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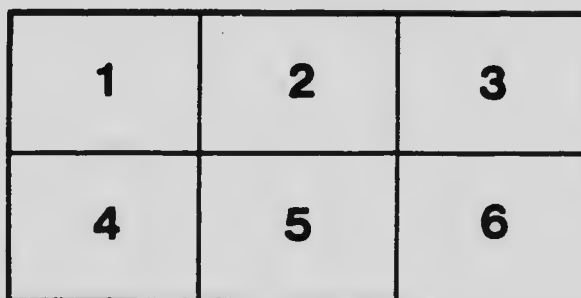
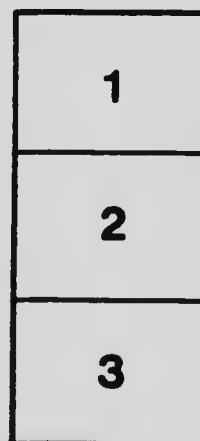
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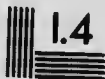
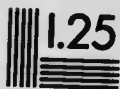
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A DICTIONARY OF MEDICAL DIAGNOSIS

A DICTIONARY  
OF  
MEDICAL DIAGNOSIS

A Treatise on the Signs and Symptoms observed  
in Diseased Conditions

FOR THE USE OF MEDICAL PRACTITIONERS  
AND STUDENTS

BY

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SECOND EDITION

TORONTO  
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## PREFACE TO THE SECOND EDITION

SINCE the first appearance of this book I have had the advantage of the friendly criticism and helpful advice of many friends. The errors of various descriptions which have thus been brought under my notice, as well as by reviewers in the Medical Journals, have been, as far as possible, corrected in this edition. May I point out to those who have done my book the honour to review it that it does not include in its scope the description of *diseases*, though in the course of a discussion on the occurrence, cause, and nature of symptoms, diseases must necessarily be often mentioned, and even occasionally very briefly described.

The articles by my colleagues have been revised. Thus Dr. Houston gives an account of the Wassermann reaction; Dr. Rankin has amplified his description of radiography of the abdomen; and Dr. MacIlwaine has introduced the antiformin method of examination for the tubercle bacillus.

A description of a number of diagnostic signs, omitted in the first edition, is now given—*e.g.*, Grocco's, Ewart's, Biernacki's, Joffroy's, Quinquaud's, Rotch's, Weber's, etc. Several diagnostic tests or reactions are added—*e.g.*, Salomon's, Cammidge's, Boiss's, von Pirquet's, etc. The index has been revised and some omissions made good.

I have again to thank those gentlemen named in the preface of the first edition, and also Dr. John Hay, for permission to use their illustrations.

H. L. MCKISACK.

BELFAST,

January, 1912.

## PREFACE TO THE FIRST EDITION

IN approaching a case of illness the observer is confronted with certain signs and symptoms, the significance of which he must be familiar with before he is able to form any intelligent conception of the morbid processes at work. It is, therefore, necessary to study carefully the language of signs as presented in the bodies of our patients.

Among the most fruitful methods of clinical teaching is that of considering fully the nature and origin of individual symptoms, observing in what respect the processes to which the symptom is attributed differ from those occurring in the healthy body. It is possible, by reviewing in this manner the subjective and objective symptoms, severally and collectively, presented by the patient, to indicate the nature and situation of the diseased condition; and it is precisely this comprehension of the nature of the process or condition, and not the mere labelling it with a name, which constitutes a rational diagnosis.

The study of individual signs and symptoms of disease deserves more attention than has been devoted to the subject by authors. For some years I have been in the habit of noting, for my own use and for teaching purposes, the significance of the various morbid phenomena, considered strictly from the observer's point of view. In the following pages I have embodied and extended these notes, and have endeavoured to lay before the practitioner and student a concise, but I hope a sufficiently explicit, description of the symptoms commonly encountered in medical affections. Where it seemed advisable I have referred to the physiology and pathology involved, indicating at the same time the affections in

which the symptom might be expected to occur. Beyond such passing references to the affections which give rise to the symptoms under consideration, and their mention in the comparative tables to be found in a number of the articles, I have avoided the discussion of *diseases*, and have restricted the descriptions to the various *signs and symptoms* of disease. The consideration of the disease is left to the text-books of medicine, for which the present work is not intended as a substitute, but as a complement.

In dealing with obscure or complicated cases the observer may at times experience some difficulty in determining which of the sections of his medical treatises he should consult in search of light. A careful study of one or more of the prominent symptoms presented by the case, with a knowledge of the morbid processes which produce the symptoms, and of the diseases in which they are likely to occur, should serve to guide the observer in the further study of the case.

In the present volume I have attempted to place within reach of the reader, in an easily available form, the information necessary to attain the objects just mentioned. In order to facilitate reference to the different subjects, these are dealt with in alphabetical order, and not in anatomical or nosological grouping.

The descriptions of the various methods of examination are restricted to such procedures as may be fairly considered clinical methods, and I have refrained from presenting numerous alternative processes where sufficiently accurate results might be secured by the methods described.

Three of the articles have been written by colleagues, whose special experience in the respective subjects lends value to their contributions. These are 'Blood Examination,' by Dr. Thomas Houston, hæmatologist to the Royal Victoria Hospital, Belfast; 'X-Ray Diagnosis,' by Dr. J. C. Rankin, physician in charge of the electrical department of that hospital; and 'Examination of the Sputum,' by Dr. J. E. MacIlwaine, medical registrar to the same hospital.

I have to express my indebtedness to Dr. T. K. Monro, for the use of a number of illustrations from his 'Manual of Medicine'; to Dr. James Mackenzie, for a similar kind permission to insert some figures from his 'Study of the Pulse'; and to Dr. L. Werner, for permission to reproduce two diagrams. The illustrations from these sources are indicated in the text.

To my colleagues on the staff of the Royal Victoria Hospital I desire to express my gratitude for kindly help and advice, and for the use of their clinical material for photographic purposes.

My thanks are also due to successive house physicians and resident pupils of the hospital for much help, and to Mr. J. Cole, who has produced the majority of the photographs, some of which I owe to the kindness of Mr. A. McMillan.

H. L. MCKISACK.

BELFAST,  
*April*, 1907.

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# A DICTIONARY OF MEDICAL DIAGNOSIS

## ABDOMEN: Abnormalities in its Shape, Size, Movements, and Surface Markings.

Topography of the abdomen—Bony landmarks—Natural surface markings—Artificial surface lines—Regions of the abdomen.

Method of examination—Palpation—Dipping—Resistance—Colour—Enlarged veins—Caput Medusæ—Umbilicus—Lineæ albicantes.

Shape of abdomen: flattened, rounded, boat-shaped—Swelling in the various abdominal regions.

Size of abdomen: diminished, increased—Obesity—(Edema—Ascites—Tympanites—Free gas in the peritoneal cavity—Enteroptosis.

Movements of respiration: increased, diminished—Movements of circulation—Epigastric pulsation—Irritable aorta—Aneurism—Pulsation of the liver—Peristalsis—Tumours movable with respiration—Fluctuation—Fœtal movements.

IN referring to the topography of the abdomen one makes use of certain natural landmarks, and of various artificial lines and areas, in order to localize an observation. The various natural fixed points are: (1) The **bony landmarks**—viz., the ensiform cartilage, the two outcurving free borders of the ribs (the costal margins), the crests and anterior superior spines of the ilia and the symphysis pubis. These, with the bodies of the lumbar vertebræ, perhaps the lower end of a kidney, the lower edge of the liver, and at times indistinct fœcal masses, are the only solid bodies to be seen or felt in a healthy abdomen. (2) Certain **natural surface markings** are also useful in fixing the locality of any observed condition: the umbilicus; the linea alba, seen as a mesial groove extending from the ensiform cartilage to the umbilicus: the lineæ semilunares, forming the outer border of the recti muscles on each side; three lineæ transversæ separating the muscular segments of the recti, the uppermost being immediately below the ensiform

cartilage, the next midway between that point and the umbilicus, and the lowest at the level of the umbilicus, all three being horizontal.

Four artificial lines, two vertical and two horizontal, divide the abdomen into nine regions (see Fig. 1). The vertical lines are drawn from the mid-point of Poupart's ligament; the upper of the two horizontal lines extends between the lowest part of one tenth rib and its fellow on the opposite side; the lower joins the two anterior superior spines of the ilia. The regions so formed are: in the middle from above downward, epigastric, umbilical, and hypogastric; on each side a hypochondriac, a lumbar, and an iliac. The hypochondriac regions are bounded above by the lower border of the 'pulmonary region' (see Thorax, p. 451); internally by the costal margin of its own side, as far as the point where the vertical line meets the ribs, thence by that line as far as the upper horizontal line.

By referring to one or other of these regions, or by stating the distance in inches from any of the fixed points at which the object is observed, a sufficiently accurate record can be made.

Abnormalities in the aspect, shape, size and movements of the abdomen are detected on examination, by inspection, palpation, and mensuration. The patient is best examined as a rule in the recumbent position, face upward, the abdominal walls being relaxed as much as possible, with which object the knees may be drawn well up. Inspection in a good light from the front, sides, and (the legs being extended) from the foot of the bed, is of the utmost importance. Palpation is best carried out with the whole palmar surface of the hand, its ulnar edge being toward the pubes; that is, if standing on the patient's right side, the observer uses his right hand, and *vice versa*. The abdominal muscles by their voluntary or involuntary contraction form a great obstacle to an efficient examination. The patient must be encouraged to maintain the body in as relaxed a condition as possible; the head and shoulders may have to be slightly elevated by a single pillow, the mouth being kept open, and the patient breathing easily. A cold hand or sudden pressure of the finger-tips will cause a defensive contraction of the recti and other muscles; it is therefore advisable to commence the palpation with a warm hand laid gently and flat upon the surface. Pressure may be increased when the abdomen has grown accustomed to the touch. By increasing the force of pressure at the end of each expiration, when the muscles involun-

tarily yield, the deepest portions of the belly may be searched. After the muscles have become familiarized with the pressure of the flat hand, the palmar surfaces of the fingers and the finger-tips



FIG. 1.—TOPOGRAPHY OF THE THORAX AND ABDOMEN.

A, mid-sternal line; B, side-sternal line; C, parasternal line; D, nipple line; E, anterior axillary line; F, right hypochondriac region; G, epigastric region; H, right lumbar region; I, umbilical region; J, right iliac region; K, hypogastric region; T, Traube's semilunar space; *ii, iii, iv, v*, on the left ribs of the same numbers.

