the direct visual acuity is not affected. If, however, V cannot be raised above $\frac{6}{12}$ or $\frac{6}{18}$ even with glasses, a careful objective examination should be made, and unless a sufficient explanation of the defect is thus obtained, *e.g.* high refractive error, irregular astigmatism, or opacity of the media, it will be well, especially when the defect is of recent origin, to determine the state of the colour sense and the extent of the fields of vision before assuming that the eye is congenitally amblyopic.

In many cases, especially in adults, where $V = \frac{6}{24}$ to $\frac{6}{60}$ and cannot be improved by glasses, the physician may be enabled to form a sound diagnosis with rapidity by determining—(1) The power of the eye to perceive colours, especially in the central parts of the field; and (2) the extent of the field of vision for hand movements, before proceeding to the ophthalmoscopic examination.

If a patient fail to see the largest type at 6 metres (*i.e.* V = less than $\frac{6}{60}$), he should be allowed to approach the card slowly until the largest types are seen. The distance must then be measured and expressed as the numerator, thus (say) $V = \frac{4}{60}$ or $V = \frac{1}{60}$, according as the individual is 4 metres or 1 metre from the type.

In no case should the expression $\frac{6}{0}$ be used to express V = less than $\frac{6}{60}$, since $\frac{6}{0}$ = infinity, and, while failing to express what is intended, gives no information as to the actual amount of the defect.

Counting Fingers.—A rougher method of testing the acuity of vision when less than $\frac{6}{60}$ is that in which the observer holds up one or more of the fingers of his hand,

invites the patient to count them, and in retiring from him varies the number of fingers so long as they are correctly counted or named. The result is expressed thus—"Counts fingers at (say) 3 metres." An observation such as this, though crude, is of value in estimating the further progress of a case.

Hand Reflex.—If the patient cannot count fingers when brought within a few inches of his face, we proceed to see whether he can perceive an up-and-down or side-to-side movement of the hand held directly before his eye. If he can, we say that he has "hand reflex."

Perception of Light.—So marked a depreciation of visual power always implies dense opacity in the media (*e.g.* advanced cataract, hæmorrhage or exudation into the vitreous), detachment of retina, or serious failure of nerve power, or both, and in such cases it is always well at the same time to determine whether the perception of light is good, not only at the posterior pole of the eye, but over the general surface of the retina, and in those cases in which hand reflex cannot be elicited, this is the last step by which we ascertain whether the eye possesses any visual power or is altogether blind.

The simplest method is to make the patient face a bright window, exclude one eye completely, and place a hand a little in front of the one to be tested, so as to shade the light from it for a moment, then uncover and recover the eye, asking the patient to say if he notices any difference, when it is light and when dark.

Care should be taken that in making these movements the moving hand does not touch the face or give any other sensory indication of its change of position. The test may be more accurately applied in a darkened room by reflecting light with an ophthalmoscopic mirror, so as to enter the eye from successively different directions. The patient is requested to direct his gaze straight forward, to declare the moment he perceives any light on his eye, and to state

whether it is coming from above or below, from his right or left side.

The strength of the light employed can be readily increased or diminished by varying the size of the flame employed or the distance from which it is reflected.

Having thus rapidly traversed the way by which we are led to determine the state of the *direct visual acuity for form* from perfection to blindness, we turn now to the other simple visual sensations.

The examination of the *sense of colour in direct vision* need only be undertaken in a special manner when there is any reason to suspect the presence of congenital colour blindness, and it then demands great care and discrimination in the method employed. Space does not permit of a proper presentation of this subject in a work of this kind, and the reader should consult the ophthalmic text-books of Berry, Juler, or Swanzy, or a special treatise, as Joy Jeffries' on "Colour Blindness."

Some clinical means for testing colour perception, chiefly in indirect vision, will be referred to later.

II. **The field of vision** (*i.e.* **indirect vision**) **should now be examined.**

There are several methods of different degrees of precision available for this purpose.

1. **Hand Movements.**—Estimation of the extent of the field of vision for *hand movements.*

This is a very rough but exceedingly useful test where the central acuity has been found markedly defective.

Place the patient with his back to the window or source of light; make him cover one eye lightly; stand directly opposite him at a distance of 2 ft., and close the opposite named eye in your own person. Then let each individual gaze at the other's open eye. The field of vision of each should now be identical, though seen from opposite aspects. The observer with a healthy eye has thus a ready means of

comparing the extent of the patient's power of perception with his own, while his outstretched hand moves radially outwards from or inwards towards the common visual axis, in different directions, but always in a vertical plane midway between his own and the patient's eye.

A decided contraction of the field, as is met with in, *e.g.*, optic atrophy or retinitis pigmentosa, or the position of an extensive scotoma, as from detachment of the retina or choroido-retinal atrophy, may be rapidly determined by this method.

2. **Campimetry.**—By the use of a large black board or velvet screen the field of vision may be projected on to a plane surface.

Such an arrangement is called a *campimeter*.

The patient should stand with his back to the window, cover one eye, and fix the gaze of the other on a small white spot affixed to the centre of the board, which should be directly in front and, at least, 18 in. from his face. Various test objects can be affixed in turn to the end of a long black rod, and moved radially outwards from the fixation point, or inwards from the periphery, and the remotest point in each direction at which the object is perceived is marked on the surface of the board in chalk. A piece of white chalk makes a convenient test object and marker combined.

A line connecting all the chalk marks, of any one colour, gives an outline enclosing the field of vision as thus obtained for that colour.

But this method is not well suited for the peripheral parts of the field, since, in order that these may be tested, the object must be removed farther from the eye, and the test becomes unequal. For the more central parts it is practically accurate in principle, and may be very conveniently employed in a manner which will be explained later on.

3. **Perimetry** affords the most exact method of estimating the field of vision.

Several perimeters have been devised, differing in mechan-

ical detail, but most consist of a graduated bar (semicircular or forming the quadrant of a circle) capable of supporting a suitable test object, and of describing the circumference of a hemisphere, whose pole—the pivot of the bar—is the point of fixation.

The eye to be examined should occupy the centre of the sphere, and thus the chief defect of the campimeter is overcome, for whatever the position of the test object on the bar, its radial distance from the eye, the size of its image on the retina, and its luminosity are constant.

The straight line which connects the fovea centralis of the retina with the point of fixation is called the *visual axis.*

Any other straight line passing into the eye from any point on the quadrant to the retina must cross the visual axis at a certain angle, which increases directly as the distance of this point from the point of fixation.

This relation is expressed on the quadrant, which is divided into degrees, starting from 0° at the pivot (point of fixation) to 90° at the free end.

If the quadrant be set in rotation every point of it will describe a circle, and all the circles are concentric. The combined circles forming the imaginary hemisphere on which the field of vision is to be mapped out may be represented on a plane surface by a few of these circles described at equal angular intervals; lines radiating at equal intervals from their common centre will represent meridians of curvature of the hemisphere. (See Fig. 141.)

Such a representation, reduced in size, affords a chart which is usually affixed to the back of the perimeter.

The test object is a disc or 1-centimetre square of white or coloured unglazed paper. If inserted into the end of a small black rod (6 or 8 in. long) it can be held by the physician in any desired position, or it may be supported in a clip attached to, but movable, along the concave aspect of the quadrant.

The observation should be made in good diffuse daylight.

Let the patient sit with his back to the window. Lightly bandage one eye or cover it with a small shade, taking care that no part of the obscurer is visible in, and therefore does not curtail, the field of the other eye. Place the perimeter on a table in front of the patient, and carefully adjust its height so that the fixing eye can be maintained without

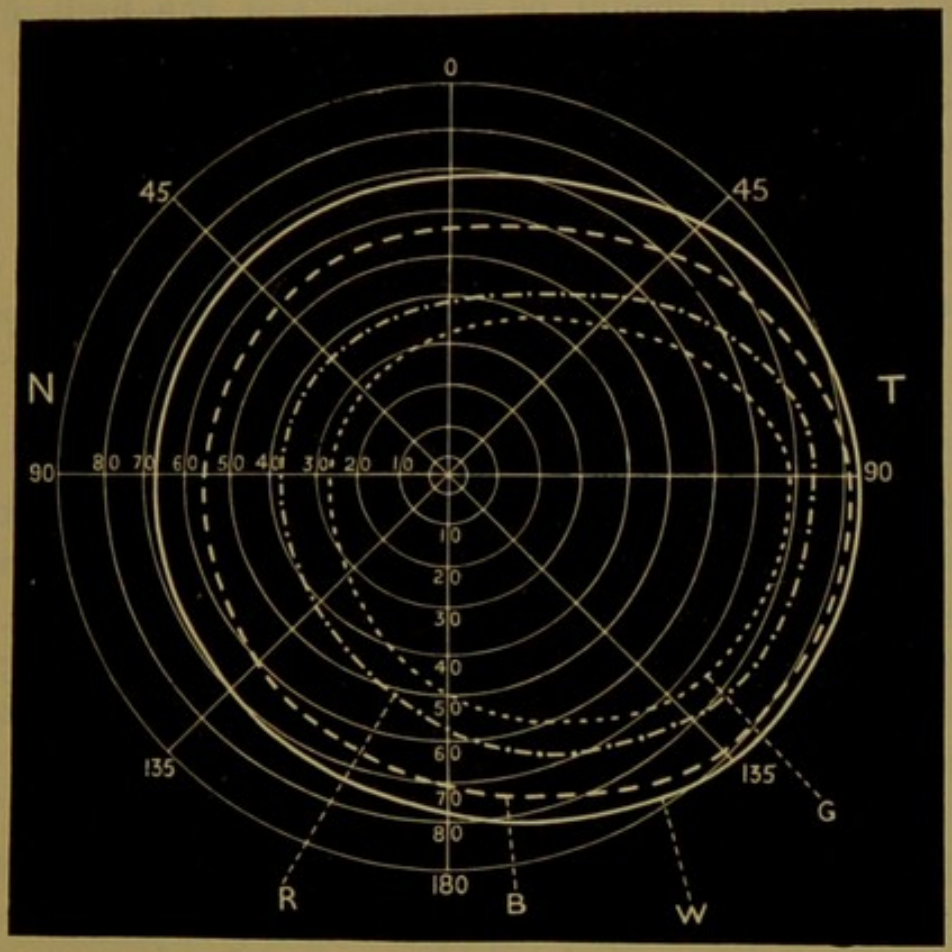


FIG. 141.—Perimetric chart showing field of vision of right eye—for white (W), blue (B), red (R), and green (G), as projected on the inner surface of a hemisphere whose pole is the point of fixation. N denotes the nasal or inner, T the temporal or outer half of the field.—Landolt.

discomfort in the same horizontal plane as the point of fixation. To ensure steadiness a head-rest for the cheek or chin is usually supplied.

The physician, facing the patient, directs him to fix his gaze on a small white spot at zero on the quadrant, and as the examination proceeds takes care to recall the patient's eye if it wanders from the fixing point.

Having set the quadrant in some definite meridian—say the horizontal—the test object is brought slowly along the arc from the periphery towards the centre. The patient is to declare the moment at which he first perceives its advance, and to state its colour if coloured. In the latter case especially he may have some hesitation in deciding, and if so the direction should be reversed, and the limit fixed approximately by inquiring for the point at which he appears to lose the colour after clearly perceiving it in the more central part of the field.

The physician having recorded this by a prick or coloured pencil mark on the corresponding spot of the chart, turns the quadrant through a few degrees, repeats the observation in another meridian, and again records the result.

The greater the number of meridians examined, the more complete the information derived; but it must be remembered that if the examination be prolonged, fallacies may arise from exhaustion of the retina. A curved line connecting similar marks on the meridians examined gives the outline of the field for the corresponding test object employed.

In moderate daylight the fields for white and black are larger than those for any of the colour tones, but Landolt has shown that in a normal eye, if the illumination be sufficiently bright, all the fields are co-extensive.

The chart (Fig. 141) shows the oval shape of the fields, that their greatest expanse is outwards and downwards, least upwards and inwards. The curtailment of the latter is chiefly due to the projection of the nose and brow.

In practice it is convenient to take first the field for white, which alone may give sufficient evidence to warrant a diagnosis, and avoids the fallacy of any congenital defect for colours.

In the selection of colour tests, care should be taken that the colours employed are as pure as can be obtained.

Red and green form one pair of complementary colours, blue and yellow another pair. Under ordinary illumination the latter have the larger fields.

It is evident that if either the red or green test object contains much admixture of yellow or blue, a considerable error in the apparent size of the fields may be introduced. The presence of a decided impurity in a colour test may be readily detected by the examiner, if his own colour sense be good, by noting whether the test object, which appears red or green when viewed directly, becomes bluish or yellowish when viewed by himself indirectly in the inner half of the field of either eye, and moved into an increasingly peripheral position.

The fields of vision may be found defective—

(*a*) In extent or continuity.
(*b*) In acuity.

(*a*) **Extent.**—Concentric contraction of the fields is met with notably in retinitis pigmentosa, and usually in atrophy of the optic nerve; but not infrequently in the latter the shrinking of the field is irregular, depending doubtless upon the order in which the process affects the nerve bundles.

In chronic glaucoma there is also gradual diminution of the field, but the inner limits are usually first curtailed, and the outer shrinks more slowly.

In embolism of the central artery of the retina, if any vision is retained at all it is in the outer part of the field, and there seldom exceeds the perception of hand movements. Embolism of a branch of the same artery will give rise to a failure in that segment of the field which corresponds to the impoverished retinal area. In detachment of the retina, in atrophy of the retina from injury or disease of the adjacent choroid, and usually in congenital coloboma of the choroid, a defect in the field will be found corresponding to the lesion as determined by the ophthalmoscope.