The shaded areas are the absolutely dull regions of the liver, heart, and spleen.

may be used to investigate deep-seated regions. In cases of peritoneal effusion (ascites) tumours or enlarged organs may escape

discovery if palpated in the ordinary manner : we then employ the method known as **dipping**. By a sudden, unexpected 'dive' of the finger-tips the muscles are taken unawares, and, the fluid being driven to one side, the tumour seems to bump against the examiner's fingers. One must guard against mistaking a contracted muscle for an intra-abdominal tumour ; the sections of the recti muscles are especially liable to deceive one, and the deeper masses of muscles may in some cases mislead. If the recti are persistently contracted, it is sometimes possible to insert the fingers under the edge of the muscle, and so palpate the deeper parts. Rigidity of the abdominal muscles as a whole is found in many healthy persons whose reflex contractions are too readily stimulated by the contact of the examining hand ; such individuals are quite incapable of producing the desired relaxation. Strongly developed abdominal muscles are often a serious hindrance from the same inability to relax ; but it is in painful inflammatory conditions of the peritoneal cavity that the chief difficulty from rigidity occurs. Here the muscles are automatically on guard to protect the damaged organs, and no desire on the part of the patient to assist the examiner will avail to induce the muscles to relax. This rigidity is fortunately in some cases a help instead of a hindrance in diagnosis ; where a localized peritonitis or other painful condition is found, a localized rigidity or 'resistance' of the overlying muscles commonly appears, and this is at times a valuable, and perhaps the only, guide to the nature of the affection. In some cases of rigidity of the muscles it may be worth while to examine the patient under an anæsthetic ; this may also be necessary in cases of abdominal distension in hysterical cases, where an apparent tumour (**pseudocystis**), occupying the greater part of the abdomen, and consisting of flatulent distension combined with lordosis, disappears as soon as the patient is anæsthetized.

In addition to the palpating hand in front of the abdomen, assistance may be obtained from counterpressure with the other hand placed behind on the lumbar region, just below the last rib ; this procedure is especially useful in examining the hypochondriac regions. At times the prone or knee-elbow position may help. By this means, for example, it may be more surely ascertained if a pulsating abdominal tumour is an aneurism or is a solid tumour transmitting pulsations from the aorta.

Percussion is of considerable value in investigating abdominal

disease, and is discussed at p. 15. Auscultation is here of much less importance, and is referred to at p. 19.

1. **Aspect and Surface Markings.**—(a) **Colour:** Pigmentation is seen in pregnancy, chiefly located along the linea alba. The yellow colour of jaundice, that of pernicious anæmia, cachexiæ of different descriptions, and the various colour abnormalities mentioned on p. 284, may be observed in this region. (b) **Enlarged Veins:** A considerable enlargement of superficial veins in the neighbourhood of the umbilicus (the blood current directed away from that point) indicates an obstruction to the blood-return in the portal circulation, the most probable causes being cirrhosis of the liver or thrombosis of the portal vein near its entrance into the liver. A radiating arrangement of the veins, with the umbilicus as a centre, is occasionally seen under the above circumstances, and is known as the *caput Medusæ*. When the veins are more definitely situated at the sides of the abdomen, and when the blood current in them is upward, the cause is obstruction of the inferior vena cava. A slight dilatation of these superficial veins might be the result of moderate compression of the vena cava (e.g., ascites), while a more marked enlargement of the veins would indicate more serious obstruction, as thrombosis of the vena cava or the pressure of tumours. (c) **Umbilicus:** A retracted umbilicus is seen in fat abdomens and in œdema of the abdominal wall. It is flattened and stretched in ascites, but projects in pregnancy and hernia. (d) **Lineæ albicantes** are seen as whitish streaks (reddish when recent). They indicate a considerable and prolonged stretching of the skin, as occurs in pregnancy, tumours, ascites, and excessive fat. In addition to their invariable occurrence in multiparæ, they are seen on the abdomen, thighs, and axillary folds of stout persons. (e) **Eruptions** of various exanthemata and other skin affections may be found on the abdomen, but, with the exception of typhoid spots, are not specially likely to occur here (see p. 366).

2. The **Shape** of the abdomen often conveys information as to the nature of morbid conditions. A **flattened shape**, bulging at the sides, is characteristic of moderate ascites. If a large quantity of fluid is present, a **general rounded swelling** and protuberance of the abdomen is found. Retraction in the epigastric and umbilical regions (the **scaphoid** or **boat-shaped abdomen**) is seen in cases of meningitis and of cerebral tumour; also in colic, especially in that due to chronic lead-poisoning. **Swelling** or **bulging** of one or

more of the regions of the abdomen: **epigastric swelling** may be due to flatulent distension of the stomach or intestine, to tumour of the liver, gall-bladder, or pylorus, or to abdominal aneurism; **right hypochondriac bulging**, to enlargements of the same organs and of the kidney; **left hypochondriac swelling**, to enlargement of the spleen and kidney, abscess, and gastric distension. **Right iliac and lumbar protuberance** may indicate affections of the cæcum and ascending colon (*e.g.*, fæcal impaction, appendicitis, malignant growths), or uterine or ovarian tumours. Enlarged or displaced right kidney may cause a swelling in these regions, while psoas abscess and inguinal hernia cause swelling of the lowest part on either side. **Left lumbar and iliac swellings** may mean displaced or enlarged kidney, perinephric abscess, enlarged spleen, cancer of the bowel, fæcal accumulation, volvulus, intussusception, psoas abscess, hernia, ovarian and uterine tumours; **umbilical swelling** may be caused by hernia, floating kidney, tumours of the liver, gall-bladder, and peritoneum, intussusception, dilated stomach, gastroptosis, enteroptosis, tubercular glands and peritonitis; **hypogastric projection** may signify distended bladder, uterine fibroid, pregnancy. The table on p. 8 gives a comparison of the commoner abdominal tumours.

3. The **Size** of the abdomen may be (*a*) decreased or (*b*) increased.

(*a*) A **Diminution** in the size of the abdomen is found in emaciation from inanition, in starvation, and in stricture of the œsophagus, in wasting diseases, in cholera.

(*b*) An **Increase** in the measurement of the abdomen occurs in **obesity**. A considerable deposition of fat takes place, not only subcutaneously, but also in the omentum, round the kidneys, and elsewhere internally. The thick fatty wall can be easily distinguished on grasping it in the fingers, and the navel is retracted. **Œdema** of the abdominal walls is an occasional cause of increased size. It is often associated with ascites, and is recognized by the pitting on pressure and the retracted navel. In case, however, that a fair amount of fluid is free in the peritoneal cavity, the umbilicus becomes flattened, or even projecting. Besides, the excess of œdema in the flanks and back, as compared with the front, facilitates the diagnosis. **Ascites**, when in large quantity, causes a general enlargement of the abdomen (see Fig. 2). Its presence is more accurately determined by means of palpation and percussion (see pp. 14

and 44). **Tympanites (meteorism)** causes a general distension of the abdomen, which is often most evident in the epigastrium. It may be possible to perceive by inspection the shape and outline



FIG. 2.—ASCITES DUE TO TUBERCULOUS PERITONITIS.

of a distended stomach, and so distinguish it from inflated bowel. This is most likely to be possible when the stomach has been artificially inflated for the purpose, and in all conditions where

## ABDOMINAL

Description of Tumour.	Situation of Tumour.	Mobility.	Shape and Consistency of Tumour.
Enlarged Spleen	Left hypochondrium, growing downward and forward	Moderately movable	Retains shape of spleen, with notches; usually hard, but less firm in acute affections
Enlarged Liver	Extends downward below costal margin in epigastric, right hypochondriac, and lumbar regions	Movable. When very large movement is restricted	Inflammatory and cirrhotic: shape normal. Cancerous: nodular and irregular. Syphilitic: irregular
Enlarged Gall-bladder	Extends downward in right half of umbilical region	Movable	Lemonade-bottle shaped; elastic or hard
Movable or Floating Kidney	Right side commonest. In hypochondriac, lumbar regions; sometimes in umbilical and iliac regions	Very movable	Normal; may be enlarged
Tumour or Cyst of Kidney	In either hypochondriac and iliac regions, extending forward and downward	Slightly movable	Solid ( <i>e.g.</i> , sarcoma), cystic ( <i>e.g.</i> , hydronephrosis), nodular ( <i>e.g.</i> , cystic disease)
Tumour of Pancreas	Epigastric and umbilical regions	Immovable	Hard, rounded swelling
Tumour of Stomach	Epigastric or umbilical regions; oftenest a little above and to the right of umbilicus (pyloric end)	Movable	Hard and nodular as a rule; most frequently rounded or spherical, but may be broad and flat
Tumour of Bowel	May be anywhere except epigastrium; commonest in left iliac fossa	Movable	Usually small, hard, and nodular; sometimes more massive
Fæcal Accumulation	Oftenest in iliac and lumbar regions; may be in umbilical or hypochondriac	Movable	Irregular masses, often cylindrical; doughy and yielding on pressure
Intussusception	Usually in umbilical and right iliac or lumbar; may be in left iliac region	Somewhat movable	Firm, sausage-shaped tumour
Appendicitis	Right iliac and lumbar, and right half of umbilical regions; centre at McBurney's point	Immovable	Irregularly rounded and hard
Enlarged Glands	Umbilical, iliac, or lumbar regions	Immovable	Irregular hard masses, often multiple
Tumour of the Omentum	Usually in umbilical region	Slightly movable	Often an irregular elongated ridge or strip across the abdomen
Uterine and Ovarian Cysts and Tumours	Hypogastric, right or left iliac, or, if very large, umbilical and lumbar regions, or even higher	Movable when of moderate size	Single, elastic, rounded (ovarian cyst); multiple, hard, irregular, or rounded (uterine fibroids)
Aneurism of the Abdominal Aorta	In middle line (epigastric or umbilical); sometimes in lumbar or iliac regions	Immovable	Elastic, pulsating, rounded



## TUMOURS

Pain and Tenderness.	Other Prominent Symptoms.
Moderate pain as a rule, sometimes severe	Commonly associated with anæmia and toxic blood states. May be due to heart or other affection obstructing portal circulation
Usually	History of alcoholism in many cases; may be secondary to primary cancerous growth elsewhere; often due to heart disease. Jaundice and ascites often accompany it. Prolonged suppuration and syphilis may be causes
Painful and tender, with severe exacerbations	Occurs usually in elderly persons; females oftenest. When due to gall-stones, pain comes in severe bouts, often followed by jaundice
Slight sickening pain on pressure	Commonest in women who have borne children; also in emaciated states; is dislocated along with other organs in enterceptosis. Dietl's crises and intermittent hydronephrosis may be observed
Usually painful	In cases of hydronephrosis seek cause of obstructed ureter— <i>e.g.</i> , impacted stone, tumour pressing on ureter, twisting or kinking of ureter in movable kidney
Painful	Found in middle or advancing age, often accompanied by jaundice and vomiting
Painful	In either sex, usually over forty; wasting, pain after food, vomiting; the vomit often contains blood, also organic acids, but no free hydrochloric acid. If pyloric (commonest site), the stomach is likely to be dilated
Pain commonly, but sometimes painless	In elderly adults. The bowels may be obstructed, or there may be diarrhœa, with mucus and blood in the stools; cachexia
Often painless	Constipation, dirty tongue, sickness, vomiting; often anæmia, piles
Painful and tender	Occurs in children as a result of violent or irregular peristalsis. The child is very ill; tenesmus, mucous and bloody stools. The tumour is sometimes to be felt per rectum
Painful and tender	Chiefly found in young adults, but may occur at any age. Vomiting, pain, which is often epigastric at first; peritonitis, either general or localized in right iliac fossa
Sometimes painless, but usually painful and tender	Evidence of tubercle elsewhere, or Hodgkin's disease (rarely); ascites. In young adults or children
Painful	Occurs in elderly adults as a rule, often accompanied by ascites
Usually painless	In females, adults usually. Metrorrhagia frequently accompanies fibroids; cachexia and symptoms of abdominal pressure
Painful	Most frequently affects male adults; history of syphilis, over-exertion

the gaseous distension of the stomach is disproportionately greater than that of the intestine. A general inflation of the abdomen with a hyperresonant percussion note (see p. 15), and possibly a displacement upward of the heart and liver, are sufficient to indicate tympanites. The condition is produced by extreme distension of the bowels and stomach, the walls of which may be practically paralysed. It is seen in peritonitis, in typhoid fever, in obstruction of the bowels, in gastro-intestinal disturbances, and in hysteria.

An obstruction to the passage of the fluids and gases of the alimentary tract at any portion of its course causes a remarkable dilatation of the proximal region of the canal. In children a hugely dilated colon may produce an enormous enlargement of the abdomen. **Free gas in the peritoneal cavity** also causes a general enlargement of the abdomen. It results from rupture or perforation of the intestine or stomach (typhoid ulcer, gastric ulcer, duodenal ulcer, tubercular ulcer, violence). It is to be recognized by a consideration of the history of the case, the sudden onset of distension, pain, probably dyspnoea and collapse. The absence of all dulness on percussion (p. 15), even of that due to the liver, is an important corroboration. **Cystic** and occasionally **solid tumours**, especially when combined, as they often are, with ascites, may give rise to considerable general enlargement of the abdomen (see Fig. 3). **Enteroptosis**, an undue mobility and displacement of the intestines and most of the other organs in the abdomen, is found chiefly in women, and is due to a relaxation of all the supporting structures of these organs, that is, weakness of the abdominal walls (after pregnancy, in debilitated states, in old age); the disappearance of subcutaneous and intra-abdominal fat; and the stretching of mesentery and peritoneal ligaments. It is naturally in the upright position that the abnormal condition is best observed. The epigastrium is not distended, but, on the contrary, may be sunken, while the lower part of the abdomen is thrown forward into a pouch, the recti and oblique muscles being stretched and separated from their fellows.

4. Certain abnormalities in the **Movements** of the abdominal wall may be seen :

(a) **Movements of Respiration.**—In children and in men breathing is much more abdominal than in women, in whom thoracic breathing is better marked. **Excessive abdominal respiration** is

seen in painful conditions of the chest—*e.g.*, fractured ribs, pleurisy, pericarditis, pneumonia; in weakness of the chest muscles from any cause (rarely), such as progressive muscular atrophy or other central or peripheral nerve lesion affecting the thoracic muscles; in dyspnœa from any cause (see p. 125), when thoracic breathing is also exaggerated; in mechanical obstruction to thoracic breathing, such as pleural effusion, pneumothorax, a



FIG. 3.—TUMOUR OF THE LIVER WITH ASCITES.

A, Position of apex-beat in fourth interspace; *iv*, *v*, *vi*, on the ribs of the same numbers. The shaded area is dull on percussion, owing to malignant disease of the liver with ascites.

flattened or otherwise imperfectly expansile chest. **Diminished abdominal breathing**, or, in other words, defective action of the diaphragm, is seen in painful conditions of the peritoneum or of the diaphragm. Thus it occurs in peritonitis from any cause, and in diaphragmatic pleurisy. It also results from weakness of

the diaphragm, as seen in paralysis due to lesions of the phrenic nerve or of its spinal centres, and it very often is due to mechanical obstruction to the proper action of the diaphragm, as may occur in abdominal distension from any of the causes named above, or from the pressure of a large pericardial effusion.

(b) **Movements due to the Circulation.**—Pulsation in the abdomen is rarely seen except in the **epigastrium**, where it is a fairly common symptom. Here the time of the pulsation is of importance. Systolic epigastric pulse is due to the contraction of the right ventricle in most cases. It may be observed in health at times when the heart is acting forcibly, as in emotional states, and as a result of exercise. It is found in cases where the right ventricle is dilated and hypertrophied, in consequence of mitral disease. A systolic pulsation in the epigastrium may be due to displacement of the apex-beat to the right from the various causes of this dislocation mentioned at p. 472; or, lastly, the movements of the heart may be transmitted to the surface through the liver or a tumour in the epigastrium. A pulsation occurring a fraction of a second (about one-tenth) after the ventricular systole is frequently observed in the epigastrium of dyspeptic patients, and especially of those who are neurotic. It is merely an exaggerated pulsation of the abdominal aorta—the so-called **irritable aorta**—and is without serious diagnostic significance. The pulsations from a normal or from an irritable aorta may be transmitted to the surface through a solid **tumour** in the epigastrium. In this case the pulsation may disappear from the tumour when the patient is placed in the knee-elbow position. In rare instances the epigastric pulsation may be due to **aneurism** of the abdominal aorta (which is much rarer than those of the thoracic aorta). Here the pulsation is ample, and the tumour is laterally expansile. The pulse in the lower limbs is weakened, and the patient suffers pain in the back and in the region of the tumour.

**Pulsation of the liver** may be the cause in rare cases of epigastric pulsation. In order to demonstrate it the edge of the liver should, if possible, be grasped by the fingers and thumb of one hand, or, what is usually more practicable, it may be pressed between two hands, one placed behind supporting the last two or three ribs, and the other in front, placed over the right hypochondrium. If the liver is pulsating, a general expansion of the organ may be felt with each systole, but this must be carefully distinguished from

pulsation transmitted through the liver from the heart or the aorta. The liver pulse may be venous or arterial. In venous hepatic pulsation the wave is originated in the dilated and hypertrophied right ventricle, in cases of mitral disease; is transmitted thence through the tricuspid valve, which under these conditions must be incompetent; it then travels against the languid venous current, and is perceived in the liver. In such cases the larger tributaries of the superior vena cava will also exhibit pulsation, which is plainly seen in the veins of the neck. Arterial hepatic pulsation may on rare occasions be found in cases where the pulsations of the arteries generally are exaggerated. This is almost exclusively in cases of aortic incompetence. In the latter condition the wave starts, of course, from the left ventricle, passing through the aorta to the hepatic artery. See Liver, pulsating, p. 189.

(c) Movements transmitted to the abdominal walls from the **muscular contractions of the viscera** may be seen at times. It is usually possible to distinguish the peristaltic movements of the stomach from those of the intestines. The former, when visible, may be observed to traverse the abdomen from left to right; the wave forms a wider and more projecting mass than that of the intestine, which commonly passes from right to left. In order that these movements may become visible, two conditions are necessary—viz., the abdominal walls must be thin, and the visceral contractions must be fairly active. In the case of the stomach these conditions are usually fulfilled when the pylorus is stenosed; whether this be due to cancer or to cicatricial contraction, the patient will probably be wasted and the stomach will be dilated, and to some extent also hypertrophied, in consequence of the difficulty it has had in evacuating its contents. It may be said as a rule, to which the exceptions are few, that visible peristalsis of the stomach signifies pyloric obstruction. Visible intestinal peristalsis is of more frequent occurrence than that of the stomach, and may be seen after careful observation in most cases where the walls of the abdomen are thin; it is most active, like that of the stomach, where the bowel has some difficulty in passing on its contents. When, therefore, the movements are vigorous, one suspects an obstruction of the intestine.

(d) Movements transmitted to the abdominal wall from **movable tumours**. The mobility of a tumour is examined by palpating its form, then directing the patient to draw full breaths, when its

capacity to move with respiration is tested; further, it is moved by the palpating hand in all directions if possible, and the direction in which it is most easily displaced is noted. It may also change its position with change in the patient's posture. If a tumour move downward with inspiration and upward with expiration, it is obviously disturbed by the movements of the diaphragm. The kidney, liver, and spleen become unduly movable at times (floating kidney, liver, or spleen), and may alter their position freely, either with respiration or with change of posture of the body. Tumours of the stomach, especially those at the pyloric end; faecal accumulations and tumours of the bowel; the enlarged gall-bladder and spleen; the kidney, even when normal in size and position; tumours of the omentum and liver, and of the ovaries and uterus, all exhibit mobility in various degrees. Enlarged glands, inflammatory exudation or abscess, the result of appendicitis or other localized inflammation, tumour of the pancreas, aneurism of the abdominal aorta, are fixed and uninfluenced by respiration, change of posture, or by palpation.

(e) Movements caused by the **presence of fluids**. In abscesses or other cystic tumours fluctuation may be recognized in the usual manner. The fingers of both hands being placed on the swelling, sharp pressure is made with one hand; the displaced fluid raises the fingers of the other hand. If the collection of fluid be large, as in the case of ascites, hydronephrosis, ovarian and parovarian cysts, a more distinctive fluctuating wave can be elicited. The palm of one hand is placed flat on one side of the abdomen or of the cyst; a sharp tap with the finger-tips or fillip with the finger-nail of the other hand is then delivered on the opposite side of the abdomen or cyst. The wave produced in the fluid by the stroke is felt by the first hand as a distinct tap. Even in the absence of fluid a similar wave may occasionally be evoked, particularly in fat abdomens; it is transmitted through the semifluid subcutaneous fat from one side of the abdomen to the other. In order to avoid error from this cause, in executing this procedure in a suspected case of intra-abdominal fluid an assistant places the ulnar edge of his hand firmly on the abdominal wall at right angles to the direction of the wave, thus cutting off the passage of superficial waves.

(f) Movements produced by the **fœtus in a pregnant uterus** may be mentioned, but are not likely to give trouble in diagnosis.

**ABDOMEN : Percussion-Sounds of.**

Conditions producing resonance—Area of abdominal resonance—Hyperresonance due to meteorism or free gas in the abdominal cavity—Loss of liver dulness—Diminished resonance, due to inanition, fluid free in abdomen, or encapsuled, and solid tumours—Situation of the dulness in ascites, in tumours of the spleen, kidneys, etc.

The general principles of percussion-sounds are discussed at p. 279, and their application to the examination of the chest are considered at p. 450. As stated in those pages, the stroke delivered by the fingers or other instrument produces a sound which varies in tone and pitch according to the elasticity or resounding quality of the adjacent structures. The best resounding medium is an air-containing space, the air being at a moderate pressure. The tympanitic or drum-like quality of the note is best heard when the collection of air is of considerable bulk; thus it is more resonant, of lower pitch and of longer duration when the surface over the stomach is percussed than when the intestine is the resounding medium. Substances which contain no free air or which are inelastic cause the percussion sound to have a dull, unmusical quality, void of resonance.

On percussing over the abdomen a variety of sounds are elicited in health. All the surface below the level of the diaphragm, with the exception of that over the liver and spleen, is tympanitic. The resonance differs, as just stated, in pitch and quality in accordance with the volume of gas in the subjacent viscus. It may thus be possible to distinguish the stomach, colon, and small intestine by their respective notes, though at times this is impossible, owing to the variable amount of gas which may happen to be present in one or other of the organs.

**Exaggeration** of the normal tympanitic resonance (**hyper-resonance**) is found in meteorism (see p. 7). In this condition the tension is sometimes so great as to displace upwards the liver, spleen, and heart. The dull areas due to the presence of these viscera may be not only displaced, but may be diminished in size. In the case of the liver this is probably due in part to a rotation of the organ on a transverse axis, so that a smaller portion of its convex surface is in contact with the anterior thoracic wall, and the liver dulness in some such cases may be quite lost.