In some cases, though the fields are of normal *extent*, a

localised area of blindness, a scotoma, may be found interfering with their *continuity*.

The optic disc is a physiological scotoma about 15° to the outer side of and below the point of fixation.

Scotomata may be small or large, single or multiple. They are described as *positive* if the patient is conscious of their interference in his field, *negative* if, like the optic disc, they do not obtrude themselves upon his consciousness.

In the latter case they may not be discovered by the physician unless he takes the precaution of ascertaining, not only the outlines of the fields, but also whether the test object is properly perceived along each meridian, from the limit inwards to the fixing point.

In mapping out a scotoma it is often a good plan to attach the test object to the quadrant and slowly rotate it through as many degrees to the right or left as may be necessary to determine where it can and where it cannot be perceived by the patient. By combining circular movements of this kind with the to-and-fro radial movements along the quadrant previously described, the dimensions of the scotoma may be pretty accurately demarcated.

As already indicated, every ray which enters the eye, except that coming from the point of fixation, crosses the visual axis before it reaches the retina.

[*Note.*—The point of intersection of all the entrant rays is the *nodal point* of the eye. It is situated close behind the crystalline lens in the anterior part of the vitreous.]

Consequently, rays coming from the right half of each field are focussed on the left half of each retina, and similarly with the right half of each retina we perceive objects situated in the left half of each field, so far at least as the fields are coincident; objects situated above the visual axis are focussed on the lower part of the retina, while those below the line of fixation imprint their images on the upper part of the retina.

Clinical and pathological observation have combined to prove that the right half of each retina is innervated by the nerve fibres which compose the chief part of the right optic tract, while those which supply the left half of each retina come from the left optic tract. The inner half of each retina is thus supplied by fibres which have decussated at the optic chiasma.

We are now able to understand the occurrence of symmetrical scotomata, *i.e.*, defects in corresponding parts of both fields of vision.

For while a destructive lesion of one optic nerve, as at 1 or 1′ in Fig. 142, will produce complete blindness of the corresponding eye, a lesion of the left optic tract at 2 will cut off communication between the *left half of each retina* and the brain, and produce blindness in the *right half of each field* (right homonymous hemianopsia); similarly a lesion 2′ will render the *right half of each retina* functionally useless, and produce blindness in the *left half of each field* (left homonymous hemianopsia).

A lesion in the chiasma at 3 will destroy the function of the *inner half of each retina*, and produce blindness in the *outer half of each field* (temporal hemianopsia); while it will require a very symmetrically disposed lesion at 4 and 4′ to cut off the sensory fibres from the *outer half of each retina* and produce blindness in the *inner half of each field* (nasal hemianopsia).

Homonymous hemianopsia is the commonest variety; temporal hemianopsia is fairly frequent; but only one authentic case of nasal hemianopsia appears to have been described, and, considering the peculiar lesion required, its uniqueness is not surprising.

Superior and inferior hemianopsia, *i.e.* defective vision in the upper and lower halves of the fields respectively, have been described, but are much more likely to be due to retinal disease, *e.g.* detachment of the retina in the same part of each eye, than to symmetrical lesions in

the course of the nerve tracts, or in the visual areas of the brain.

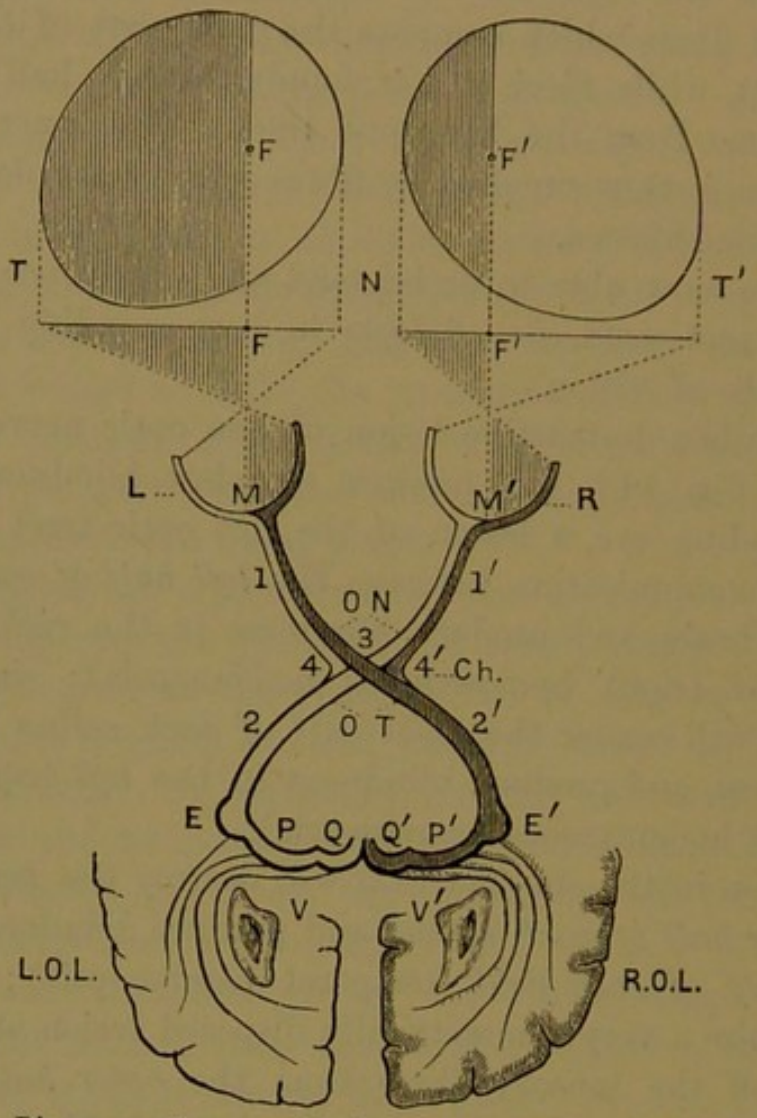


FIG. 142.—Diagrammatic representation of the visual conducting paths and fields of vision, to show the crossing of the rays in the media, and the relation of the half-fields to the eyes and to the cerebrum.

L. Left eye. *R*. Right eye.
M & *M'*. Macula lutea in each.
F & *F'*. Point of fixation.
(In binocular vision *F* & *F'* coincide, and the fields overlap.)
N. Between the nasal or inner half-fields.
T & *T'*. The temporal or outer half-fields.
O N. Optic nerves. *Ch*. Chiasma.
O T. Optic tracts.
E E'. External geniculate body.
P P'. Pulvinar (posterior end of optic thalamus).
Q Q'. Corpora quadrigemina.
V V'. Posterior ends of lateral ventricles.
L.O.L. Left occipital. *R.O.L*. Right occipital lobe.
For 1, 1', 2, 2', 3, 4, 4', see text.

In no case of supposed hemianopsia should a diagnosis be made without a very careful objective examination. If the entire half-field of each eye is functionless, the hemianopsia is said to be *complete*. If a part of it retains visual power, the case is *incomplete*. In the latter case the scotoma may be sector-like, or insular, or quite irregular.

It is by no means easy to determine the exact seat of a lesion producing lateral homonymous hemianopsia, since it may be on the optic tract only a short distance behind the chiasma, or nearer the basal ganglia, or above these in the occipital cerebral substance as high as the occipital cortex. There is, however, one objective test associated with the name of **Wernicke**, which, though of doubtful value, we may conveniently refer to here.

It depends on the supposition that there is a special set of nerve fibres concerned purely in the conduction of the stimulus of light from the retina to the third nerve nucleus in the floor of the aqueduct of Sylvius, that these fibres do not convey *visual sensations*, but leave the optic tract at the level of the basal ganglia, and do not pass up to the occipital cortex.

Wernicke suggested that if the lesion causing hemianopsia was situated above the point of emergence of these pupillary light reflex fibres, the pupillary reaction to light ought to be carried on perfectly, though the light stimulus was applied only to the blind half of the retina. But if the lesion occurred below the point of emergence of the said fibres, the careful admission of light into the eye in such a way as to fall only on the blind half of either retina ought to be followed by no contraction of the pupil.

Seguin recommends that the light should be thrown into the eye from an ordinary ophthalmoscopic mirror at an angle of 40° to 60° from the visual axis. It is evident that the test can be of no service unless the tract lesion is *absolutely destructive* in its effects on the fibres which conduct visual sensations. This leads us to the second

aspect in which the fields of vision may show deficiency, namely—

(*b*) **Acuity.**—There may be various grades of impairment of visual acuity. Speaking generally, it would appear that whenever visual failure is essentially due to disease of the optic nerves or tracts, the sense in which the failure is earliest exhibited is that of colour, and notably in the perception of red and green. In slowly advancing optic atrophy blue and yellow may still be perceived throughout small fields, when red and green are no longer perceptible under ordinary illumination.

It is probable, too, that in all cases the sense of form for objects seen indirectly or directly fails in some proportion to the colour sense, but the peripheral form sense has never received much attention. It may be tested readily by employing strips of white cardboard having one, two, or three black squares (2 cms. square) respectively placed on them at equal intervals, the patient being requested to tell how many squares are visible on any one exposed strip, and the moment at which they come into view when brought along the quadrant of the perimeter.

We have as yet no satisfactory method of testing the light sense (in the exact meaning of that term) in indirect vision, but it is scarcely necessary to point out that after all vision for white and colours has disappeared in any scotomatous area, and when even hand movements are no longer perceived, some perception of bright light may still be retained.

In hemianopsia it is usual to find that all the senses are greatly subdued. If no perception of light remains in the affected half-fields the hemianopsia is said to be *absolute*; and the same adjective may be applied to any other form of scotoma which is entirely destitute of sensation.

If, however, some perception remains, the defect is said to be *partial*.

Some disturbances of the field of vision which affect the

more central parts of the field, and are most readily detected by a colour test, still remain to be mentioned.

In practice these may be diagnosed so much more expeditiously with a small campimeter rather than a perimeter, that we shall conclude this section with a brief account of this simple apparatus and its use.

The Examination of the Colour Sense in the Pericentral parts of the Retina

For this purpose a piece of stiff black pasteboard about 18 in. square may be conveniently employed, having a small white spot marked at the centre of its surface.

A narrow strip of similar material, 6 or 8 in. long, should also be prepared, with a small disc of one of the fundamental colours (red, green, blue and yellow) affixed near the free end of each surface.

The patient stands with his back to the window. The physician, facing him at a distance of 2 ft., holds the board in one hand against his own breast, requests the patient to cover one eye and fix the gaze of the other on the central white spot. The smaller the test object and the greater the distance from the patient at which it is exposed, the more delicate and searching becomes the examination. Very minute scotomata may sometimes be detected in this way only.

Taking the colour-bearer in his free hand, the physician quickly applies one end to the centre of the board, so that the white spot is now replaced by a coloured disc of (say) red, and invites the patient to name it, or at least to say whether it appears grey or has any colour. Whether the reply be satisfactory or not, the disc should next be moved radially outwards in different directions, care being taken that the patient's eye does not follow it or wander from the fixing-point. Now, if in any eccentric position the colour is more clearly perceived than at the centre, the colour sense at the macular region is certainly at fault, since normally it

is most acute at this part, and, provided ophthalmoscopic examination reveals no opacity in the media nor visible change in the fundus to account for it, we may conclude that the nerve fibres supplying the fovea centralis are morbidly affected at some part of their course.

With a little care the extent and shape of the colour-scotoma may be mapped out.

If central, involving the point of fixation and its immediate neighbourhood irregularly, but extending chiefly to the inner side (nasal side of field), the case is probably one of inflammation affecting the optic nerve in the orbit—retrobulbar neuritis.

If extending chiefly to the outer side of the fixing-point, and corresponding to (*i.e.* subtending) that part of the retina between the optic disc and macula, known as the "papillo-macular area," the case is almost certainly one of toxic amblyopia, due to the abuse of tobacco, and perhaps alcohol.

Retrobulbar neuritis usually affects only one eye, while toxic amblyopia commonly, if not always, affects both, and the scotoma is symmetrical.

The test should be made with red and green, for blue and yellow are generally well perceived, except in severe cases.

It should be borne in mind that *congenital colour blindness* affects from 2 per cent. to 3 per cent. of the male population of this country, and a less proportion of females.

It is commonest for red and green, but may be distinguished from the foregoing by the fact that *per se* it in no way affects the direct visual acuity for form as tested, *e.g.*, by Snellen's types.

If, however, in a congenitally colour-blind person, from any cause, the form sense has been rendered defective, a proper diagnosis might still be made by remembering that congenital colour blindness affects the whole field, while in the above affections, if uncomplicated, good colour perception is retained beyond the limits of the scotoma.

With this simple apparatus the various forms of hemi-

anopsia, if approximately complete in extent, may be also readily diagnosed, and practically it is only when we wish to determine precisely the state of the peripheral parts of the fields for different colours, or require a permanent record, that we need have recourse to perimetry.

Phosphenes.—This term is applied to the visual sensations induced by mechanical stimulation of the retina—*e.g.*, the pressure of the tip of the little finger on the outer side of the globe occasions the sensation of a bright crescent with a dark centre in the inner part of the field.

In an intelligent individual it is possible to make use of this as a test of the retinal capacity for stimulation when from any cause it is not possible to see the fundus clearly.

Sense of Taste

The sense of taste is somewhat complex. The posterior third of the tongue with the fauces and pharynx are brought into connection with the centres by means of the glosso-pharyngeal nerves; the anterior two-thirds are supplied by the lingual nerve. The sensory branches concerned with taste pass up the lingual as far as the point where it is joined by the chorda tympani, by which they proceed. Their farther course is a matter of dispute, some observers holding that the fibres pass into the fifth nerve to reach the brain, while others regard the seventh nerve as the path followed by the taste fibres. The former view is probably correct. Subjective perversions of the sense of taste are rarely met with except in mental disease.

The sense of taste is tested by the use of substances devoid of aroma—such as sweet, bitter, sour, or salt bodies. Finely powdered quinine, salt and sugar may be used. The patient protrudes the tongue, and one of the test substances is gently rubbed in at the point to be tested. The patient keeps the tongue protruded, while the observer asks him the nature of the substance applied. Is it salt, sweet, or bitter?

the answer being given by a motion of the head, or in writing. After testing one point of the tongue with one of the test substances, the mouth should be rinsed with water. A convenient method of testing the sense of taste is with a weak galvanic current, which normally produces a metallic-like taste.

Hyperæsthesia of the sense of taste is rare, but may be met with in hysteria.

Anæsthesia, or loss of the sense of taste, may be peripheral, from coating of the tongue, abnormal dryness, or the action of heat or cold. It may be due to defective conduction, from disease of the nerves in their course. Destruction of the centre of the fifth nerve, or of its cranial root, produces loss of the sense of taste.

When loss of the sense of taste involves the anterior two-thirds of the tongue it is due to lesion of the fibres of the lingual nerves. When taste is lost in the anterior two-thirds, accompanied by loss of tactile sensibility, and without other indications of affection of the fifth nerve, the lesion is in the lingual nerve. When taste is lost without affection of tactile sensibility, the lesion is in the chorda tympani. Such a condition is seen in cases of middle ear disease. When facial paralysis accompanies the loss of taste the lesion is between the geniculate ganglion and the point of emergence of the chorda tympani. When paralysis of the second division of the fifth accompanies the loss of the sense of taste, the lesion is in the fibres between the spheno-palatine ganglion and the Gasserian ganglion. When the root of the fifth nerve near its point of emergence is the seat of the lesion, loss of the sense of taste is accompanied by anæsthesia of the whole area supplied by the fifth nerve.

The Sense of Hearing

The first object of the physician is to test the amount of hearing power actually present. This is done by three

methods, all of which should be employed in every case so far as practicable.

(1) **The Watch.**—For this purpose a watch is used which has been tested on a number of healthy individuals. Let us assume that the one employed is normally heard at 30 in. It is put at this distance from the patient's ear and gradually approached until its ticking is perceived. Assuming, for the sake of demonstration, that the distance is then 12 in., the hearing power may be conveniently expressed as $\frac{12}{30}$.

(2) **Conversation.**—It is next necessary to test the power of hearing conversation. This is best done by directing the patient to sit with the ear it is desired to examine towards the experimenter, while the other is occluded by the finger. Words are then whispered or spoken as the physician gradually approaches until they are heard and repeated. It is thus noted what is the distance at which whispered or ordinary conversation is heard. Sometimes only a loud voice near the ear is perceived, and occasionally not even that.

(3) **The Tuning-Fork.**—The tuning-fork test depends upon the fact that the normal ear perceives vibrations as musical notes when repeated at regular intervals from 16 to 32,500 vibrations per second. The limits vary in a characteristic way with disease of the conducting or perceiving apparatus. Again, there is a fairly definite ratio in the normal ear between the duration and loudness of the tuning-fork vibrations by air and by bone conduction, and this ratio is altered more or less definitely according to the part of the ear affected by disease. The examination is most easily carried out with the tuning-fork for the lower tone limits, with **Galton's whistle** for the upper tone limits.

Weber's Test consists in placing a vibrating tuning-fork upon the middle line of the head, either at the vertex, forehead, or upper incisor teeth. If the hearing is impaired in one ear only, or unequally in the two ears, and the vibrating fork is heard *better* in the worse hearing ear, the lesion is an obstructive one, *i.e.* in the sound conducting apparatus, not

in the nerves. *Vice versâ* if the sound is heard worst in the worse hearing ear, then the trouble is in the perceptive apparatus.

Jenné's Test.—If the conducting apparatus in any given case is normal, and a vibrating fork is pressed upon the mastoid until it ceases to be heard by bone conduction, and is then held opposite the meatus, it is heard by air conduction. If the conducting apparatus is affected to any marked extent, the vibrating fork, allowed to die away on the mastoid, is not heard when brought opposite the meatus. In labyrinthine affections, on the other hand, the fork is again heard when held in front of the meatus. In many cases Weber's and Jenné's tests give valuable information, yet there are many doubtful, or border-line, cases where they cannot be relied upon.

Examination of the Ear

The inspection of the meatus and tympanic membrane is best done by using a reflector (focal distance about 5 in.) and a suitable ear speculum. The best light for the purpose is got from a window on a tolerably clear day; a candle will, however, be found sufficient. The patient is seated with the ear to be examined away from the light. A speculum is then introduced into the meatus up to the commencement of its osseous portion, the auricle being at the same time pulled upwards and backwards in order to straighten the canal. Any obstruction in the latter, if present, will in this way be seen; otherwise a view of the membrana tympani is obtained. This structure *in the normal condition* shows—(1) The handle of the malleus, running from its anterior and upper part downwards and backwards to a little below the centre as a yellow unmistakably osseous ridge. (2) Meeting this at an obtuse angle (open anteriorly) is seen the bright spot, usually triangular in shape and always conspicuous by its lustre. (3) The mem-

brane itself, oval in shape, and bluish-grey in colour. (4) The upper part of the handle of the malleus called the short process, from which run two delicate folds — the more marked passing backwards, and the other forwards (the posterior and anterior folds).

It would be useless here to discuss the various *deviations* from the normal met with; those who desire information on these points we must refer to works on otology.

Having obtained a view of the drum membrane, it may be desirable to test its **mobility.** This can be done by directing the patient to hold the nose, and blow. If the Eustachian tubes be pervious the membrane can be seen to move if inspected during this act. If this experiment (known as Valsalva's) be impossible, then air must be driven through the Eustachian tube by means of Politzer's method (or one of its modifications), or the Eustachian catheter, by an assistant while the ear is inspected; if no assistant be at hand, a pneumatic or suction speculum (known as Siegle's) may be employed.

In order to determine **the condition of the Eustachian tube** it is desirable to drive air from the nose into the middle ear, while the ear of the observer is connected with that of the patient by means of an auscultating tube. For therapeutic purposes this may be accomplished by Politzer's method, which consists in introducing the nozzle of an indiarubber bag into one nostril, while so much of it as is not filled, together with the opposite nostril, is compressed with the finger and thumb. The patient is now directed to swallow a little water which has been previously taken into the mouth. At the moment of swallowing, the bag is suddenly compressed, and air is felt by the patient and heard by the operator to impinge upon the drum membrane. Instead of swallowing, the patient may be directed to say *huck*, blow out the cheeks, etc. Fortunately the act of crying also serves the same purpose.

If, however, it be desired to appreciate fine differences in

the sounds produced by air passing through the Eustachian tube, it is better to pass the Eustachian catheter. This is a curved instrument having at its outer extremity a wide orifice, into which the nozzle of an air-bag fits. At this end there is also a ring corresponding to the curve on the beak. The best method of introducing the catheter is to pass it along the floor of the inferior meatus of the nose until it reaches the posterior wall of the pharynx; the beak is then turned inwards and the instrument withdrawn until the curve hooks upon the septum of the nose. If the catheter be now rotated until the ring (which corresponds to the point) is directed towards the outer canthus, the instrument will, as a rule, enter the Eustachian tube. It must here be mentioned that difficulties are often met with in catheterisation of the Eustachian tubes, and then other methods must be employed, such as will be found described in any work on diseases of the ear.

After the catheter is *in situ*, and while the ear of the observer is connected with that of the patient, air is driven through the instrument by means of an indiarubber air-bag. The following auscultatory phenomena are capable of detection by a trained ear.

1. A full sound of air impinging against the drum membrane.

2. A moist sound far away from the ear, if there be mucus in the pharyngeal orifice of the Eustachian tube.

3. A faint moist sound associated with the impact of the air against the drum membrane in some cases in which the tympanum contains fluid.

4. A dry whistling sound in narrowing of the Eustachian tube.

5. Air whistling through a perforated drum membrane.

Beyond mentioning that bougies are sometimes passed through the Eustachian catheter into the Eustachian tube, we shall not discuss this method of exploration, which should be rarely used, and never except by a skilled aurist.

Examination of the Larynx

For examination of the larynx there are required—(1) A laryngeal mirror; (2) a powerful light.

It is unnecessary to describe the *laryngeal mirror*, which is now so well known. It is enough to say that several sizes should always be at the physician's command, and further, that it is, for obvious reasons, well to use one as large as can be tolerated by the patient.

As to *light*, it is usually well to employ a powerful fixed light, the rays of which are concentrated on the mirror by means of a reflector. The last named may be attached either to a forehead band, or kept in position by a spectacle frame. It should have a central aperture, which, when the mirror is adjusted, should be opposite one of the observer's eyes, so that he can look through it. The actual source of light may be a strong argand burner, a Welsbach or albo-carbon light, or a powerful lamp. The electric and oxy-hydrogen lights are good. The best source of light is the sun. As most reflectors are concave, it must be borne in mind that an exact focussing of the rays should be avoided; otherwise the excessive heat produced will cause the patient pain.

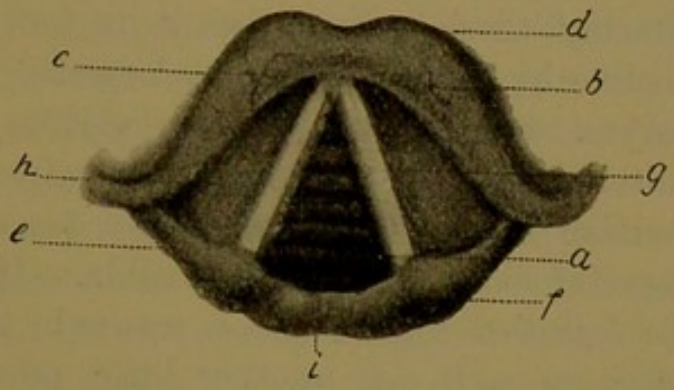


Fig. 143.—Larynx in quiet respiration. *a*, Vocal cord; *b*, anterior commissure; *c*, region of cushion of epiglottis; *d*, epiglottis; *e*, ary-epiglottic fold; *f*, arytenoid cartilage; *g*, ventricle of Morgagni; *h*, false cord on ventricular band; *i*, inter-arytenoid fold.

Method.—The patient is seated so that the light falls over his shoulder (which side is immaterial) on about a level with his ear. The physician sits opposite him, and so adjusts the reflector that a strong light is directed upon the mouth, which must now be opened. The tongue is then

protruded, and held either by the observer's left hand or by the patient. To facilitate this, and prevent the fingers from slipping, a pocket-handkerchief is employed, which, together with the hand, must be kept below the lower lip, to avoid shutting off light. The patient is now directed to breathe quietly, and the mirror—previously slightly warmed over the flame—is introduced so that it lifts upon its posterior surface the uvula. The handle of the mirror should be held like a pen, and until the uvula is reached the reflecting surface points downwards; it is then, however, held more vertically, and the angle which it forms with the horizon is gradually increased until the larynx is brought into view. During these manœuvres it is well to make the patient incline his head slightly backwards. The first part of the larynx seen is usually the epiglottis, which appears in the anterior or upper part of the mirror, while in its lower portion the arytenoid cartilages (surmounted by the cartilages of Santorini) become visible. If now these last named be followed forwards (*i.e.* upwards in the mirror) there is seen on each side another little prominence (the cartilage of Wrisberg). Finally, the elevated fold of mucous membrane (the ary-epiglottic fold) runs up to the epiglottis. Immediately inside of this appear two other folds of mucous membrane (the false cords or ventricular bands). If the patient be now told to say *eh*, the epiglottis is thrown up, and the false cords come more plainly into view, while the true vocal cords are seen as two white and glistening bands, separated from the ventricular folds on each side by a recess (the sinus of Morgagni). On inspiration the cords are seen to separate, while on expiration they tend to approximate. On phonation they are perceived to be in actual contact. There is one portion of the larynx which can only be properly studied on full inspiration, namely, the fold of mucous membrane between the arytenoid cartilages (the inter-arytenoid fold or posterior commissure). The anterior commissure is best seen, in most persons, just before

phonation; here the vocal cords meet, and just above them is a small prominence (the cushion of the epiglottis). During phonation, too, is seen on each side the sinus pyriformis, which lies between the ary-epiglottic folds and the outer wall of the pharynx. The upper surface of the epiglottis should also be examined. It shows a central ligament (the middle glosso-epiglottic) and on each side a fossa (vallecula).

Certain obstacles are often met with in attempting to make a laryngoscopic examination:—

1. The patient, owing to nervousness, may hold the breath. This difficulty is usually easily overcome by directing that respiration should go on as usual, and pointing out that it will not be interfered with by the introduction of the mirror.

2. Abnormalities of the tongue.—In some persons the dorsum linguæ arches itself up so as to leave little or no space between the tongue and the palate. In a few of these cases a tongue depressor has to be employed in order to obtain room for the introduction of the mirror. Occasionally the frenum is so short as to prevent protrusion beyond the lower incisors. Where this is the case our only course is to employ a tongue spatula, and then introduce the mirror in the ordinary way.