Less frequently hyperresonance of the abdomen results from

the escape of gas into the peritoneal cavity. This occurs in consequence of a perforation of some description of one of the air-containing viscera, and is a sign of a formidable complication in gastric ulcer, duodenal ulcer, typhoid fever, tubercular, dysenteric, or other ulcer of the bowel, appendicitis; it may be due to violence (stab, crush, etc.). Free gas in the abdomen causes, as stated at p. 10, a general enlargement of the region and hyper-resonance. The loss of liver dulness is a fairly constant symptom, but as it is also seen in meteorism, it is a positive sign of only moderate value. On the other hand, the presence of liver dulness is a stronger point of evidence against perforation of the stomach or bowel. If there be free gas in the peritoneal cavity, liver dulness is lost in the anterior regions when the patient lies on his back; but the lateral and posterior surfaces over the liver are, under these circumstances, dull on percussion. If now the patient is turned on to his left side, or partly on to his face, the previously dull area is found to be clear. This, of course, is due to the free gas rising to the highest point, and allowing the heavy organs to fall deeply into the abdomen. This change of percussion-sound on change of position is the important sign.

**Diminution** of resonance is found when from any cause the gases contained in the abdominal organs are decreased in quantity. The bowels may be shrunken and empty in various debilitated states, especially in starvation, stricture of the œsophagus, wasting diseases, cholera. The percussion-note in such conditions is less tympanitic than normal. Dulness in the most dependent parts of the abdomen, with clear resonance in the higher regions, indicates free fluid in the abdomen. In this condition, with the patient lying on his back, dulness is found in the flanks, and perhaps in the iliac fossæ, while the surface surrounding the umbilicus, for a greater or less distance, in accordance with the amount of fluid present, gives a resonant note, owing to the fact that the intestines float up to the highest available level of the abdomen. When the patient changes his position, the areas of dulness and clearness are altered, as the light, gas-containing intestines are floated into the uppermost levels of the fluid (see Figs. 4 and 5). Thus, in the sitting or standing position the dull area occupies the hypogastric and umbilical regions; when lying on his side, the patient's uppermost flank is clear; in the knee-elbow position the umbilical or epigastric regions are dull, while the flanks are clear. The amount of fluid in the peritoneal cavity must of necessity cause



considerable variation in the area and degree of dulness. It may be discovered with difficulty as a narrow layer of dulness in one or both flanks, disappearing on turning the patient over on his side. On the other hand, if in excessive quantity, the bowels may be unable to reach the surface at any point, and the whole surface of the abdomen is dull in consequence. Free fluid in the abdominal cavity (*Ascites*, *q.v.*, p. 44) is the result of inflammation of the peritoneum (especially tuberculous), obstruction of the portal vein, or tumours. Other non-ascitic fluids may at times be discovered



FIG. 4.—ASCITES.

The shaded area shows the level of dulness in the recumbent position. See Fig. 5.

in the abdomen by similar signs—*e.g.*, blood, or the fluid contents of the ruptured or perforated stomach or bowels. In these cases free gas may also escape into the peritoneal cavity.

Should the fluid be encapsuled (cyst, abscess, adherent peritoneum restricting the movement of the fluid), the change of situation of the dull area on change of the patient's position is not observed, or only very slightly. The intestine is not floating free in the fluid, and therefore does not rise to the highest level of the fluid. Thus an ovarian cyst occupies the hypogastric and iliac

regions, or if it has attained large dimensions, it occupies all the central regions of the abdomen; it pushes the more movable intestines aside, with the result that the sides and upper parts of the abdomen are resonant, while the central and lower parts are dull. This distribution of the percussion-sounds alters but slightly, or not at all, with change in the patient's position. An enlarged spleen pushes in a similar manner the stomach and bowels before it in its growth, causing a left-sided dullness, corresponding in area to the size of the organ. If the kidney



FIG. 5.—ASCITES.

The same patient as Fig. 4. On raising the body the level of dullness has mounted to the umbilicus.

attains a large size, as may occur in the cystic disease of the organ, or in hydronephrosis, a tumour in either flank is found. Owing to the situation of the normal kidney, it must be very greatly enlarged before it succeeds in removing the intestines which lie in front of it, and its relations to the colon retain that bowel in front of it, even when the kidney is enormously enlarged. We therefore find that moderately enlarged kidneys are palpable, but do not give a dull note on percussion; but very greatly hypertrophied kidneys are dull, except where they are crossed by the

colon. The dull note in this latter case extends back from the region of intestinal clearness in front to the lumbar regions posteriorly.

The urinary bladder, when it has arrived at a certain state of distension, causes a dull note immediately above the symphysis pubis: if it contain 10 to 15 ounces in the case of men, or 15 to 20 ounces in women, the percussion-note is quite dull in the locality named. It is possible that in some cases the bowels may be interposed between the distended bladder and the surface, so that its presence is only discovered by deep or forcible percussion.

The various organs mentioned in describing the shape, size, etc., of the abdomen may give a dull note on percussion, if they have come to the surface of the abdomen; even if they are covered by overlying intestine, their presence may be recognized by 'deep percussion' (see p. 281).

### ABDOMINAL AUSCULTATION.

The value of this method of examining the abdomen is limited. Friction sounds may be heard at times on listening over the liver or spleen, in cases of peritonitis involving those organs (perihepatitis or perisplenitis).

The entrance of fluid into the stomach from the œsophagus is normally heard six or seven seconds after it has been swallowed; in stricture of the gullet the sound is either delayed or abolished (see Examination of the CEsophagus, p. 265.)

The passage of fluids from the ileum into the cæcum may be recognized by placing the stethoscope over the cæcum. It is best heard from four to six hours after breakfast (A. F. Hertz).

Abdominal aneurism may give rise to a systolic bruit, and a similar sound may often be elicited by pressure of the stethoscope over the aorta in the abdomen.

The fœtal heart may be heard beating in cases of pregnancy, and the placental bruit may also be noted.

### ABDOMINAL REFLEXES.

On stroking the lowest part of the side of the thorax and the side of the abdomen, the oblique and recti muscles of the same side contract in part, causing a depression of the region involved and traction of the umbilicus toward the side irritated. The reflexes are named *epigastric*, *umbilical*, or *hypogastric*, according

to the level of the contracting region of the abdominal wall. They occur in health and in disease (see Reflexes, p. 343).

**ACHOLIA** (Gr. *a*, privative; *χολή*, bile).

A deficiency in the quantity of bile produced by the liver. This is shown by the paleness of the stools, and by the absence of bile from the urine and skin (see Fæces, p. 139).

**ADVENTITIOUS SOUNDS.**

On auscultating the chest in disease of the intrathoracic organs, two classes of sounds may be distinguished. The first is composed of the various sounds natural to the region, produced by respiration and circulation, and modified to a greater or less extent by the morbid state; the other class consists of all new and abnormal sounds—the **adventitious sounds**. These are fully considered in the article on the Auscultation of the Thorax, at p. 407 *et seq.* Here it may be sufficient to enumerate the chief varieties of adventitious sounds to be met with :

A. Due to respiration :

- (a) Rhonchi, sonorous and sibilant.
- (b) Râles (crepitant, subcrepitant, mucous, gurgling).  
Any of the foregoing may partake of qualities which may cause them to be classed as consonating, metallic, or cavernous râles.
- (c) Friction sounds (pleural).
- (d) Bell sound.
- (e) Metallic tinkling.
- (f) Succussion sound.

B. Due to circulation :

- (a) Endocardial murmurs.
- (b) Exocardial sounds : (i.) pericardial friction sound ;  
(ii.) pericardial splashing sound ; (iii.) pleuro-pericardial friction sound ; (iv.) cardio-pulmonary and other sounds.

**ÆGOPHONY** (Gr. *αἴξ*, a goat; *φωνή*, the sound of the voice).

A bleating, whining quality of voice, heard as a rule on auscultation over a pleural effusion ; it may at times be heard over pulmonary consolidation (see p. 416).

**ÆROPHAGIA** (Gr. *ἀήρ*, air; *φαγῆν*, to devour).

Nervous eructations and belchings of gas. They may occur in attacks lasting a few hours or days. A considerable part of the gas is believed to be atmospheric air which has been swallowed. The symptom may be observed in cases of neurasthenia and of hysteria.

**AGEUSIA** (Gr. *ἀ*, privative; *γεῦσις*, taste).

Deficiency in acuteness of the sense of taste, or its complete abolition (ageusia), is most frequently caused by affections of the tongue or mouth, and by conditions which impair the sense of smell. It is less frequently due to disease of the trigeminal or glosso-pharyngeal nerves, or of their central connections. Facial paralysis is often accompanied by partial and one-sided ageusia (hemiageusia), owing to implication of the chorda tympani.

**AGRAPHIA** (Gr. *ἀ*, privative; *γράφω*, to write).

The inability to write, either spontaneously or from dictation. The defect is the result of a lesion involving the cortical centres presiding over written and spoken speech (see Speech Disorders, p. 373).

**AKORIA** (Gr. *ἀ*, privative; *κόρος*, satiety).

An insatiable and morbid hunger, often combined with a perverted desire for unwholesome or unsuitable substances as food. It may occur in diabetes, in hysteria, in idiocy, or in dementia (see Appetite, p. 35).

**ALAR CHEST (Pterygoid Chest).**

A characteristic form or subtype of chest, in which the shoulders slope excessively, the neck is long, and the scapulæ project backwards. An exaggerated obliquity of the ribs, with consequent diminished capacity of the thorax, is the chief feature in the condition (see p. 468).

**ALBUMINURIA.**

The tests best suited for the practitioner in examining the urine or albumin are described in the article on Urinary Examination

(p. 531). The clinical significance of the occurrence of albumin in the urine is considered in detail in the section on Urinary Abnormalities (p. 513).

### **ALBUMOSURIA.**

The occurrence of hetero- and deuterio-albumoses in the urine may be demonstrated by the biuret reaction, by their precipitation by nitric or picric acids (the precipitate dissolves on heating), and by sodium chloride (see Urinary Examination, p. 533). Their clinical significance is indicated in the article on Urinary Abnormalities, p. 524.

### **ALEXIA** (Gr. *ἀ*, privative; *λέξις*, a word).

Inability to understand written characters, a symptom of certain forms of aphasia (see Speech Disorders, p. 373).

### **ALLOCHEIRIA** (Gr. *ἄλλος*, another; *χείρ*, the hand).

Inability to localize pain. On pinching or pricking one limb, the pain may be referred to the opposite side of the body, or to some other region. This symptom may at times be observed in locomotor ataxia, multiple sclerosis, and hysteria (see Sensation Disorders of, p. 360).

### **ALTERNATE PARALYSIS.** See **Crossed Paralysis**, p. 113.

### **AMAUROSIS** (Gr. *ἀμαυρόω*, to darken).

A term used to signify absolute blindness, as distinguished from amblyopia, which indicates a partial loss of sight. The word 'amaurosis' was formerly applied only to those cases of blindness for which no obvious causal lesion in the eye was found. Many of these cases have now been shown by modern methods of examination to be due to local affections of the eye—*e.g.*, disease of the optic nerve, of the retina, or of the choroid; still, the term 'amaurosis' continues in use to signify loss of sight, even in those conditions where the eye lesion is obvious. Among the conditions without definite eye lesions may be mentioned migraine, hysteria, hæmorrhage, electric shock, poisoning by quinine or salicylate of soda. Disease of the brain and spinal cord may be the cause of

blindness, which may then be termed cerebral or spinal amaurosis. In blindness from local affections of the eye causing loss of sight it is also customary to make use of the same term (see Vision, Disorders of, p. 543).

**AMBLYOPIA** (Gr. ἀμβλῦσις, d. Ped; ὤψ, the eye).

A term employed to indicate dimness of vision, as distinguished from amaurosis (*q.v.*, p. 221) or total blindness. It is understood that the defective vision is not of a nature that can be rectified by the use of glasses—thus, myopia and presbyopia cannot be properly named amblyopia. Various toxic states give rise to the symptom, of which the most important form is that due to the abuse of tobacco and alcohol. Here the loss of sight affects chiefly the central regions of the field of vision, the condition being known as toxic central amblyopia, or central scotoma. Defective vision also results from diabetes, uræmia, poisoning by quinine, salicylate of soda, iodoform, etc. (see Vision, Disorders of, p. 543).

**AMPHORIC SOUNDS** (Gr. ἀμφορεῖς, an ancient wine vessel of large dimensions, holding about nine gallons).

The various sounds perceived by auscultation, or elicited by percussion over the thorax, have, under certain conditions, a curious but not disagreeable quality. There is an added reverberation and metallic quality in the sounds produced by percussion of the patient's chest, and by his voice, cough, and respiration, which may be imitated by speaking or blowing into a narrow-mouthed vessel, or by flicking the moderately-distended cheeks with the mouth closed. The conditions giving rise to the sound are a smooth walled, air-containing cavity of large dimensions, preferably, but not necessarily, communicating by an opening with the external air. These conditions are best fulfilled in pneumothorax, but may also be found in a large pulmonary cavity, the walls of which are not in apposition, and are moderately smooth. A similar sound is sometimes observed on percussing or on listening over the stomach or intestines.

The amphoric quality, then, being added to one or other of these sounds, one finds, in the case of percussion, **amphoric**

or **metallic resonance**; the respiratory sounds become **amphoric breathing**.

Further reference will be found in the articles on Percussion and Auscultation of the Thorax (pp. 450 and 407).

### **AMPHOTERIC REACTION** (Gr. ἀμφότερος, in both ways).

In testing the reaction of urine, it may be found that it turns blue litmus red, and that red litmus is turned blue by the same specimen of urine. This is due to the effect of both acid and basic phosphates on the litmus, and the reaction of the urine is therefore said to be amphoteric.

### **ANÆMIA** (Gr. ἀν, privative; ἄμα, blood).

Definition of anæmia—Symptoms commonly associated with anæmia—Subjects to be investigated by examination of the blood—Colour index—Microcytes—Megalocytes—Normoblasts—Megaloblasts—Gigantoblasts—Poikilocytosis—Anæmic degeneration—Leucocytes—Ehrlich's classification of leucocytes—Characters of the blood in childhood—Primary anæmia—Secondary anæmia—Comparative table of diseases characterized by anæmia.

The term 'anæmia' is commonly used to denote, not a loss of blood, as its etymology suggests, but a decrease in the more important constituents—viz., red cells, hæmoglobin, or albumin. The total quantity of blood in the body is not necessarily diminished, but, on the contrary, may in some cases (*e.g.*, chlorosis) be even increased. A variety of qualitative changes may be observed in the different constituents of the blood, which in many cases form a useful means for identifying the condition under examination.

While anæmia must be regarded as a mere sign in many diseases, it is commonly accompanied by certain other signs and symptoms, of which it is itself the chief cause. One observes in most cases of anæmia, however produced—

1. Pallor of the skin and mucous membranes. The colour of an anæmic person is in most instances a dull, slightly pinkish white, but other shades are often seen—in chlorosis a greenish tinge; in pernicious anæmia a yellowish, rather than a pinkish, shade. The anæmia of malignant disease has a peculiar sallow, unhealthy tint; in pregnancy, in Addison's disease, and in anæmias too vigorously treated with arsenic, a bronzed or pigmented shade may be added to the pallor.



2. A systolic murmur, best heard near the left side of the sternum, at the level of the second or third costal cartilage; also we frequently hear a systolic murmur, whose point of maximum intensity is the region of the apex-beat. A loud, rumbling murmur, either systolic in time or, more commonly, continuous—the **venous hum**, or **bruit de diable** (see p. 444), is heard close above the clavicle, near its sternal end.

3. Œdema, chiefly of the lower extremities.

4. Certain subjective symptoms, among which may be mentioned languor and weakness, palpitation, dyspnœa, vertigo, syncope, headache, ringing in the ears.

5. Diminution in the number of red corpuscles, and in the amount of hæmoglobin and proteid substances in the blood.

While due attention must, in all cases of anæmia, be given to the general symptoms presented by the patient, it has to be borne in mind that no proper understanding of the case can be acquired without an examination of the blood. The technique of the examination has of late been much simplified, and may be carried out by the practitioner in many cases. The methods to be adopted are described in the article on Blood Examination, by Dr. Houston, at p. 58.

For clinical purposes it is usually necessary to investigate (a) the number of red corpuscles per cubic millimetre of blood; (b) the percentage of hæmoglobin present as compared with normal blood; (c) the character of the red cells—*i.e.*, whether larger or smaller than normal, whether misshapen, nucleated, unduly pale, pigmented, or otherwise divergent from normal erythrocytes; (d) the number of colourless corpuscles in a cubic millimetre of blood; (e) the varieties of white corpuscles found in a stained film, and the proportionate count of these various forms; (f) any abnormal constituent of the blood must be noted, such as micro-organisms, parasites, etc. In many affections, independent of anæmia, it is desirable to determine the specific gravity, the opsonic power, the quantity of calcium salts present, freezing and coagulating points, etc. (see Blood Examination, p. 74).

The **colour index** is the relation of (a) the number of red cells present to (b) the percentage of hæmoglobin. The number of red cells in a cubic millimetre of the blood under observation being ascertained, this number is reduced to its due percentage of a normal or standard blood; the two percentages—that of the hæmoglobin and that of the red cells present—can then be at

once compared. The proportion of hæmoglobin present to that of red corpuscles is known as the **colour index**, and is thus expressed numerically :

$$\text{Colour index} = \frac{\text{Percentage of hæmoglobin.}}{\text{Percentage of red corpuscles.}}$$

Normally, this works out as unity, the normal percentage of hæmoglobin being, of course, taken as 100, and normal blood, containing 5,000,000 red cells per cubic millimetre, is also 100 per cent., giving the equation : Colour index =  $\frac{100}{100} = 1$ .

On the other hand, if we find a sample of blood to contain only 50 per cent. of the quantity of hæmoglobin normally present, and at the same time it contains, say, 3,000,000 of red cells per cubic millimetre (that is, 60 per cent. of the normal), we get the expression : Colour index =  $\frac{50}{60} = 0.83$ . The colour index in this example is, therefore, 0.83.

In chlorosis it is found that the loss of hæmoglobin is usually much greater than the loss of red cells—that is to say, each red cell is poor in hæmoglobin. We may find, for example, 40 per cent. of hæmoglobin in a sample of chlorotic blood containing 80 per cent. of the normal number of red cells, showing the low colour index of 0.5.

On the other hand, the diminution in the number of the red cells may be as great as, or even greater than, the reduction in the quantity of hæmoglobin (as may be observed in pernicious anæmia). Thus we may find a sample of blood to contain 50 per cent. of the normal quantity of hæmoglobin, while the red cells number 2,000,000—that is, 40 per cent. of the normal. The colour index in this case is high—viz., 1.25.

In anæmic conditions a high colour index—*i.e.*, one reaching or exceeding unity—is characteristic of progressive pernicious anæmia. It is also found in many of the cases of anæmia due to the presence of the tapeworm *Bothriocephalus latus*, and possibly in that produced by *Ankylostoma duodenale*, oxyuris, and ascaris (secondary pernicious anæmia). All forms of anæmia, with the above exceptions, show a low colour index, the lowest figure being, as a rule, touched in chlorosis.

(c) The character of the individual red cells often supplies useful information. The more severe forms of anæmia are marked by an extreme variety in size and shape of the corpuscles. In-

stead of presenting a normal diameter of  $7\mu$  to  $8\mu$  ( $\mu = \frac{1}{1000}$  of a millimetre =  $\frac{1}{254000}$  of an inch), they may vary from cells of  $1\mu$  to  $4\mu$  in diameter (**microcytes**), to forms measuring as much as  $20\mu$  in diameter (**megalocytes**). Tallquist states that an abundance of microcytes points to a rapid destruction of the blood; and megalocytes, when present, indicate an attempt at active regeneration on the part of the blood.

**Nucleated red cells** may be found at times. In health a very few of them may be discovered in the blood of infants. When of normal size, with large, ovoid, deeply staining nucleus, they are termed **normoblasts**, and may be found in any anæmia, moderate or severe in type—*e.g.*, post-hæmorrhagic anæmia, chlorosis, etc. In the severer anæmias the normoblasts are commonly accompanied by larger forms, from  $10\mu$  to  $20\mu$  in diameter, known as **megaloblasts**, and by still larger cells of the same type, **gigantoblasts**.

'When the majority of a considerable number of nucleated red cells are megaloblasts, especially if gigantoblasts are present, and unequal mitotic figures are observed, the diagnosis of primary pernicious anæmia is justified. The presence of a few megaloblasts alone is usually found only in primary pernicious anæmia, but may occur in pernicious secondary anæmia. Possibly the discovery of a single gigantoblast may warrant the diagnosis of primary pernicious anæmia' (Ewing).

**Poikilocytosis**.—A number of red cells of misshapen and distorted appearance may be found in cases of severe anæmia, the condition being termed by Quincke **poikilocytosis**, and the cells **poikilocytes** (Gr. *Ποικίλος*, varied).

**Anæmic Degeneration**.—Under the term **anæmic** or **polychromatophilic degeneration**, or **polychromasia**, Ehrlich drew attention to anomalies in the staining capacity of certain red cells in cases of chronic anæmia, and ascribed them to a progressive coagulation necrosis of the cell in the act of losing its hæmoglobin to the plasma. Other observers, however, regard these cells, on the contrary, as the youngest red cells of the blood (see Blood Examination, p. 67).

**Granular degeneration** of the red cells has been frequently observed in pernicious anæmia.

(d) **Leucocytes**.—The number of white corpuscles to be found in a given quantity of blood varies considerably in health, being influenced by digestion and other physiological processes, by the

age, individual peculiarities, etc., of the subject. The average number present in 1 cubic millimetre may be stated as about 8,000, ranging, however, very much above and below that figure in healthy individuals. A considerable increase in the number of colourless cells is observed in many diseased conditions, and is termed **leucocytosis** (*q.v.*, p. 175), while a distinct diminution in their number is known as **leucopenia** (see p. 187).

(e) The **differential count** of the white cells—that is, the percentages of the different forms of white corpuscles found in any specimen of blood—is of great diagnostic value. A full account of the recognized varieties of white cells, and of the methods to be followed in identifying them, will be found in the article on Blood Examination, at p. 67. Here it will suffice merely to enumerate the forms which have been observed in health and in disease. Ehrlich's classification and percentages are as follows:

(i.) Lymphocytes, ('small lymphocytes'), occurring in health in the proportion of 22 to 25 per cent. of all the leucocytes present.

(ii.) Large mononuclear leucocytes ('large lymphocytes'). These cells are believed by Ehrlich to be distinct from the small lymphocytes.

(iii.) Transitional forms, comprising with group (ii.), from which, according to Ehrlich, they are derived, a proportion of 2 to 4 per cent. of the white cells.