3. Hyperæsthesia of the pharynx may be so marked as to make laryngoscopic examination well-nigh impossible. This obstacle can almost invariably be overcome by painting the pharynx and base of the tongue with a solution of cocaine (10 to 20 per cent.).

4. The epiglottis in some persons has a tendency to overhang the larynx and obscure the image. The intonation of *e* may cause it to rise, or, failing this, several rapid short respirations. In exceptional cases these means fail, and it is then necessary to spray the throat with a solution of cocaine, and afterwards, holding the mirror in the left hand and making the patient hold his tongue, to elevate the epiglottis by means of a laryngeal probe introduced with the right hand. This proceeding, however, requires for its

satisfactory execution considerable practice in laryngoscopic manipulation. External palpation of the larynx is sometimes of use—especially in cases of displacement or suspected fracture.

Results.—In examining the larynx it is necessary to consider deviations from the normal as to—(1) Colour, (2) quantity of secretion, (3) swelling or hypertrophy, (4) the presence of tumours (or foreign bodies), (5) localised infiltrations, (6) ulcerations, (7) mobility.

To discuss each of these points would necessitate more space than is at our command. The last mentioned, however, requires a few words of explanation. On normal inspiration the vocal cords are separated, on normal expiration they are approximated, and on phonation they are in contact. It is therefore necessary in each case to study the movements of the cords outwards on inspiration, and inwards on phonation. It must be remembered that it is—in cases of paralysis due to involvement of the recurrent nerve—common for the affected cord to remain immobile in the position of phonation, so that the paresis is only detected on inspiration.

Internal palpation by means of the finger is necessary, particularly in young children, in whom laryngoscopy is often impossible, when the presence of a foreign body is suspected. This method—which must, of course, be very rapidly executed—may also prove of assistance in the diagnosis of œdema glottidis, when the epiglottis and ary-epiglottic folds may be felt as tense prominences.

Sounding or probing the larynx with a bent laryngeal probe directed by means of the laryngoscope is often very valuable. The tongue being held by the patient, the mirror is introduced with the physician's left hand, and the probe, which is bent at a suitable angle, with the right. This method is serviceable in detecting the consistence and attachment of tumours, and also in diagnosing the presence of necrosis. Auscultation in the ordinary sense of the term

is not often practised as an aid to laryngoscopic diagnosis. Any interference with the voice should, however, indicate the propriety of a laryngoscopic examination. It must also be borne in mind, however, that paresis of one vocal cord may exist without any interference with phonation.

Dyspnœa due to laryngeal disease is commonly chiefly inspiratory, but in subglottic tumours, it may, on the other hand, be expiratory.

Laryngeal cough, especially in children, is characterised in many cases by a peculiar metallic dulness, often spoken of as "croupy."

In doubtful ulcers of the larynx it is often well to remove some of the secretion from their surface by means of a brush or pledget of cotton wool (attached firmly to a suitably bent forceps), and then to examine the secretion so removed for *tubercle bacilli*.

The Nose and Naso-Pharynx

In examining the nose and naso-pharynx we trust as much as possible to sight.

Anterior Rhinoscopy.—This method of examination is carried out with the aid of a strong light and a nasal speculum. In order to concentrate the light upon the nose a laryngeal reflector is employed. The patient is seated, as in laryngoscopy, facing the physician. A speculum is then introduced, and that known as the Duplay-Charrière is probably the most efficient. When it is used, however, the nose should again be inspected by simply tilting up the tip, in order to exclude a perforation of the anterior part of the septum, which may be covered by one of the blades. The speculum, which consists of two valves separable by means of a screw, is gently introduced straight backwards and then dilated. By this means the inferior turbinated body is seen extending as a thick fleshy mass from the outer wall of the nose towards the septum. In not a few persons this struc-

ture, which is composed in great part of erectile tissue, is so large that it obscures everything else, and is in actual contact with the septum. This enlargement may be due to hypertrophy or to simple erection. If the latter only be present then the application of a solution of cocaine (10 per cent.) will reduce the structure to its normal dimensions. The anterior end and mesial surface for a considerable distance backwards may then be inspected. If, now, the patient be directed to turn so that the speculum is directed inwards, a considerable portion of the septum can be examined. If it be desired to inspect the floor of the inferior meatus, the patient is directed to bend the head forwards; while, in order to see the middle turbinated body, the head is thrown backwards. The last-named structure then appears as a shelf, extending from the outer wall almost to the septum. Between the two there appears, however, in health a narrow orifice—the olfactory cleft. In order to determine the consistence or point of attachment of any tumour, the presence of carious bone, etc., a probe, guided by the eye, is employed.

Posterior Rhinoscopy.—When it is desired to *examine the posterior nares*, we require a reflector, a small laryngeal mirror (the angle of which, with the shaft, should be somewhat smaller than in the form commonly employed for laryngoscopy), and a tongue depressor. The patient being seated by the side of a strong light, the tongue is depressed, the mirror warmed, and introduced while light is concentrated on the throat. The mirror must be so placed that its reflecting surface is not obscured by the uvula, and points upwards and forwards. If there be ample space between the palate and posterior wall, posterior rhinoscopy is not very difficult, while in some persons it is, owing to insufficient space, impossible. In such cases Voltolini's palate hook will be found useful. In others the palate tends to rise too much; the patient must then be directed to breathe through the nose, or else to intone *ng* (or the French word

on). If the parts be extremely irritable, cocaine may be applied, as in laryngoscopy. When the mirror has been introduced the first object which usually catches the eye is the septum of the nose (or vomer). To each side of this can be seen the middle turbinated bodies, and below them the upper part of the inferior turbinateds in their posterior aspect come into view. The septum appears as a pale ridge widening at its upper part, while the turbinated bodies are tumour-like and of a more bluish-grey tint. If now the mirror be turned towards the side so that its reflecting surface points upwards and outwards, the orifice of one Eustachian tube is seen as a dimple, bounded behind and above by an elevated ridge, posterior to which lies a depression known as the fossa of Rosenmüller. In order to see the vault of the naso-pharynx, the reflecting surface must be directed upwards as much as possible. Rhinoscopy, applied to the posterior nares, is by no means an easy manœuvre, and an idea of the whole naso-pharynx and posterior nares can only be got by piecing together the fragments visible in the small mirror employed. Various appliances are employed for holding forward the soft palate so as to obtain more space, but these methods can only be of use to those who have had considerable practice and experience.

It has also been proposed to examine the posterior nares by means of a long speculum introduced through the anterior nares. This method of examination is, however, rarely practised.

In many cases, especially when rhinoscopy is unsuccessful, it is well to explore the posterior nares with the index finger. The observer first washes the hands. The patient being seated, the observer stands at his patient's right side, and steadies the head by passing his left arm behind it. The index finger of the right hand is then introduced into the mouth, slipped behind the palate, and the posterior nares rapidly explored. To describe the feeling of the various parts would serve no good purpose; practice alone will

enable the physician to derive information from this method of examination. Care must, however, be taken not to mistake the prominent orifices of the Eustachian tubes for tumours. Their occurrence on both sides, and their cartilaginous consistence, will help to prevent such an error.

When examining children it is well to push the cheek between the teeth with the left hand in order to avoid being bitten.

Other objective methods of examination are subordinate to sight and touch applied directly to the part; but in certain cases they are of importance as indicating the necessity for an examination of the nose. Thus a nasal character of the voice, a constantly open mouth, and hanging lower jaw, heavy breathing through the nose, snoring at night, and an offensive discharge from one or both nostrils, are symptoms which are often prominent in nasal disease.

CHAPTER XII

CLINICAL BACTERIOLOGY

The important position which bacteriology now takes in medicine, renders a certain knowledge of the methods employed in diagnosis essential to the physician. The isolation and recognition of micro-organisms is, it is true, necessarily carried out in the laboratory by the specialist, but the student must know how to obtain material for examination, how to examine the material when obtained, and how to stain and recognise the commoner pathogenic organisms.

Clinical bacteriology should include—

(1) The examination of the blood.

(2) The examination of pathological secretions and excretions.

(3) The examination of the products of inflammation.

The Examination of the Blood

The examination of the blood for micro-organisms must be carried through, as indeed must all bacteriological observations, with the greatest attention to technical detail. If the blood is to be obtained by puncture the observer first cleanses his hands. The part to be punctured is next selected. The skin is carefully washed with soap, water, and a nail brush, and dried. It is next scrubbed with a mixture of alcohol and ether, then with a solution of perchloride of mercury (1 in 1000), and the mercury finally removed by washing with alcohol. The part is then dried

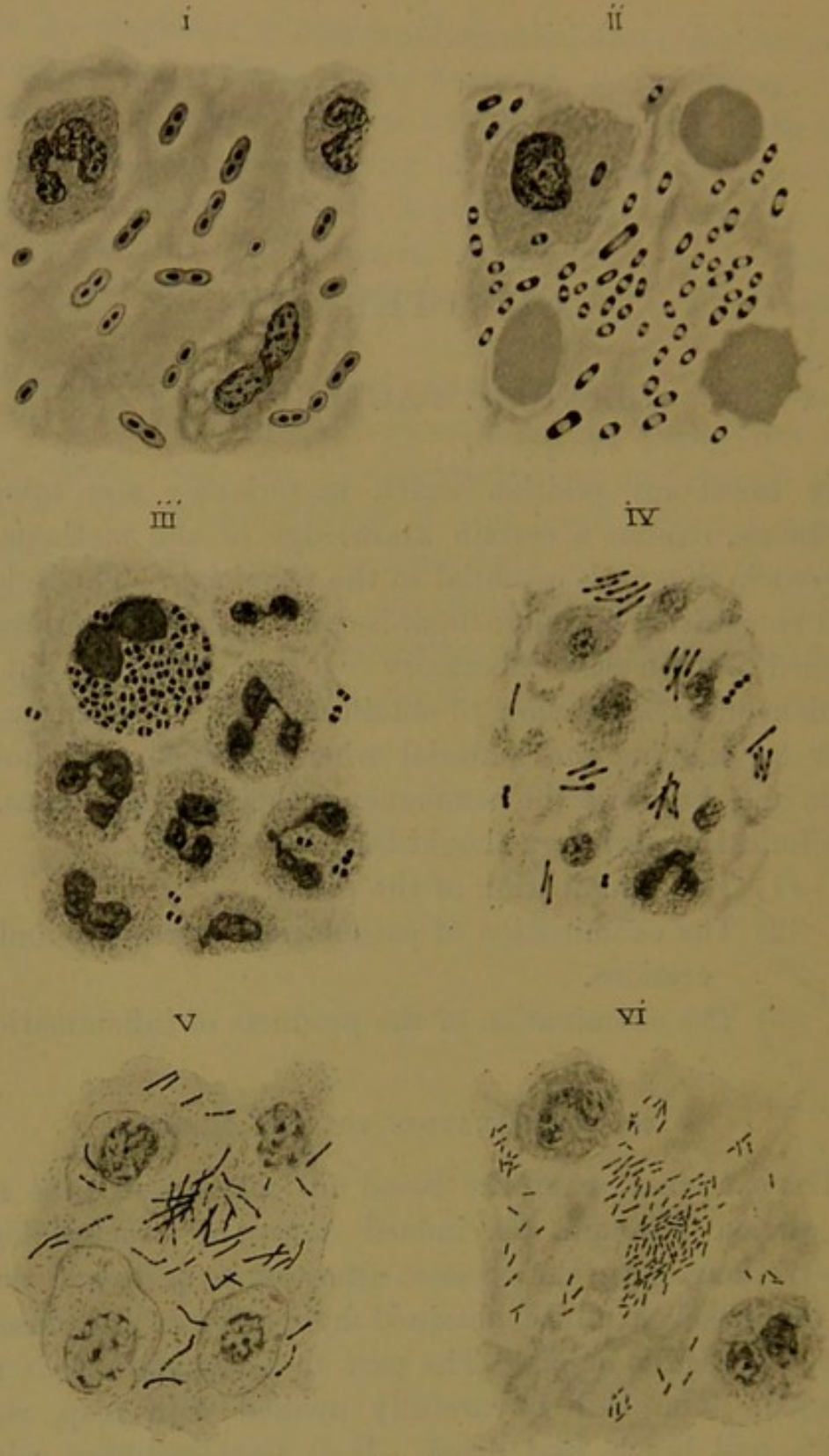


FIG. 144.

i. Film from sputum—case of pneumonia. Stained to show capsules. × 1000.
ii. Film from sputum—case of pneumonic plague. *B. pestis* shows polar staining. × 1000.
iii. Film from pus—case of gonorrhœa. Gonococci in leucocyte. × 1000.
iv. Film from throat—case of diphtheria. Neisser's stain to show bipolar staining. × 1000.
v. Film from sputum—case of acute phthisis. Neelsen's stain. × 1000.
vi. Film from sputum—case of influenza. Stained carbol-fuchsin. × 1000.

with sterile gauze or cotton wool. The part is next pricked with a needle or tenotomy knife, which has been sterilised in the flame or by washing with the perchloride solution. The first drop of blood which exudes is rejected lest there be any possibility of contamination, subsequent drops being used. A better method of obtaining blood for examination is to puncture a vein and draw off a small quantity of blood. The skin over the selected vein is sterilised as above. A ligature having been placed round the limb above the point selected, the needle of a sterilised hypodermic syringe is then passed into the vein, the point of the needle being directed towards the periphery. A quantity of blood can thus easily be drawn off for examination with very little discomfort to the patient. It is rarely necessary to make use of splenic puncture to obtain blood for examination. The operation should only be carried out after due consideration of the individual case and the necessity for the procedure.