(iv.) Polymorphonuclear or polynuclear leucocytes form the chief constituents of the white cells, amounting to about 70 per cent.

(v.) Eosinophile cells, comprising 2 to 4 per cent.

(vi.) Mast cells, occurring very sparsely, about 0.5 per cent.

These six forms include the varieties of white cells found in the healthy adult; in disease the same cells may occur, though perhaps in different proportions, and, in addition, the following:

(vii.) Myelocytes, of which two varieties are recognized—the 'mononuclear cells with neutrophile granulation,' and the 'mononuclear eosinophile cells.' Both these cells are normally found in the bone-marrow; the first is usually known as the myelocyte, and the second as the eosinophile myelocyte.

Other rare forms, whose clinical significance is not yet defined, are the 'small neutrophile pseudo-lymphocyte' (Ehrlich), and the 'stimulation forms' (Türk).

(f) A bacteriological examination of the blood may exceptionally be desirable for diagnostic purposes.

(g) The condition of the serum may require investigation (see p. 85).

It may happen, infrequently perhaps, that the anæmic condition under observation occurs in an infant or young child. We must then bear in mind that normally certain characters are typical of infancy—viz. (i.) lymphocytes form a larger proportion of the white cells than is stated above; (ii.) blood formation more easily reverts to the embryonal type, as may be seen by the presence of normoblasts; (iii.) leucocytosis is more actively excited by chemotactic influences (see *Leucocytosis*, p. 177); (iv.) the spleen enlarges readily in all anæmias of infancy; (v.) in rickets hyperæmia of the bone-marrow is a feature (Ewing).

The symptom anæmia may be usefully employed in linking together a number of morbid conditions possessing this feature in common, and a consideration of the differences in type of anæmia presented is an important aid in arriving at an understanding of the nature of the affection.

**Primary Anæmia.**—In the large majority of affections characterized by anæmia this symptom is obviously secondary to disease in some organ or tissue other than the blood or blood-forming tissues. There are, however, at least two affections in which it has not yet been proved that the blood state presented by them is secondary to disease elsewhere. They are therefore provisionally described as **primary anæmias**, and they are (1) **chlorosis** and (2) **primary pernicious anæmia**. In addition, a group of anæmic conditions in which the lymph-glands, spleen, and bone-marrow are the seat of morbid changes in varying degrees, may fairly be included among the primary anæmias. Among these may be mentioned leukæmia, Hodgkin's disease, splenic anæmia, Banti's disease, anæmia infantum of von Jaksch, and perhaps chloroma. While anæmia in the sense of reduction in the quantity of hæmoglobin is present in these affections, it is not a prominent feature in all of them.

The primary anæmias present certain features in common, justifying the use of the term 'anæmia of the primary type.' This type may be regarded as characterized by considerable decrease in the number and in the hæmoglobin contents of the red cells (and especially of the first—in other words, there is often a high colour index); by marked changes in the size and form of the red

cells (poikilocytosis), and by the presence in many cases of normoblasts and megaloblasts. No characteristic changes in the white cells need be specified. The commoner or 'secondary type' of anæmia presents a less severe grade of impoverished blood; poikilocytes are less frequently observed, and nucleated cells are only found in the most severe cases, and then only in very small numbers.

The best example of a primary type of anæmia is seen in pernicious anæmia; it is also observed in severe cases of chlorosis, and in extreme cases of severe secondary anæmias. In one important particular already referred to pernicious anæmia differs from other severe forms of anæmia—that is, in its high colour index.

**Secondary Anæmia.**—Anæmia secondary to disease or injury which primarily affects some organ other than those concerned in the formation of the blood is of an extremely common occurrence. The condition of the blood in these cases shows many features in common. The quantity of hæmoglobin is reduced in a proportion as large as or larger than that of the red cells—that is, the colour index is low. In moderate degrees of anæmia no morphological changes are seen in the red corpuscles except a decrease in their colour. Severe secondary anæmias show important changes in the structural and nutritional characters of the red cells: poikilocytosis may occur; normoblasts may be found in considerable numbers in grave cases, and occasionally a few megaloblasts may be discovered. The white corpuscles are sometimes increased in number, as is often the case in post-hæmorrhagic anæmia, while in other instances they may be diminished in quantity. The symptoms of secondary anæmia differ in no essential particular from those of the primary forms. The primary affection from which the blood state is derived will, of course, add its share of morbid signs.

Osler classifies secondary anæmias, on the basis of ætiology, as follows:

1. **Anæmia from Hæmorrhage.**—This may be either a rapid or a gradual loss of blood, and may be from injury or from disease. Familiar instances of the latter are gastric ulcer, phthisis, cancer of the stomach or bowel, fibroid tumours and other affections of the uterus, piles, purpura, intestinal parasites, etc.

2. **Anæmia from Long-continued Drain on the Albuminous Materials of the Blood.**—*e.g.*, chronic suppuration,

TABLE OF AFFECTIONS CHARACTERIZED BY ANÆMIA

Disease.	Etiology.	General Condition.	Blood Condition.
<b>Chlorosis</b> ..	In female adolescents from constitutional disturbance or bad hygiene	Colour of patient greenish. Degree of anæmia variable. Spleen, liver, lymph-glands not materially affected	Colour index very low. Red cells moderately decreased in number. Bulk of blood increased. White cells without characteristic change
<b>Pernicious Anæmia</b>	Adults of either sex. Cause often unknown; may follow repeated hæmorrhages or other anæmic conditions	Colour of patient yellowish. Anæmia often intense. Spleen perhaps enlarged. Pyrexia common. Urine often dark colour from increased urobilin	Colour index high. Red cells often much decreased in number. Normoblasts, megaloblasts, gigantoblasts may be found. Poikilocytosis. If leucocytosis present, indicates complications
<b>Leukæmia:</b> <b>(a) Myelæmia..</b>	Adult men chiefly. Cause obscure; may be traumatic	Spleen much enlarged. Progressive asthenia. Moderate anæmia	Colour index low. Red cells moderately decreased (perhaps two or three millions). Extreme leucocytosis, of which myelocytes are 40 to 50 per cent.
<b>(b) Lymphæmia</b>	Patient is younger. Disease rarer than (a). Cause equally obscure	Lymph-glands generally enlarged. Spleen often enlarged. Progressive asthenia and anæmia	Colour index low. Moderate anæmia. Marked lymphocytosis
<b>Hodgkin's Disease</b>	Most frequent in early and late adult life. Cause uncertain; perhaps local injury or irritation	Spleen and lymph-glands enlarged; the latter at first separately and distinctly enlarged; later become adherent, but rarely suppurate	Colour index low. Moderate anæmia of secondary type
<b>Splenic Anæmia</b>	All ages attacked. Cause unknown	Spleen enlarged. Severe anæmia	Colour index low. Anæmia usually intense
<b>Banti's Disease</b>	All ages. Cause is obscure	Enlarged spleen, followed by cirrhosis of the liver, with its usual symptoms of ascites, jaundice, etc.	Colour index low. Anæmia usually intense
<b>Anæmia Infantum</b> .. ..	Children under four years. Rickets, syphilis, intestinal catarrh, tubercle	Spleen greatly enlarged; liverslightly; lymph-glands still less enlarged	Colour index low. Intense anæmia approaching the primary type. Leucocytosis, with large percentage of lymphocytes
<b>Chloroma</b> ..	Cause unknown	Tumours, chiefly in orbits and temporal fossæ. May present a green colour. Exophthalmos, optic neuritis, hæmorrhages	Considerable anæmia. Lymphocytosis. A few myelocytes may be found
<b>Secondary Anæmias</b>	The primary affection is to be sought	Varies with the primary affection	Colour index low. In most cases a moderate anæmia, of secondary type

Bright's disease, prolonged lactation, wasting diseases, such as cancer and phthisis.

3. **Anæmia from Inanition** (defective nutrition from any cause).—This may be the result of an unsuitable or deficient food-supply, as may be seen daily in the tea-fed anæmic operatives in factories. The food may be obstructed in its passage to the stomach, as in cancer of the stomach; the digestive organs may be unable to prepare the food so as to permit its assimilation, as in chronic forms of dyspepsia.

4. **Toxic Anæmia**—the result of poisons in the blood: syphilis, rheumatism, lead, mercury. Infectious and other fevers cause a rapid deterioration of the blood, partly owing to toxæmia, but also as a result of imperfect digestion and disturbance of the blood-forming function. In this group may also be placed parasitic diseases of the blood in which anæmia is observed—viz., malaria, bilharziosis, piroplasmosis. The diagnosis of these tropical affections is made by the discovery of the parasite in the blood (see Blood Examination, p. 72).

**ANÆSTHESIA** (Gr. *ἀν*, privative; *αἴσθησις*, a sensation).

Loss of tactile sensibility, the result of interruption to the passage of afferent nerve impulses. The methods of testing for the symptom and its clinical significance are considered in the article on Disorders of Sensation (p. 358).

**ANALGESIA** (Gr. *ἀν*, privative; *ἄλγησις*, sense of pain).

Diminution or loss of the capacity to perceive painful stimuli. The clinical significance of the symptom is considered in the article on Disorders of Sensation (p. 356).

**ANARTHRIA (Dysarthria)** (Gr. *ἀν*; *ἄρθρῶν*, to utter distinctly).

A speech defect, in which the chief fault is imperfect articulation of the words, which may, however, be well chosen and properly placed in the sentences. Defective rhythm or imperfectly controlled force and rapidity of the spoken words also characterizes a series of speech defects (scanning speech, stammering, choreic speech), which may for convenience' sake be considered under anarthria. The term, however, was applied to, and is generally understood to indicate, an imperfect articulation of the words, such as is most typically observed in bulbar and pseudo-bulbar



paralysis. The lesion is situated in some region of the motor tract, from (and including) the cortical centres in the Rolandic area which govern speech movements to the nuclei in the pons, medulla, and anterior cornua of the cord, thence to the peripheral terminations of the motor nerves implicated. The typical condition is, therefore, a paralysis of the muscles which are necessary for the correct pronunciation of words. The same muscles have in some instances other duties to perform—*e.g.*, mastication, movements of the tongue in eating, etc.—which are equally impaired by the lesion which causes anarthria. In this respect anarthria differs from aphasia, for in the latter condition it is only the movements of speech that are affected. The subject is more fully considered in the article on Speech Disorders, p. 373.

**ANASARCA** (Gr. *ἀνά*, through; *σάρξ*, the flesh).

A dropsical accumulation of lymph in the tissues throughout the body. The term merely differs from œdema in the more general distribution of the symptom, œdema being a localized form of the same condition. The subject is considered in the article on Œdema, (p. 260).

**ANISOCORIA** (Gr. *ἄνισος*, unequal; *κόρη*, the pupil).

Inequality in size of the two pupils is sometimes observed in health. It may depend upon undue dilatation of one pupil, the other being normal, or one of the pupils may be diminished in diameter, or both pupils may be of abnormal dimensions.

The pathological conditions giving rise to inequality of pupils are numerous, the more important of them being the unilateral action of mydriatics or myotics, lesions of the third nerve or of its nucleus, lesions of the sympathetic nerve fibres, locomotor ataxia and general paralysis of the insane, iritis, keratitis, glaucoma, and other local affections of the eyes. (See Pupils, p. 316.)

**ANKLE-CLONUS.**

Rhythmical extending movements of the foot, set up by putting the calf muscles on the stretch (inducing reflex readiness or 'myotatic irritability' of the muscles), and keeping up the extension of the muscles by pressure on the sole of the foot. This is rarely seen in health. (See Reflexes, p. 339).

**ANKLE-JERK (Achilles Tendon Reflex).**

The foot is dorsally flexed, so as to stretch the calf muscles. Muscular reflex readiness, or 'myotatic irritability,' is thereby induced, and a tap on the tendo achillis suffices to cause an extending jerk of the foot. This phenomenon is sometimes seen in healthy subjects, more frequently than the ankle-clonus, but much less frequently than the knee-jerk (see p. 339).

**ANOREXIA** (Gr. *án*, privative; *ὄρεξις*, an appetite).

Diminution or loss of appetite is a symptom common to very many diseased states. It may be due to—(1) disease of the stomach and other organs of digestion; (2) general constitutional diseases not specially affecting the digestive organs—*e.g.*, fevers, tuberculosis, syphilis, affections of the liver and kidneys, anæmia, malignant disease, etc.; (3) emotions—*e.g.*, anxiety, suspense, or annoyance. (See Appetite, p. 36.)

**ANOSMIA** (Gr. *án*, privative; *ὄσμη*, smell).

Loss of the sense of smell is usually the result of affections of the nose, but may be the result of disease of the intracranial portion of the olfactory apparatus. It may also occur in hysteria and in lesions of the fifth nerve, in the latter instance because of the dryness of the nasal mucous membrane following injury to the trigeminal nerve. (See Abnormalities of Smell, p. 372.)

**ANTERIOR AXILLARY LINE.**

A vertical line drawn on the thorax, passing through the spot where the anterior axillary fold joins the chest-wall, the arm being held out horizontally. It is used in referring to the topography of the chest (see p. 464).

**ANVIL SOUND.** See **Bell Sound**, p. 56.

**AORTIC AREA.**

The term is applied to that portion of the chest-wall in the immediate vicinity of the second right costal cartilage. At this place sounds originating at the aortic orifice are, as a rule, best heard. An important exception is the murmur of aortic regurgitation, which is frequently best heard in the lower part of the sternum, and to its left side. This region is therefore sometimes spoken of as the **secondary aortic area** (see p. 435).

**APE-HAND.** See **Contracture**, p. 104.

**APEX-BEAT.**

This term is used to indicate that portion of the chest-wall with which the apex of the heart comes in contact at each systole of the ventricles. In the normal adult thorax it is situated in the fifth left interspace, internal to the vertical line through the nipple. Many departures from this topography are to be observed as a symptom of heart and other affections. The subject is considered in detail in the article on the Shape and Movements of the Thorax at p. 472.

**APHASIA** (Gr. *á*, privative; *pháris*, speech).

A disordered condition of the speech, in which the patient may be unable to understand or to express written or spoken language, or may make use of inappropriate or wrongly-placed words in his speech. It is due to a lesion of the brain, involving the centres concerned in the reception, storing, and recalling of the memories and conceptions which are essential to the function of intelligible speech. The subject is more fully considered in the article on Disorders of the Speech (p. 378).

**APHEMIA** (Gr. *á*, privative; *phémí*, to speak).

The term was formerly used as a synonym for aphasia, but is now usually restricted to motor aphasia.

**APHONIA** (Gr. *á*, privative; *phónē*, the voice).

Defective vibration of the vocal cords from any cause gives rise to loss of voice. The subject is considered in the article on Abnormalities of the Voice (p. 381).

**APPETITE** (L. *appeto*, to long for).

The natural desire for food and its modification by disease are the only forms of disturbed appetite that need be considered here.

The sensation of hunger is probably due not so much to the absence of food from the stomach as to the want of the products of digestion in the blood. After a period the usual enrichment of the blood from the alimentary canal is expected by the tissues, and, if not forthcoming, the want is announced to the consciousness by a sensation which is referred to the stomach. Probably changes in the gastric mucous membrane are the immediate

cause of sensory impulses giving rise to the sensation of hunger. This may be compared with the analogous condition in the mucous membrane of the soft palate in thirst.

In disease one may observe—(a) diminution or loss of appetite (**anorexia**); (b) excessive appetite (**hyperorexia**, **akoria**, or **bulimia**); (c) perverted appetite (**pica**).

(a) **Diminished or Lost Appetite** is one of the most familiar symptoms of ill-health. It may be due to—(1) Diseases of the stomach and other organs of digestion. One of the first effects of almost every organic stomach affection is the loss of appetite. In some cases of gastric ulcer, however, the appetite may persist. The gastric neuroses (rarer occurrences than is commonly supposed) may present this symptom, sometimes in an extreme degree. In hysteria, for example, the condition known as *anorexia nervosa* may persist for a considerable time, in which the desire for food is entirely abolished. On the other hand, some functional or neurotic stomach affections exhibit an excessive or a perverted appetite. (2) General constitutional diseases are frequently marked by loss of appetite—*e.g.*, fevers, tuberculosis, syphilis, debilitated states following or accompanying disease elsewhere than the stomach, such as malignant disease, anæmia, affections of the liver and kidneys, etc. (3) Emotions, such as anxiety, suspense, or annoyance.

(b) **Excessive Appetite** is rarely of diagnostic value. It may occur in diabetes, in hysteria, in idiocy, and in dementia. It is sometimes observed in the gastro-intestinal irritation of children.

(c) **Perverted Appetite**.—A perverted or insatiable appetite is sometimes seen in pregnancy, in hysteria, in chlorosis, in idiocy, and in dementia. It is sometimes known as **pica** (L. *pica*, the magpie), from the greedy and omnivorous habits of the bird.

#### **APRAXIA** (Gr. *ἀ*, privative; *πραξις*, a doing).

Inability to recognize objects and to discern their uses. It is due to a cerebral lesion, which so interrupts the course of afferent or sensory impulses of all descriptions that they are unable to evoke any memory from the psychical centres as to the nature of the stimuli causing the nerve impulses. It is associated with the function of speech and the conceptions connected with language; it is, therefore, considered in the article on the Disorders of Speech (p. 380).

**APROSEXIA.**

In cases of enlargement of the tonsils, with naso-pharyngeal obstruction from adenoid growths, the child is frequently seen to be deficient in mental activity. He is dull, lethargic, and unable to fix his mind for any length of time on his work or play. The condition has been termed aprosexia.

**ARCUS SENILIS** (*L. arcus*, an arch; *senilis*, belonging to old people).

An arch or rim of opacity on the cornea near, but not quite reaching, the junction of the cornea with the sclerotic. It is the result of a fatty degeneration of the corneal tissue, and was formerly believed to indicate a fatty or atheromatous degeneration of the heart or other organs. This is not the case, and it is really of no diagnostic value. It does not contra-indicate operations on the cornea, as a wound of the cornea in this condition is found to heal readily enough.

**ARGYLL-ROBERTSON PUPILS.**

The pupils fail to respond by contraction to the stimulus of light, while they contract in the normal manner in the act of accommodation to near vision. This phenomenon is observed commonly in locomotor ataxia, and also in general paralysis of the insane (see p. 324).

**ARM-JERKS.**

Deep or so-called tendon reflexes of all the available tendons of the arm may, in certain conditions, be elicited by putting the respective muscles on the stretch, and then giving a gentle tap with the finger-tip. (See Reflexes, p. 339.)

**ARRHYTHMIA.**

Varieties of arrhythmia—Origin of the heart-beat—Nature of the stimulus originating the heart-beat—Maximal contraction—The refractory period—Extra systole—Condition of heart muscle in healthy action—Conducting muscular fibres—Heart-block—Conditions concerned in the heart's contraction—Causes of arrhythmia—Intermittent pulse—Bigeminal and trigeminal pulse—Pulsus alternans—Pulsus paradoxus.

Disturbances of the normal rhythmical beat of the heart take a variety of forms. The contractions may be simply **irregular**, both in force and interval, without order or method in the irregularity;

or the disturbed rhythm may assume a certain degree of consistency or character of its own. Thus there may be an omission of a beat at more or less regular intervals (**intermittent pulse**); or the beats may be alternately weak and strong (**alternating pulse**); or they may occur in groups of two or three strokes, separated from neighbouring groups by a somewhat prolonged interval (**bigeminal** or **trigeminal pulse**); or the auricles may contract more frequently than the ventricles (**heart-block**); or one ventricle only may at times take part in the systole (**hemisystole**), etc. The causes and nature of the disordered rhythm are in many cases only imperfectly understood, but the researches from the clinical and laboratory standpoints by Gaskell, Engelmann, Mackenzie, Wenkebach, Howell, and others, have within recent years added much to our knowledge of the subject. A short review of the present current opinions may be permitted at the outset.

Previous to Gaskell's well-known experiments (1883) it was generally taught that the rhythmical contractions of the heart depended upon the nervous system, and the presence of nerve ganglia in the heart muscle demonstrated by Remak and by Bidder supported this opinion. Since that period, however, evidence has been accumulating, and is now by most physiologists considered convincing, that the heart-beat is not of nervous origin, but is originated and sustained by the inherent qualities of the muscular fibres of the heart, stimulated by some local excitant, probably of a chemical nature.

It is at the entry of the great veins into the auricles that the heart-beat commences; it then proceeds as a peristaltic wave from the auricles to the ventricles. These contractions may be continued for hours after the heart has been removed from the body, provided a supply of suitable fluid be constantly passed through the heart; indeed, a fragment of the heart muscle may similarly pulsate if immersed in suitable fluid. It has been found that a solution of calcium, sodium, and potassium salts (Ringer's solution) is extraordinarily efficient in maintaining the contractions of the excised heart, and this in spite of the fact that the fluid is devoid of all nutritive qualities, showing that the heart contains in itself an abundant supply of energy-yielding material. It is possible that the presence of the salts may set up a chemical decomposition in some substance in a state of loose combination in the cardiac tissue, and that the products of this decomposition, after oxidization (for oxygen is necessary to the contraction), may

form a hypothetical 'inner stimulus,' whereby the contraction is initiated or effected.

It was first shown by Bowditch that, once a sufficient stimulus has accumulated, the heart contracts with its full power, quite irrespective of the strength of the stimulus. This **maximal contraction** furnishes one of the distinctions between the heart and the skeletal muscles, and it is observed, not only during this natural and presumably chemical stimulation, but also as a result of electrical or mechanical stimulation. As soon as the stimulus has initiated a contraction, the material in the cardiac tissues available for the production of energy is, for the moment, completely used up, and fresh stimuli produce no effect until a sufficient accumulation of energy-yielding material has been decomposed. This period of non-response to stimuli, or **refractory period**, begins immediately before the ventricular systole; in the earliest portions of the diastole a contraction is with difficulty

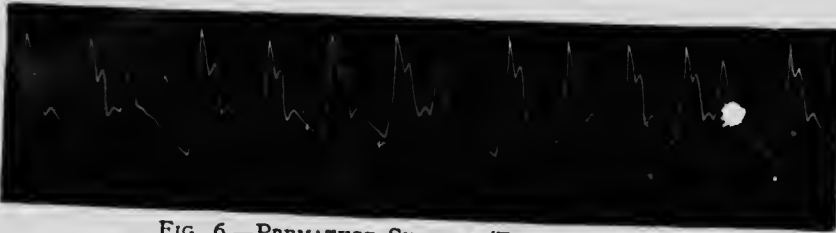


FIG. 6.—PREMATURE SYSTOLE (EXTRA SYSTOLE).