The sample of blood having been obtained by one of these methods is next examined. Clean cover-slips and slides, prepared by one of the methods described under the microscopic examination of the blood, being in readiness, a fresh film is made and examined under the oil immersion lens. The micro-organisms which may be found in a fresh film from the circulating blood are the spirillum of relapsing fever, the anthrax bacillus, and the malaria plasmodium.

A dry film is next fixed and stained. Fixation may be with the formalin and alcohol solution, or with the alcohol and sublimate solutions recommended by Muir and by Gulland, or by heat, passing the film three times rapidly through the flame of a bunsen.

Muir's Method.—The film is floated face downwards on a saturated solution of perchloride of mercury in 0·75 per cent. sodium chloride. The film remains in this solution for five minutes and is then washed, for at least half an hour, in 0·75 sodium chloride solution to remove the

mercury, and then passed through successive strengths of methylated spirits. The film is then stained.

Gulland's solution consists of—

Absolute alcohol	25 c.c.
Pure ether	25 „
Alcoholic solution of corrosive sublimate (2 grms. in 10 c.c. alcohol)	5 drops.

The films are fixed for five minutes, well washed with water, and are then ready for staining. The staining of the film may be carried out with methylene-blue and eosin, carbol-thionin, carbol-fuchsin, or by Gram's method. Of methylene-blue, Löffler's alkaline solution gives the best results. It consists of—

Saturated alcoholic solution of methylene-blue	30 parts.
1 in 10,000 aqueous solution of caustic potash	100 „

Gram's Method.—The following solutions are required—

(1)

Aniline oil	4 c.c.
Distilled water	100 „

Shake well together, filter, and add 11 c.c. of a concentrated alcoholic solution of gentian violet, shake and set aside for twenty-four hours.

(2) Gram's solution—

Iodine crystals	1 part.
Potassium iodide	2 parts.
Water	300 „

The cover-slip is taken in a pair of Cornet's forceps, flooded with solution No. 1, and gently *warmed* over the flame of a bunsen for two to five minutes, drying being prevented by adding more of the solution if necessary. Next transfer the slip to a watch glass containing solution No. 2 for one-half to two minutes. Wash in 95 per cent. alcohol until the blue colour is almost or wholly lost. Next,

counter-stain with Bismarck-brown (saturated solution in equal parts of water and alcohol) for two minutes, or with eosin; wash, dry, and mount in xylol-balsam. The method gives beautiful results, but does not stain a number of bacteria, including the spirillum of cholera, the bacillus of glanders, of malignant œdema, of pneumonia (Friedländer), and of typhoid fever, the micrococcus of gonorrhœa, and the spirillum of relapsing fever.

The following micro-organisms may be found in the blood—

Anthrax bacilli.
Tubercle bacilli.
Typhoid bacilli.
Malta fever bacilli.
Plague bacilli.
Yellow fever bacilli.
Glanders bacilli.
Influenza bacilli.
Relapsing fever spirilla.
Malaria plasmodia.
Pneumococci.
Gonococci.
Pus cocci.

Tubercle bacilli are exceedingly rare in the circulating blood, so rare in fact, that search for them is hopeless. Typhoid and Malta fever bacilli may be obtained and cultivated from the splenic blood, but are rarely found in the circulating blood. Influenza bacilli are best found in the sputum; glanders, anthrax, and plague bacilli do occur in the blood, but can better be demonstrated in the fluids obtained from the local foci of infection. Pus cocci can be more readily demonstrated by cultivation. The spirilla of relapsing fever and the malaria plasmodia not being susceptible of cultivation outside the body in the present state of our knowledge, can only be demonstrated in film preparations.

Culture Methods are employed for the detection and recognition of the different organisms which may occur in the blood, especially the pyogenic organisms, in cases which it is proposed to treat by serum therapeutics. For the methods and culture media required, the student is referred to the different text-books of bacteriology.

The Agglutination-Reaction of the Blood Serum.—In certain diseased conditions, such as typhoid fever, Malta fever, general infection with the *B. coli*, the blood serum of the infected individual acquires the power of acting upon the specific organism of the disease. When the specific organism is brought under the influence of the blood serum (*a*) motility is lost, (*b*) agglutination takes place. This power of the blood serum is of the greatest importance as an aid to diagnosis. In typhoid fever it is known as Widal's reaction. In carrying out the observation the blood serum must be diluted, as undiluted healthy serum will in some cases give agglutination. The diluting medium for the serum may be normal saline solution or bouillon. It is not necessary to centrifuge the blood, as was originally done, as the presence of blood corpuscles is no disadvantage. The ear or finger of the patient having been cleansed is pricked and a drop of blood obtained. Using the leucocyte pipette of the Thoma-Zeiss hæmacytometer, the blood is drawn up to the mark 0·5 and the diluting medium added to the mark 10. This gives a dilution of 1 in 20. Take a cover-slip and on it deposit a small drop of saline solution or bouillon with a small-sized platinum loop. With a platinum needle take a small quantity of a twenty-four hours' growth of the *B. typhosus* and mix in the saline or bouillon to form an emulsion of the organism. To this emulsion add two loopsful of the diluted serum. The dilution is now 1 in 30. Mount as a hanging-drop preparation, ringing the specimen with water, or if the specimen is to stand in a warm, dry atmosphere, ring with vaseline to prevent evaporation. The preparation is then examined under the microscope. The presence of the corpuscles renders focussing easy. The bacilli are seen very actively motile amongst the corpuscles. The specimen is examined from time to time. When agglutination occurs the bacilli are seen massed together in smaller or larger clumps, and have lost their motility. In a case of typhoid fever the agglutination

occurs within two hours, the time varying in different cases. If, with a dilution of 1 in 30, agglutination is not present in two hours the observation is considered negative. Having obtained agglutination and noted the time necessary to obtain the reaction, it is desirable to ascertain the greatest dilution of the serum which will produce agglutination. This is easily done by using the original diluted serum (1 in 20) with different proportions of saline solution or bouillon to increase dilution. In making the observation it is always desirable to have control observations with normal serum.

In examining the serum of suspected Malta fever or *B. coli* infection the observation is carried out on similar lines.

The Examination of Pathological Secretions and Excretions

Sputum.—In the examination of the sputum the micro-organisms which are of the greatest importance to the physician are the tubercle bacillus, the specific micro-organisms of pneumonia, the influenza bacillus, and the plague bacillus.

Fungi—moulds, yeast, fission fungi, and sarcinæ do occur, but are rare. Actinomyces occur when the lung and pleura are affected with the fungus, but the disease is rare.

The **tubercle bacillus** is the most important pathogenic organism found in the sputum. To demonstrate the bacillus, advantage is taken of the fact that it is "acid fast," that is to say, the staining material, fuchsin, is not washed out by treating the film with a dilute mineral acid. Some sputum is taken in a shallow vessel and a small portion of one of the muco-purulent masses is selected, and with a needle or forceps transferred to a cover-slip. A second cover-slip is applied and a thin film spread out by pressure. If too large a proportion of sputum be taken the surplus which is expressed is removed with filter paper, and the paper

immediately burned. The cover-slips are then slid apart, when a thin film should adhere to each. The film is allowed to dry in the air, then taken in forceps and fixed by passing three times in rapid succession through the flame of a bunsen or spirit lamp. The film is now ready for staining. The following solutions are required—

(1) Fuchsin . . . . . 1 part.
Absolute alcohol . . . . 10 parts.
Dissolve and add 5 per cent. aqueous solution of phenol . . . 100 ,,
(2) 20 per cent. solution of sulphuric acid.
(3) Saturated watery solution of methylene-blue.

Solution (1) is taken in a watch glass and the film floated on face downwards and stained for at least five minutes. The staining is aided if the solution be gently warmed till it steams by holding some distance above the flame of a bunsen, care being taken not to overheat lest the alcohol ignite. When the film is sufficiently stained it is removed, mixed in water, and decolorised by treating with the acid solution. Two small porcelain evaporating basins are taken, one with the acid solution and one with water. The cover-slip grasped in forceps is rinsed in the acid solution which removes the colour, then in water, when some of the colour may return. It is again treated with acid till on rinsing in water no colour returns. The film is next stained with the contrast stain — methylene-blue for half a minute, then washed in water. If a permanent specimen is not desired the film while still moist is placed upon a slide, the surplus water removed with filter paper and examined. If a permanent specimen is desired the film is dried in the air, passed rapidly through the flame of a bunsen to complete the drying, and mounted in xylol-balsam. The balsam is most easily manipulated if used of considerable consistency and spread out by warming the slide over the flame of a bunsen after the film is in position.

On examining the film under the high power, or better, with an oil immersion lens, the tubercle bacilli, if present, will be found stained of a rose-pink colour, while the ground of the preparation is blue, stained with the methylene-blue.

The **pneumococcus** (Fränkel) occurs in the sputum of acute pneumonia in the form of small oval cocci about 1 μ in longest diameter, arranged generally in pairs, but also in chains of four to ten. The free ends are often pointed like a lancet, hence the term *Diplococcus lanceolatus* has often been applied to it. The cocci have a capsule which usually appears as an unstained halo.

In film preparations the cocci may be easily stained with weak solutions of any of the basic aniline stains or by Gram's method. The best method of staining to demonstrate the capsule of the cocci is probably that devised by Richard Muir. The essential part of the process consists in the use of a special mordant prepared by mixing 2 parts of a 20 per cent. watery solution of tannic acid, 2 parts of a saturated watery solution of mercuric chloride, and 5 parts of saturated watery solution of potash alum. A thin film of the sputum is spread and gently dried over a flame. A few drops of the mordant are filtered on to the film, allowed to remain for about two minutes, then thoroughly washed in water, next in methylated spirits, and again in water. The film is now stained in Ziehl-Neelsen's carbol-fuchsin for about two to three minutes, while gentle heat is applied. After washing in water the mordant is again applied for about two minutes, and washed off as before. Finally the film is stained for about two minutes in saturated watery solution of methylene-blue, washed in water, differentiated in methylated spirits, dried thoroughly, and mounted in xylol-balsam or else dehydrated in absolute alcohol, clarified in xylol and mounted in balsam. The bodies of the cocci appear red, while the capsules are stained blue. The pneumococcus is rarely absent from the sputum of acute croupous pneumonia.

The **pneumobacillus** (Friedländer) somewhat resembles

the pneumococcus. The form, however, is more of a short rod-shape, and has blunt rounded ends; it is rather broader than the pneumococcus. It is now usually classified amongst the bacilli. The capsule has the same general characters as that of the pneumococcus. It stains readily with basic aniline dyes, but loses the stain with Gram's method, and is coloured with the contrast stain—saffranin or Bismarck-brown. It can thus be distinguished from the pneumococcus. It is much less frequently present in the sputum of croupous pneumonia than the pneumococcus; sometimes it is associated with the latter; sometimes, but very rarely, it occurs alone.

The **influenza bacillus** as seen in the sputum is a minute rod not exceeding 1·5 μ in length and 0·3 μ in thickness. It is straight with rounded ends, and sometimes stains more deeply at the extremities. The bacilli may occur singly, but are usually aggregated into clumps, but do not form chains. They show no capsule. In film preparations they stain feebly with basic aniline dyes, and are best demonstrated by staining for five to ten minutes with a weak solution (1 in 10) of carbol-fuchsin. They lose the stain in Gram's method.

The **plague bacillus** occurs in the sputum in great numbers in the pneumonic form of plague. The bacilli are small oval rods, somewhat shorter than typhoid bacilli and about the same thickness; they have rounded ends, and in stained preparations a portion is sometimes left unstained in the middle of the bacillus, giving the so-called "pole staining." They stain readily with the basic aniline stains, but are decolorised by Gram's method.

The Urine.—In microbic diseases the organisms causing the disease are frequently excreted in the urine. This is well demonstrated in typhoid fever, when a pure cultivation of the *B. typhosus* can frequently be obtained from the urine,—indeed the bacillus can be more readily isolated from the urine than from the fæces. The term *bacteriuria* has been given to the urinary condition resulting from the

excretion of micro-organisms. To obtain the urine for bacteriological examination, the greatest care must be taken to, as far as is possible, prevent contamination. The parts should be washed with soap and water and then with corrosive sublimate solution. A sterile catheter is passed. The first portion of the urine drawn off is rejected, and a sample, taken from the later portion, is received into a sterile test-tube or flask, and from it cultivations are made. In making film preparations from the urine the centrifuge is always used. The precipitate is examined in the fresh condition and as stained films.

In acid cystitis the **Bacillus coli communis** is usually present as a causal agent. The bacillus closely resembles the typhoid bacillus, has the same staining reactions, and is motile.

In alkaline cystitis there is usually a mixed infection from **pyogenic cocci** and other micro-organisms. The cocci can be readily stained with methylene-blue or by Gram's method. The **tubercle bacillus** is present in tuberculous affections of the urinary tract. Care must be taken in obtaining the sample of urine for examination, as the **smegma bacillus** bears a close resemblance to the tubercle bacillus and has the same "acid fast" properties. If there is doubt as to identity of the organism, inoculation experiments would solve the difficulty.

The alkaline fermentation of healthy urine on standing is due to the **Micrococcus ureæ**, which contains a ferment capable of inducing the addition of water to the urea molecule and thus forming ammonium carbonate.

The **alimentary canal** contains many varieties of micro-organisms. Of these the most important from a clinical standpoint are the *B. coli*, *Sarcinæ ventriculi*, torulæ, and leptothrix. In addition there are numerous bacilli and micrococci. Micro-organisms when present in quantity in the stomach, as seen in the stasis of impaired gastric motility, exercise a deleterious influence upon digestion.

Sarcinæ are easily recognised under the microscope by their characteristic grouping like corded bales of wool. They may be present in gastric dilatation. **Torulæ** are round or oval cells many times larger than bacteria. They often reproduce by budding, a portion of the cell protruding, and finally being cut off to form a new individual. Spore formation also occurs. They are frequently present in the vomited matter in gastrectasis.

The Products of Inflammatory Exudation

The Serous Cavities.—Inflammatory exudations into the serous cavities are to be regarded as the result of the action of micro-organisms. The organism may be identified by (*a*) film preparations, (*b*) cultivation and isolation, (*c*) by its pathogenic effect in suitable animals.

Pleural effusion when serous is to be regarded as probably due to the action of the tubercle bacillus, but the abundant effusion of fluid with the paucity of the bacilli make their detection difficult.

Pleural effusion when sero-purulent or purulent may be due to the presence of more than one micro-organism. Amongst the most important are the pneumococcus, the *Streptococcus pyogenes*, the *Staphylococcus albus*, the *B. coli*, and the *Micrococcus tetragenus*. More rarely the typhoid bacillus or the influenza bacillus may be found. Of these the pneumococcus takes the first place even when the inflammatory process is not associated with acute croupous pneumonia. The organism in the purulent fluid is frequently found in a degenerated condition, but can usually be recognised on staining films by a method which demonstrates the capsule, such as Richard Muir's method.

Pericardial Fluid.—When the effusion is simple it rarely comes under examination during life. The tubercle bacillus may be the cause in some cases. In suppurative conditions any of the pyogenic organisms may be present, the infection

of the pericardium being secondary to suppuration elsewhere in the body. In primary suppurative pericarditis the pneumococcus may be the causal agent.

Peritoneal effusions, when inflammatory, are the result of organismal activity, the *B. coli* group taking a prominent place. In the female, especially when the inflammation originates in the pelvis, the gonococcus may be the active agent. At times the infection of the peritoneum is of so mixed a character that it is difficult to say which organism is the causal agent.

The different organisms which have been identified in peritonitis are the *B. coli* group, gonococci, pneumococci, streptococci, staphylococci, Friedländer's pneumobacillus, typhoid bacillus, tubercle bacillus, and the fungus actinomycoides.

Cerebro-spinal effusions in acute and subacute inflammation of the cerebral and spinal meninges may be obtained for examination by lumbar puncture. The tubercle bacillus, the most important in such effusions in children, is so difficult to find that negative results in film preparations are of no diagnostic value. The pneumococcus and other diplococci, such as the *Diplococcus intracellularis meningitidis* of Weichselbaum, present in epidemic cerebro-spinal meningitis and the diplococcus of Still, may be found, and are morphologically indistinguishable and closely resemble the gonococcus. Other organisms whose presence may account for the effusion include the pyogenic bacteria, the influenza bacillus, and the pneumobacillus of Friedländer.

Joint effusions may result from the invasion of the commoner pyogenic organisms (streptococci, staphylococci), or may be related to acute articular rheumatism, pneumonia, typhoid fever, scarlet fever, etc., or may be associated with gonorrhœa. The character of the effusion varies with the gravity of the infection. In the graver infections it is purulent. Film preparations made from the fluid may be stained by the different methods which have been described in the preceding pages.

Membranous exudations.—Fibrinous exudations on mucous surfaces, leading to the formation of false membrane, may result from the action of streptococci, but the most important and frequent causal agent is the **B. diphtheriæ.** The bacilli are slender rods, straight or slightly curved, their length about 3 μ to 5 μ, their thickness a little greater than that of the tubercle bacillus. Their size therefore varies somewhat. The bacilli are found in the false membrane and in the secretion of the pharynx and larynx in the disease.

In making the diagnosis in a given case a sterilised cotton-wool swab, or better still the loop of a platinum needle, is used, and with either instrument a small portion is removed from the edge of the suspected exudation. If no exudation be present a scraping of the mucous surface is taken. The swab or loop is then drawn lightly over the surface of a blood serum culture tube and incubated at a temperature of 37° C. If the bacillus is present a growth appears in twelve to twenty-four hours as an opaque whitish film. The growth from other organisms appears later than that of the diphtheria bacillus. Film preparations are made from the growth. If it is not practicable to make cultivations, films may be made from a portion of the suspected exudation. The bacillus stains with aniline dyes and retains the colour with Gram's method. The best stain for differentiating the bacillus in film preparations is that described by Neisser.

The solutions required are—

(1)	Methylene-blue	1 grm.
	Alcohol (96 per cent.) . . .	20 c.c.
	When dissolved add—	
	Aqua distill.	950 „
	Glacial acetic acid	50 „
(2)	Vesuvin	2 grms.
	Aqua distill. (boiled) . . .	1000 c.c.

The film is stained in solution No. 1 for three seconds,

washed in water, then stained for five seconds with No. 2 solution, washed, dried, and mounted. The body of the bacillus is stained brown, containing blue granules—as a rule two—one at each end, or only one at one end, seldom any in the middle. The granules are oval and never found free. In other organisms resembling the diphtheria bacillus the granules are rounded, and always situated at the end close to one another. A bacillus has recently been described, the *Hoffman* bacillus, which may appear on a blood serum tube within twelve to twenty-four hours of inoculation from a suspected case of pharyngitis. Its relation to diphtheria is not so far definitely determined. It is a short bacillus, which occurs in diplococcic form, stains readily with all the basic aniline dyes, retains the stain in Gram's method, but does *not* stain by Neisser's method.

Pus.—The chief organisms of pus are the cocci of suppuration, the streptococci and staphylococci. In addition to those, many organisms have pyogenic properties. The pneumococcus, tubercle bacillus, *B. coli*, and the bacillus of plague have been described.

The **Micrococcus tetragenus** may be recognised by its occurrence in groups of four, often provided with a capsule. It is chiefly found as a saprophyte in the pus of tuberculous vomicæ. The **gonococcus** is the specific organism of gonorrhœa. It occurs usually in diplococcus form, the adjacent margins of the two cocci being flattened or even slightly concave. It is contained almost exclusively in the pus cells, except in the early stages of gonorrhœa, when the discharge is glairy, when a number may be found free or adhering to the surface of desquamated epithelial cells. It is easily stained with a watery solution of any of the aniline dyes. It decolorises on treatment by Gram's method, and thus differs from most pyogenic cocci.

The **B. pyocyaneus** is sometimes found in purulent formations. The **tetanus bacillus** is found in the discharge from wounds on which tetanus has supervened. It

is easily recognised in film preparations, if it be present in sporulating form, by its characteristic drumstick-shaped appearance.

Actinomycotic pus contains colonies which can be recognised by the naked eye as small bodies varying in size up to a pin's head, the younger transparent or jelly-like, the older opaque and of various colours, white, yellow, greenish, or almost black. The best way to observe them is to spread out the pus upon a slide and hold the preparation up to the light. If one of the colonies is selected and washed with saline solution and examined unstained, the clubs, if present, are at once recognised under the low power. To study the filaments the colony should be broken down on a cover-glass, dried, and stained with a simple solution of any of the basic aniline dyes, or with dilute carbol-fuchsin. The fungus stains with Gram's method.

APPENDIX

CASE-TAKING

The following scheme of systematic case-taking is that which is in use in the Wards of the Clinical Professors in the Edinburgh Royal Infirmary.

NAME—AGE—OCCUPATION—PLACE OF BIRTH—MARRIED OR SINGLE—PLACE OF RESIDENCE—DATE OF ADMISSION—DATE OF EXAMINATION—COMPLAINT—DURATION OF ILLNESS [*Insert Thermometer*].

History.

Hereditary Tendencies—Habits as to Food and Drink—General Surroundings at Home and at Work—Previous Illnesses and Accidents—Time and Mode of Origin, and Course of Present Illness.

State on Admission.

General Facts.—Condition as to Height and Weight—Development—Muscularity—Obvious Morbid Appearances, as Jaundice, Dropsy, Cyanosis—Evidences of Injury or Previous Disease—General Appearance and Expression of Face—Temperament (if well marked)—Attitude (if unusual)—Temperature.

Alimentary System.—Lips—Teeth—Gums—Tongue—Secretions of Mouth—Fauces—Deglutition—Appetite—Thirst—Sensations during Fasting—Sensations during and after Eating (*Comfort or discomfort—Pain—Weight—Distension—Heartburn—Nausea*)—Acidity—Flatulence—Eructation—Waterbrash—Vomiting (*Characters, Macroscopic and Microscopic, and Chemical Examination of Vomited Matters when necessary*)—State of Bowels and Character of Fæces. Abdomen—

Inspection (*Prominence—Retraction—Distension—Flaccidity*) —Palpation (*—of Parietes—of Contents normal or abnormal—Tenderness — Fulness — Fluctuation*) — Percussion (*Vertical Dulness of Liver in Mammillary line, and, if necessary, outline of Liver, Stomach, etc.*)

Hæmopoietic System.—Lymphatic Vessels and Glands — Ductless Glands (*Spleen — Thyroid*) — Microscopic Characters of Blood (*Corpuscle Counting, and Determination of Hæmoglobin, and Specific Gravity of Blood, if necessary*).

Circulatory System.—Subjective Phenomena (*Pain—Palpitation —Faintness—Dyspnœa*)—Pulse (*Frequency—Rhythm—Character*)—Arteries, Capillaries, and Veins (*Cardiographic and Sphygmographic Tracings if necessary*)—Inspection (*Form and Appearance of Præcordia*)—Palpation (*Position and Character of Cardiac Impulse*)—Percussion (*Superficial and Deep, Outline if necessary*) — Auscultation (*Rhythm and Quality of Sounds in Mitral, Tricuspid, Aortic, and Pulmonary Areas, over General Surface of Heart and Main Vessels*).

Respiratory System.—Breathing (*Frequency—Rhythm—Type—Painfulness*)—Cough—Sputa (*Macroscopic and Microscopic Characters*) — Nares — Pharynx — Larynx (*Voice — Pain — Tenderness—Laryngoscopic Examination if necessary*) — Inspection (*Form and Action of Thorax, Measurement if necessary*)—Palpation (*Vocal Fremitus*)—Percussion (*Anterior and Posterior at corresponding points on the two Sides of Chest*)—Auscultation (*Determination at each point, during Natural and Deep Respiration, of the Duration of Respiratory Sounds, their Type or Character, Accompaniments or Superadded Sounds, and of the Vocal Resonance*).

Integumentary System.—Subjective Phenomena (*Pain—Itching*) —Skin (*Dryness—Moisture*)—Obesity—Emaciation—Œdema —Emphysema—Eruptions (*Distribution—Elements of Skin involved—Type—Cause*).

Urinary System.—Subjective Phenomena (*Pain, or Uneasiness in Loins, Bladder, or Urethra*) — Micturition (*frequency*). **Urine.** — Quantity — Colour — Specific Gravity — Chemical Reaction—Normal Constituents (*Amount of Urea, Chlorides, and Phosphates where necessary*) — Abnormal Constituents (*Albumin, Blood or Blood Pigment—Sugar—Bile*)—Deposits (*Macroscopic and Microscopic Characters*).

Reproductive System.—**Male**—Subjective Phenomena—Functions—Testicle—Epididymis—Prostate—Urethra. **Female**—Subjective Phenomena—Catamenia—Abnormal Discharges—Vagina—Uterus (*Examination, Digital, with Speculum and Sound if necessary*)—Ovaries.

Nervous System.

Sensory Functions.—Sensations (*Pain—Heat—Cold—Formication—Numbness—Tingling*)—Sensibility to Touch—Heat—Tickling—Pain. Muscular Sense. Sight (*Ophthalmoscopic Examination if necessary*). Condition of Pupil. Hearing (*Otoscopic Examination if necessary*). Taste. Smell.

Motor Functions.—Organic Reflexes (*Swallowing—Breathing—Micturition—Defæcation, etc.*)—Skin Reflexes—Tendon Reflexes—Voluntary (*Systematic Examinations of Groups of Muscles if necessary*). Co-ordination—Electric Irritability (*Faradic, Galvanic*).

Vasomotor and Nutritive Functions.—(*Local Congestions—Pallor—Œdema—Inflammation—Sloughing—Wasting—Perspiration, etc.*)

Cerebral and Mental Functions.—Intelligence (*Hallucinations—Illusions—Delusions—Torpor—Coma*)—Attention—Memory—Speech (*Comprehension of Language, heard, seen; Utterance of Language, spoken, written*)—Sleep.

Cranium (*Peculiarities*)—**Spine** (*Form and Appearance—Percussion—Hot Sponge Test*).

Locomotory System.—Bones—Joints (*Pain—Swelling—Effusion—Mobility*)—Muscles (*Rigidity—Flaccidity—Cramp—Twitching, general or fibrillary—Hypertrophy—Atrophy*).

Provisional Diagnosis.

Treatment.

Medicinal—Dietetic—General Directions.

Further Reports.

In Acute Cases report Daily, or more frequently if necessary.

In Chronic Cases report Once or Twice a week, always noting any Change of Diagnosis or Treatment.

At Conclusion of Case, note the result.

INDEX

	PAGE
ABDOMEN	189
,, Inspection of	189
,, Palpation of	191
,, Percussion of	192
,, Regions of	189
Abdominal Aneurysm	215
,, Lymphatic Glands	204
,, Reflex	332
,, Walls, Movements of	191
Acarus folliculorum	26
,, *scabiei*	26
Accompaniments	165
Acetic Acid	224
Aceto-Acetic Acid	293
Acetone	291
Achorion schönleinii	29
Acid, Acetic	224
,, Butyric	224
,, Lactic	224
Acids, Free	223
,, Organic	224
Actinomyces	185, 432
Actinomycotic Pus	432
Addison's Disease	22
Ægophony	168
Agglutination, Reaction of Blood	422
Albumin in Urine	270
Albuminometry	272
Albuminuric Retinitis	376
Albumoses	275
Alkaloids	301
Alkaptonuria	264
Alvine Discharges	227
Ametropia	382
Ammonia	259
Ammonio-Magnesium Phosphate	295
Amnesia	357
Amœba coli	229
Amphoric Breathing	163
,, Percussion Sound	149
,, Resonance	169
Anæsthesia	307
Analgesia	310
Anasarca	23
Aneurysm, Abdominal Aorta	215
,, Arch of Aorta, Signs of	82
,, Descending Thoracic Aorta	84
,, Innominate Artery	84
,, Pressure Signs of	82
Animal Parasites of Skin	26
Ankle Clonus	333
,, Jerk	333
Ankylostomum duodenale	236
Anus	204
Anvil Sound	179
Aorta, Aneurysm of	82
,, Arch of	33, 81
,, Descending Thoracic	84
,, Examination of	81
,, Relations of Arch	33
Aortic Area	45
,, Incompetence	58
,, Murmurs	56

	PAGE
Aortic Obstruction	57
,, Orifice	33
Apex Beat	35
Aphasia, Auditory	357
,, Conduction	358
,, Graphic Motor	360
,, Motor	357
,, Visual	358
Aphemia	357
Areas, Thoracic	126
Argyria	22
Arterial Wall, Condition of the	91
Arteries	92
,, Carotid	33, 84
,, Condition of Arterial Walls	91
,, Examination of	81
,, Radial	85
Ascaris lumbricoides	234
,, *mystax*	235
,, *vermicularis*	235
Ascites	193
Ascitic Fluid	196
Astigmatism	383
Athetosis	321
Auricular Appendices	33
Auriculo-Systolic Murmur	59
Auscultation	2, 10
,, Definition	2
,, Immediate and Mediate	10
,, *Vide* Heart and Lungs	43, 152

Babinski's Phenomenon	334
Bacillus coli communis	427
,, *diphtheriæ*	185, 430
,, Hoffman's	430
,, *influenzæ*	426
,, *pestis*	426
,, *pyocyaneus*	431
,, *tuberculosis*	185, 423, 427
Bacteriology, Clinical	417
Bacteriuria	426
Base of Heart	31

	PAGE
Bell Sound	151
Benzo-purpurin	223
Biceps Jerk	331
Bile Acids	284
,, in Urine	283
,, Pigments	283
Bilharzia hæmatobium	234, 300
Biuret Reaction	271
Bladder	215
Blood, Agglutination-reaction of	422
,, Alkalinity of	121
,, Coagulation Time of	121
,, Enumeration of Corpuscles	104
,, Estimation of Colouring Matter of	108
,, Examination of	103, 417
,, ,, ,, Fresh Film	112
,, Films, Examination of	118
,, ,, Fixation by Heat	115
,, ,, ,, ,, Reagents	115
,, ,, Staining	116
,, in the Urine	278
,, Microparasites in	122, 417
,, Microscopic Examination of	112
,, *Plasmodium malariæ*	122
,, Plates	120
,, Specific Gravity of	121
,, Spectroscopic Examination of	119, 125, 269
Blood vessels, Examination of	81
Borborygmi	202
Bothriocephalus latus	233
Bromine	300
Bronchial Breathing	154, 160
Bronchitis	169
Bronchophony	167
Broncho-vesicular Breathing	156, 163
Butyric Acid	224

Cæcum	203
,, Gurgling in	204

	PAGE
Calcium Carbonate	295
,, Oxalate	251, 295
,, Phosphate	295
Campimetry	391
Capillaries	99
Capillary Pulse	100
Carboluria	264
Cardiac Conditions, Points in the Diagnosis of	73
,, Dulness	40
,, Murmurs, *Vide* Heart	51
Cardiography	39
Carotid, Arteries	84
,, Common	33
Case-taking, Methods of	433
Cavernous Breathing	162
Central Artery, Embolism of	376
,, Choroiditis	378
Cerebro-spinal Effusions	429
Cestoda	231
Charcot-Leyden Crystals	184
Chest—*Vide* Thorax	131
Cheyne-Stokes Respiration	137
Chloasma	22
Chlorides, Quantitative Estimation of	265
,, Quantity of	266
Cholesterin	297
Choluria	283
Choroid	379
,, Tubercles in	378
Choroiditis	377
,, Acute	377
,, Central	378
,, Disseminated	378
Chromogens	244
Chrysophanic Acid	301
Chvostek's Sign	330
Chyluria	246
Circulatory System	31
Clavicles, Flattening under	133
Clonus, Ankle	333
,, Knee	333
Coagulometer, Wright's	121
Cocci, Pyogenic	427
Co-efficient of Haeser	250
Co-efficient of Trapp	250
Colon, Position of	202
,, Stricture of	203
Colour Sense, Examination of the	401
Columnar Epithelial Cells	298
Coma	354
,, Vigil	354
Compensation, Failure of	77
Congo Red	223
Consciousness	354
Continued Fever	14
Continuous Current	322
Contracture	318
Co-ordinating Functions	338
Copaiba	301
Corpuscles, Red Blood	104, 114
,, White	106
Counting Fingers	388
Cracked-pot Percussion Sound	150
Cranial Nerves	342
,, Reflexes	335
Creatinin	263
Cremasteric Reflex	332
Crepitations	165
Crisis	15
Culture Methods	421
Cyanosis	20
Cylindrical Lenses	368
Cyrtometer	131
Cystin	296
Decubitus	4
Defæcation	340
Degeneration, Reaction of	328
Delirium	354
Delusions	338, 355
Deviation	344
,, Conjugate	346
Diacetic Acid	293
Diagnosis	1
Diaphragm, Respiratory Action of	138
Diarrhœa	227
Diastolic Murmur	65, 68
Digestion, Activity of	226

	PAGE
Dioptre	367
Direct Vision	379
Diphtheria Bacillus	430
Diplopia	345
Displacements of Organs	179, 200
Distomum hepaticum	233
,, *lanceolatum*	234
,, *pulmonale*	185
Dyspnœa	136
,, due to Laryngeal Disease	413
Ear, Examination of	406
Echinococcus	185, 300
Effusion, Cerebro-spinal	429
,, Joint	429
,, Pericardial	428
,, Peritoneal	429
,, Pleural	428
Electrical Irritability	322
Embolism of the Central Artery	376
Emmetropia	382
Emphysema	169
,, Subcutaneous	23
Endocardial Murmurs	53
,, ,, Propagation of	61
Epigastric Pulsation	37, 190
Epithelial Cells	298
Erythrasma	30
Esbach's Process	272
,, Solution	273
Eustachian Catheter	360, 408
,, Tube	407
Exocardial Murmurs	51
Extracardiac Regions	37
Exudations, Inflammatory	428
,, Membranous	430
Eye, Examination of	362
,, Inspection	364
,, Palpation	362
,, Refractive Media of	368
,, Subjective Examination	379
,, Tension of the	363
,, Transparency of the	364
Facial Nerve	348
,, Paralysis	349
Faeces	227
Faradic Irritability, Changes in	327
Fat in Urine	297
Favus	29
Febriculæ	13
Fehling's Test	286
Fermentation Test	288
Fibrillary Contraction	321
Field of Vision	390
Filaria diurna	122
,, *nocturna*	122
,, *perstans*	122
,, *sanguinis hominis*	122, 300
Flash Test	369
Form, Direct Visual Acuity	380
Fremitus, Bronchial	141
,, Pleural	140
,, Vocal	138
,, ,, Definition	5
Friction	167
,, Fremitus	140
,, Pericardial	51
,, Pleural	167
,, Pleuro-Pericardial	52
Fundus Oculi, Examination of	371
Gall Bladder	209
,, Anatomical Relations of	209
,, Distension of	209
,, Inspection of	209
,, Palpation of	209
Galton's Whistle	405
Galvanic Irritability	326
Gastric Contents	217
,, Digestion	226
Glaucoma	362
Glosso-Pharyngeal Nerve	350
Glucose	285
,, in Urine	285
,, Quantitative Estimation of	290

	PAGE
Gmelin's Test	283
Gonococcus	429, 431
Gram's Method	420
Gratiolet, Radiation of	358
Gulland's Method	115, 420
Gums	187
Gunzburg's Reagent	223
HÆMACYTOMETER	104
Hæmatemesis	270
Hæmatoporphyrin	244, 283
Hæmaturia	278
Hæmic Murmurs	78
Hæmin Test	281
Hæmoglobin	108
Hæmoglobinometer	108
,, Gowers'	108
,, Haldane's	109
,, Oliver's	110
Hæmoglobinuria	283
Hallucinations	355
Hand Reflex	389
Hayem's Fluid	105
Head, Altered Carriage of	345
Heart, Accentuation of Second Sound	50
,, Alterations in Rhythm	48
,, Anatomical Relations	31
,, Areas	45
,, Auscultation	43
,, Deep Dulness	40
,, Intensity of Sound	47, 49
,, Irregularity	49
,, ,, Intermittence of First Sound	49
,, Modifications of First Sound	45
,, ,, Second Sound	49
,, Palpation of	38
,, Percussion of	39
,, Reduplication of Second Sound	51
,, Superficial Dulness	40
Heller's Nitric Acid Test	271
,, Test	281
Hemianopsia	397
Hemiplegia	320
Hippuric Acid	263, 296
Histon	276
History, Definition of	1
Hoffman Bacillus	431
Hydrochloric Acid	223, 265
,, Estimation of	265
Hydro-pneumothorax	180
Hyperæsthesia	307
Hyperalgesia	310
Hypermetropia	385
Hyperpyrexia	16
Hypoglossal Nerve	351
ICTERIC URINES	283
Icterus	21
Illusions	338
Images, Method of Double	345
Indeterminate Breathing	152, 161
Indican	245
Indoxyl	244
,, Potassium Sulphate	264
Induced Current	326
Influenza Bacillus	426
Infusoria	231
Innominate Artery	84
,, Vein	33
Inosite	264
Insanity	355
Inspection, Definition of	2
,, General	4
,, Local	5
,, Negative Evidence from	37
Integumentary System	18
Intestine, Large—*Vide* Colon	202
,, Small	202
Irregularity of Heart	49
JAUNDICE	21
Jaw Jerk	330
Jenne's Test	406
Johnson's Test	272
Joint Effusions	429

	PAGE
KIDNEY, Anatomical Relations of	213
,, Floating	214
,, Palpation and Percussion of	213, 214
Kjeldahl's Process	252
Knee Clonus	338
,, Jerk	333
LACTIC ACID	224
Lactose	291
Ladder Pattern	191, 202
Laryngeal Cough	413
,, Disease, Dyspnœa due to	413
Larynx, Examination of	409
Law of Normal Galvanic Irritability	326
Lenses, Cylindrical	368
,, Spherical	368
Leptus autumnalis	26
Leucin	296
Leucocytes, Basophil	119
,, Enumeration of	106
,, in urine	297
,, Oxyphil	119
,, Polymorphonuclear	119
Leucocytosis	113
Levulose	291
Light, Perception of	389
Liver, Anatomical Relations	205
,, Diminution of	209
,, Enlargement of	209
,, Inspection of	205
,, Palpation of	206
,, Percussion of	206
Locality, Sense of	305
Lungs, Abnormal Percussion Sounds	146
,, Anatomical Relations	127
,, Auscultation of	152
,, ,, in Abnormal States	157
,, Character of Percussion Sound	141
Lungs, Diminished Expansion	134
,, Expansion	130
,, General Increased Expansion	135
,, Inspection and Mensuration	130
,, Lobes of	129
,, Method of Percussion	143
,, Percussion of	141
,, Physical Examination of	129
Lymphocytes	119
Lysis	14
MACULA	374
Magnesium Phosphate	295
Malaria, Plasmodium of	122
Mammary Line	126
Meatus	406
Mechanical Irritability	321
Megaloblast	118
Megalocytes	113
Melanin	246
Mental Functions	353
Metallic Tinkling	178
Methods and Terms	4
Microblast	118
Micrococcus tetragenus	431
,, *ureæ*	427
Microcytes	113
Micro-Organisms in Sputa	181
Microsporon audouini	27
,, *minutissimus*	30
,, *furfur*	30
Micturition	340
Milk-Curdling Ferment	226
Mitral Area	45
,, Murmurs, Differential Diagnosis	71
,, Orifice	33
,, ,, Murmurs at,	58
,, Regurgitation	64
,, Stenosis	58, 65
Monoplegia	320
Moore's Test	285
Motor Aphasia	357

	PAGE
Motor Areas	315
,, Functions	315
,, ,, Voluntary	317
,, Points	324
Mouth	186
Mucin	275
Mucus	220
,, Cells	298
Muir's Method	419
Murmurs, Anæmic	78
,, Aortic	57, 62
,, Arterial	79
,, Cardiac	51
,, Differentiation of Multiple	71
,, Direction of Propagation	61
,, Endocardial	53
,, Exocardial	57
,, Extent of Area of audition of	61
,, in Anæmia and Debility	78
,, Intensity of Loudness	70
,, Mitral	58, 64, 81
,, Obstructive Aortic	56
,, Point of Maximum Intensity	61
,, Position of, in Cardiac Cycle	56
,, Pulmonary	60, 66, 80
,, Summary of	68
,, Systolic and Diastolic	69
,, Time of	54
,, Tone, Quality, or Character	69
,, Tricuspid	60, 67, 80
,, Venous	78
Muscles, Flaccidity	317
,, Mechanical Irritability of	321
,, Movements of	319
,, Rapid Wasting of	318
,, Rigidity	323
Muscles, Size	319
,, Tone	317
Muscular Sense	314
Mydriasis	338
Myopia	383
Myosis	338
Myxœdema	24
NASO-PHARYNX	413
,, ,, Examination of,	188, 413
Neisser's Stain	430
Nematoda	234
Nerve, Eleventh	350
,, Fourth	343
,, Fifth	347
,, Ninth	350
,, Seventh	348
,, Sixth	344
,, Third	342
,, Tenth	350
,, Twelfth	351
Nerves, Cranial	342
Nervous System	303
,, ,, Anatomy and Physiology of	303, 315
Neuro-Retinitis	375
Nitrogen of Urine	252
Noise, Definition of	7
Normoblast	118
Nose, Examination of	413
Nucleo-albumin	275, 278
Nummula	182
OBSTIPATION	227
Ocular Media, Refractive Power of	367
Œdema	23
Œgophony	168
Œsophagus	189
Oligocythæmia	106
Oliver's Peptone Test	284
Omentum, Great	210
Ophthalmoplegia	346
Ophthalmoscope	365
Optic Atrophy	376

	PAGE
Optic Disc	373
,, ,, Anæmia of	375
,, ,, Hyperæmia of	375
,, Nerve, Inflammation of	375
Organic Reflexes	340
Orientation, False	345
Orifices of Heart	32
Orthopnœa	137
Oxalate of Calcium	251, 295
Oxy-butyric Acid	293
Oxyuris vermicularis	236
PAIN, SENSE OF	308
Palpation	2, 5
,, *Vide* Thorax and Abdomen	138, 191
Pancreas, Anatomical Relations of	210
,, Lesions of	210
Papillitis	375
Paragraphia	357
Parakresol	264
Paralysis	320
Paraphasia	357
Paraplegia	320
Parasites, Intestinal	229
,, Skin	26
Parasternal Line	126
Pavy's Test	287
Pectoriloquy	168
Pediculus capitis	26
,, *corporis*	26
,, *pubis*	26
Peptone	226
,, in Urine	275
Perception of Light	389
Percussion, Definition of	5
,, Duration	8
,, Gentle and Strong	6
,, Immediate	6
,, Intensity	7
,, Mediate	6
,, of Organs—*Vide* under respective Organs.	
,, Pitch	9
Percussion, Quality	9
,, Resistance	9
Pericardial Fluid	428
,, Friction	61
Pericardium, Effusion into	43
Perimetry	309, 391
Peritoneal Effusion	429
Peritonitis	196
Pettenkofer's Test	284
Pharynx	188
Phenol	264, 301
Phenyl-hydrazin Test	287
Phosphates in Urine	251, 295
,, Quantitative Estimation of	268
Phosphenes	320, 403
Phosphoric Acid	267
Phthisis	175
Physical Examination, Definition of (*vide* respective Organs)	1
Pigeon-breast	131
Pityriasis versicolor	30
Plague Bacillus	426
Plantar Reflex	333
Pleural Effusions	428
Pleurisy	172
Pleuro-Pericardial Friction	52
Pneumobacillus	425
Pneumococcus	185, 425
Pneumonia	171
Pneumothorax	176
Poikilocytosis	113
Polarimetry	289
Politzer's Method	407
Polychromatophil Corpuscles	118
Post-diastolic Murmurs	59
Posterior Rhinoscopy	414
Posture, Perception of	314
Potash, Chlorate of	301
Præcordia, Bulging	34
,, Inspection	34
,, Palpation	38
,, Pulsation	34
,, Retraction of	35
Presbyopia	384
Presystolic Murmur	59

	PAGE
Proteids, Clinical Significance of	277
,, in Urine	270
,, Separation of	276
Proteoses	274
Protozoa	229
Ptosis	343
Puerile Breathing	158
Pulmonary Area	45
,, Artery	34
,, ,, Murmurs in	80
,, Murmurs	60, 67, 80
,, Orifice	33
Pulsation, Character of	37, 92, 94
,, ,, in Different Arteries	99
,, Duration of	96
,, Epigastric	37
,, Extent of Præcordial	37
,, Extra-Apical	36
,, in Second Space	36
,, of Right Ventricle	36
Pulse, Capillary	100
,, Dicrotic	97
,, Force	95
,, Fully Dicrotic	92
,, Hyperdicrotic	98
,, in different Arteries	99
,, Intermission of	94
,, Irregularity of	93
,, Low and High Tension	99
,, Method of Observing	85
,, Normal Characters	89
,, Phenomena to be Studied	89
,, Radial	85
,, Rate of	92
,, Rhythm of	93
,, Secondary Waves	97
,, Size of	94
,, Sphygmographic Tracings	88
,, Sustained Pressure	98
,, Venous	101
Pupil Reflexes	335
Pupilloscopy	366
Purin Bases	263
Pus	251, 431
Pyro-catechin	264
REACTION OF DEGENERATION	328
Rectum	204
Reduplication of Heart Sounds	48, 51
Reflex Action, Decrease of	334
,, ,, Excess of	335
,, Functions	329
Refraction	382, 384
Resistance, Sense of	9, 151
Resonance, Vocal	10, 167
Resorcin	224
Respiration, Alterations in Rhythm and Rate of	136
,, Cheyne-Stokes	137
,, Frequency of	131, 136
,, Types of	130
Respiratory Movements, Frequency of	131
,, System	126
Retina, Detachment of the	377
,, Hyperæmia of	375
Retinal Vessels	374
Retinitis	376, 377
,, Albuminuric	376
,, Hæmorrhagic	355
,, Pigmentosa	377
Retinoscopy	369
Rhinoscopy	413
Rhonchi	164
Robertson (Argyll), Phenomenon	337
Romanowsky Stain	124
Round Epithelial Cells	298
Round-worm	234
SACCHAROMETER	289
Salicylates	300
Santonin	301
Sarcina pulmonis	185
Sarcinæ ventriculi	226, 428
Scabies	26

	PAGE
Scapular Line	126
Scapulo-humeral Reflex	331
Scotoma	396
Sense of Hearing	404
,, Locality	306
,, Pain	308
,, Sight	361
,, ,, Subjective Examination of	379
,, Smell	263
,, Taste	403
,, Temperature	307
Sensibility, Ordinary	304
,, Perverted	311
Sensory Disturbances	315
,, Functions	303
Serum Albumin	270, 274
,, Globulin	270, 274
Sexual Functions	342
Shadow Test	369
Sherrington's Fluid	106
Sigmoid Flexure	204
Skatoxyl Potassium Sulphate	264
Skin, Animal Parasites of	26
,, Blueness of	20
,, Bronzedness of	22
,, Colour of	19
,, Examination of	18
,, General Conditions	18
,, Greyness of	22
,, Moisture of	22
,, Pallor of	19
,, Redness of	19
,, Special Affections	25
,, Vegetable Parasites of	27
,, Yellowness of	21
Sleep	353
Snellen's Test Type	380
Sound	6
,, Abnormally Clear	148
,, Absolute Dulness	148
,, Definition of	6
,, Dull	147
,, Empty, Shallow, Full	144
,, Increase in Clearness	148
,, Intensity of	7
Sound, Musical and Non-Musical	7
,, Pitch of	9
,, Quality of	9
,, Slight or Comparative Dulness	147
Spasms	321
Special Sensibility	295
Spectrum	280
Speech	355
,, Centres	356
,, Lalling	360
,, Slurring	360
,, Staccato	360
Spherical Lenses	368
Sphygmograph	87
Spinal Accessory Nerve	351
Spirals	184
Spleen, Anatomical Relations of	211
,, Enlargement of	213
,, Inspection, Palpation and Percussion of	211, 212
Sputum	181, 423
,, Crystals in	184
,, Fibrinous Casts in	184
,, Microscopic Examination of	183, 423
,, Parasites of	184
Squamous Epithelial Cells	298
Stains	116, 124
Staphyloma	378
Starch	226
Stenosis of Cardiac Orifices—*Vide* under respective Orifices.	
,, Relative	57
Stethoscope, Mode of Using	10
Stomach	196
,, Anatomical Relations of	196
,, Auscultatory Percussion of	201
,, Contents, Examination of	217, 221
,, Inspection of	198

PAGE

Stomach, Palpation of . . 200
,, Percussion of . . 198
,, Splashing in . . 201
Stupor 354
Subcutaneous Emphysema . 23
,, Tissues . . 23
Succussion 11
,, Hippocratic . 11, 180
Sugar 226
,, in Urine, Clinical Significance of . . . 291
Sulphates, Detection of . . 269
,, Quantity of . . 270
Sulphuric Acid . . . 269
Superior Vena Cava . . 33
Supinator Jerk . . . 331
Suprarenal Capsules . . 215
Symptoms, Definition of . 1

Tactile, Sense . . . 305
Tænia mediocanellata . . 231
,, *solium* . . . 232
Tannin 301
Tape-worms 231
Taste, Sense of . . . 403
Teeth 186
Temperature, Elevated . . 12
,, Intermittent . 15
,, Regularly Remittent . 14
,, Relapsing . 16
,, Relation to Pulse 17
,, Sense of . . 307
,, Sustained 14, 15
,, Types of . . 14
Tenesmus 227
Tension of the Eye . . 363
Tetanus Bacillus . . . 431
Thorax 131
,, Alar 132
,, Circumference . . 133
,, Diameters of . . 133
,, Diminished Expansion 134
,, Distention of . . 133
,, Drawing in or Retraction of . . . 135

PAGE

Thorax, Emphysematous . 132
,, Flattening of . . 133
,, Form of . . . 131
,, Hyper-resonance of . 144
,, Movement of . . 134
,, Pain on Palpation of 139
,, Palpation of . . 138
,, Percussion of . . 141
,, Pigeon-Breast . . 131
,, Pitch of Percussion Sound . . 9, 143
,, Retraction of . . 135
,, Rickety . . . 131
,, Semi-Circumference . 133
,, Surface Lines and Areas 126
Thrills 39
Tinea tonsurans . . 27, 53
Tinkling, Metallic . . 178, 207
Tongue 186
Torulæ 428
Trachea, Bifurcation of . . 129
Tracheal Resonance . . 149
,, Tugging . . . 83
Transparency of the Eye 344, 364
Traube's Area . . . 199
Trematoda 233
Tremors 325
Triceps Jerk 331
Trichina spiralis . . . 237
Trichocephalus dispar . . 237
Trichophyton megalosporon . 27
Tricuspid Area . . . 45
,, Murmurs . 60, 67, 68
,, ,, Point of Maximum Intensity 67
,, Orifice . . . 33
Triple Phosphates . . . 295
Trismus 285
Trommer's Test . . . 353
Trophic Functions . . . 353
Tube Casts 298
Tubercle Bacillus . . . 423
Tubular Breathing . . . 161
Tympanic Membrane . . 407
Tympanitic Sound . . 146, 148
Tyrosin 296

	PAGE
Uffelmann's Test	224
Urate of Ammonia	294
,, Sodium	294
Urates	251, 294
Urea	254
Urethral Filaments	299
Uric Acid	251, 259, 294
,, Quantitative Estimation	261
Urinary Deposits, Microscopic	293
,, System	239
Urine, Accidental Constituents of	300
,, Acidity of the	247
,, Albumin in	275
,, Albumoses in	275, 278
,, Alkalinity of the	248
,, Ammonia of the	259
,, Ammoniacal	249
,, Bacteriology of	426
,, Chemical Characters of the	242, 251
,, Chromogens of	244
,, Colour of the	244
,, Inorganic Constituents	242, 265
,, Method of Examination	240
,, Milky Appearance of	246
,, Naked-eye Deposits in	250
,, Nitrogen of	252
,, Odour of the	247
,, Organic Constituents	242
,, Parasites in	300
,, Physical Characters of	241
,, Pigmentation due to Drugs	247
,, Proteids in	270
,, Quantity of the	243
,, Reaction of the	247
,, Specific Gravity of the	249
Urinometer	249
Urobilin	244
Urochrome	244
Uroerythrin	244
Urorosein	245
Vagus	350
Valsalva's Experiment	407
Valvular Lesions, Secondary Results of	75
Vasomotor Functions	352
Vegetable Parasites of the Skin	27
Veins	100
,, Movements of	100
,, Pulse in	101
Vena Cava	33
Venous Stigmata	19
Vermiform Appendix	203
Vertigo	345
Vesicular Breathing	155
,, ,, Varieties of	158
Vesiculo-Bronchial Breathing	163
Visceral Functions	339
Vision	379
,, Field of	390
Visual Acuity	381, 400
Vocal Fremitus—*Vide* Fremitus	138
,, ,, Absence of	140
,, Resonance	10, 167
Vomit, Characters of	219
Vomiting, Causes of	216
Weber's Test	405
Wernicke's Sign	377, 399
Widal's Reaction	422
Word-Blindness	358
,, Deafness	357
Xanthoproteic Reaction	272

THE END.

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