The premature contraction in this case occurs rhythmically after every fourth normal beat (Mackenzie).

evoked, but as the diastole advances the muscle becomes more and more sensitive. Owing to functional or organic disease, a refractory period may be shortened, or a stimulus may effect a contraction much too soon, giving rise to an **extra systole**, or **premature systole**, of greater or less strength, following quickly upon a normal beat. This extra systole has the usual refractory period, which may now coincide with the moment at which the following stimulation should have taken place, and will, therefore, abolish it (see Fig. 6). The occurrence of extra systoles very commonly causes rhythmical irregularities of all descriptions, and especially intermission.

The myogenic theory of the heart-beat assumes that not only is the heart muscle capable of receiving a stimulus (it is excitable), and of responding to the stimulus (contractile), but that it is able to transmit the stimulus and the contraction onwards to its

furthest contractile regions (conductile). The transmission of energy from the auricles to the ventricles, via the muscle fibres, is rendered possible by the presence of a bundle of muscle fibres, described by His junior; it passes from the interauricular septum, through the fibrous auriculo-ventricular junction, to the inter-ventricular septum. It is believed that, in certain diseased conditions, the passage of contractions along this bundle from the auricles to the ventricles may be impeded or abolished, giving rise to a want of regularity in the ventricular contractions, the condition being known as **heart-block**. The rare combination of symptoms termed Stokes-Adams disease is a well-marked example. Here there is a slow ventricular and radial pulse, with rapid contractions of the auricles, as seen by the frequent pulsations in the veins of the neck. Syncope, convulsions, and other signs of disturbed cerebral circulation are observed.

The following statement by Howell summarizes these views as to the conditions concerned in the heart's contraction:

1. The heart possesses within itself a store of energy-yielding material, such that it may continue to give many hundreds or thousands of contractions after its supply of nutriment has been cut off.

2. Each contraction, whether caused normally or by an artificial stimulus, is maximal, and therefore probably uses up all of the energy-yielding material which is at that moment in an irritable condition—that is to say, in such a condition that it may be acted on by a stimulus.

3. The amount of this material in irritable form is nil during the phase of systole, but increases in amount throughout the period of diastole. We know, for example, that if stimulated just at the beginning of diastole, the heart muscle gives a small contraction, and that the contraction, which may be obtained later by artificial stimulation, increases in extent the farther the diastole has progressed.

4. If the above statements are correct, it follows that the store of energy-yielding material in the heart exists in some non-irritable form, and that during the phase of diastole a portion is converted into an irritable form capable of being acted on by a stimulus.

5. The presence of certain inorganic salts is necessary for this transformation from the non-irritable to the irritable condition.

The myogenic theory of the heart-beat does not exclude the



nervous system from all participation in the act; the inhibitory influence of the vagus and the accelerating effect of sympathetic nerve impulses are important factors in the maintenance of cardiac rhythm.

Regarded from a clinical standpoint, the beat of a healthy heart is seen to recur at strikingly regular intervals. Some defect in the rhythm may at times be noticed without any apparent cardiac lesion to account for it. The intermission of a beat (almost always an extra systole), or an irregularity in the force and time of certain contractions, may be a lifelong occurrence at intervals or constantly in persons who have no other evidence of heart affection. In elderly subjects in particular, and sometimes in children, some irregularity may be noticed which in all probability does not depend on disease, but is a functional disturbance.

Disorder of the rhythm is the result of some interference affecting injuriously those qualities which, as stated above, must be possessed by the heart muscle in order that its contractions may recur at regular intervals—viz., its excitability, its contractility, and its conductivity. In addition, influences brought to bear upon it through its connections with the central nervous system (vagus and sympathetic nerves) play an important part in production of irregularity. Valvular lesions, and particularly those of the mitral valve, probably cause irregularity by their damaging effect on the heart muscle. More definite affections of the myocardium are equally provocative of the symptom—viz., fatty degeneration, atheroma of the coronary arteries, acute dilatation, and the heart failure of pneumonia, typhoid or other acute disease. Various toxic substances in the circulation act chiefly through the nervous system, but in all probability exert their deleterious effects directly on the heart muscle as well. Of these may be mentioned tobacco, alcohol, tea, belladonna, digitalis, aconite, and the toxins of bacterial infections. The influence of the nervous system is more distinctly seen in the irregularity and palpitation occurring in emotional and neurotic states, in the disturbance of rhythm due to digestive derangements (partly reflex and partly due to pressure of distended stomach and bowels), and in the effects of intracranial lesions. The condition of extreme irregularity with palpitation is termed *delirium cordis*.

The various types of irregularity to be observed have received much attention from investigators, but the clinical significance of the different forms is not well understood. Careful employment

of the sphygmograph (see Pulse, p. 301) has been repaid by the acquisition of much important information.

The following varieties of arrhythmia may be distinguished :

**Intermittent Pulse.**—At intervals a beat is apparently missed at the peripheral arteries. The beat preceding the pause may be noticed to be double, or it may only be possible to recognize the ineffectual extra systole which usually causes this irregularity by auscultating the heart. It is sometimes termed **pulsus intercidens**. This is often produced in hearts which have no other sign of disease, and may be transient and without pathological significance, but is commonly a sign of failing compensation.

**Bigeminal Pulse and Trigeminal Pulse.**—The occurrence of two or three beats followed by a pause, probably the result of an extra systole occurring at regular intervals.

The above forms are often said to be partially irregular, in distinction from those completely irregular instances which are without any element of regularity.

The want of regularity in the rhythm is usually associated with irregularity in the force and volume of the pulse-wave. In this latter respect one observes several types, viz. :

**Pulsus alternans**, the alternate occurrence of a large and a small pulse-wave.

**Pulsus paradoxus**, a weakening of the pulse-beats during inspiration. This may occur in cases of pericarditis, especially when inflammatory adhesions and infiltration have involved the mediastinum.

For further particulars see the article on the Pulse (p. 296).

### ARTERIES, Examination of.

The subject of the arterial pulse is considered in a separate article (p. 296). Here some points of diagnostic interest in the arterial system generally may be referred to.

Excessive pulsation of the arteries, best seen in the vessels of the neck, may be merely the evidence of excited circulation, due to emotions, hysteria, or excessive exercise. It is often seen in exophthalmic goitre, but is most typically seen in cases of aortic incompetence. Any condition giving rise to the large or bounding pulse will cause visible pulsation of the arteries.

There are two conditions of excessive pulsation of arteries which frequently suggest an erroneous diagnosis of aneurism—viz., epigastric pulsation and episternal pulsation. A very common cause for the former is the irritable aorta, observed in

cases of dyspepsia or debility among neurotic subjects. (See Abdomen, Movements, etc., p. 10.) Pulsation in the episternal notch is likewise a symptom in many instances of functional irritability of the innominate artery. It is observed also in anæmic and in elderly subjects, without other evidence of disease in the heart. It is seen in conditions of excessive pulsation of the arteries generally, referred to above, and more rarely may be due to aneurism of the innominate artery or of the transverse arch of the aorta.

Valuable information as to the condition of the walls of the arteries may be obtained by radiography (see p. 574).

Auscultation of the larger arteries is a means of acquiring diagnostic information in some cases.

The bell of the stethoscope must be laid over the artery with as little pressure as suffices to exclude external sounds. In the normal carotid and subclavian the two sounds of the heart can then usually be heard. A similar method of examining the femoral artery only elicits the systolic sound, or none at all. The stethoscope is now pressed more firmly over the vessel, so as to constrict it, and a loud, harsh, systolic murmur is heard, over not only the subclavian, carotid, and femoral arteries, but also over the brachial, and even possibly over the radial. In the case of the subclavian artery one often hears in persons apparently healthy a systolic murmur, even when the greatest care is taken to avoid pressing on the vessel. This is sometimes due to slight muscular pressure, owing to the position in which the arm happens to be placed. It may also be produced by the pressure of pleuritic adhesions or of unsuspected consolidation of the apex of the lung.

The systolic and diastolic murmurs of aortic disease are heard in the carotids, and also in the subclavians as a rule. Disappearance of the second sound over the carotid is sometimes the only sign it furnishes of aortic incompetence.

In cases of aortic regurgitation one may at times, if the pulse is excessively collapsing, hear both the systolic and the diastolic sounds (not murmurs) in the femoral arteries. The same sounds have on rare occasions been heard in cases of pregnancy, chronic lead-poisoning, and anæmia.

**Duroziez's Double Murmur.**—This phenomenon is produced by pressure of the stethoscope over the femoral artery in cases of aortic incompetence, and consists in the presence of both systolic and diastolic murmurs, similar in character to those heard over the

sternum in the same affection. In such cases, on gently placing the stethoscope over the femoral artery without pressure, one may hear one or two sounds resembling the normal heart-sounds. A little pressure gives the normal systolic arterial murmur. Now, pressing a little more, one may succeed, after carefully varying the pressure till the exact point is reached, in hearing the systolic murmur, followed by a fainter diastolic bruit. It is this double murmur which is characteristic of aortic regurgitation, and which is known as Duroziez's murmur. The diastolic murmur is caused by actual regurgitation of the blood-stream during diastole, through the constriction in the artery produced by pressure of the stethoscope.

The systolic murmur heard best over the commencement of the pulmonary artery is possibly produced in that artery, but its exact origin is conjectural. It is known as the functional, accidental, or hæmic murmur. It is further considered in the article on the Auscultation of the Thorax (p. 433).

The arteries in connection with the enlarged thyroid gland in cases of exophthalmic goitre often give out a murmur without pressure.

A murmur may be produced in the internal mammary artery during lactation,\* which may simulate that produced at the mitral or tricuspid orifice of the heart in cases of endocardial or myocardial disease.

#### **ASCITES** (Gr. *ἀσκός*, a bag, a wine-skin).

A small quantity of serous fluid is normally present in the peritoneal cavity. When it is excessive in amount, the condition is termed ascites. If the fluid be removed by puncture of the abdominal walls, it is found to be clear, straw-coloured, of a specific gravity of about 1015, and contains about 2 per cent. of albumin. Should the effusion be of inflammatory origin, the specific gravity and the percentage of albumin are somewhat higher than the figures just mentioned.

The signs by which ascites may be recognised are referred to in the articles on the percussion of the abdomen (p. 17), and on the shape, etc., of the abdomen (p. 14). They may be here briefly recapitulated: **Inspection**: If the quantity of fluid be moderate, the abdomen is flattened and bulging at the sides, and the navel is stretched and flat. If there should be a large amount of fluid present, a general enlargement and projection of the

\* See a case recorded by the writer, *Brit. Med. Journ.*, 1911, vol. i., p. 623.

abdomen is observed, and the navel is unduly prominent. **Palpation:** A fluctuation wave may be felt (p. 14). In case tumours are present along with the ascites, they may be detected by 'dipping' (p. 4). **Percussion:** Loss of the normal tympanitic resonance in the dependent regions of the abdomen. The areas of dullness change as the patient changes his posture (see Figs. 4 and 5).

**Causes.**—Peritonitis, chiefly chronic forms. This is most frequently tuberculous. The effusion is often one of the features in a general œdema due to Bright's disease or to uncompensated heart affections; less frequently the anasarca results from anæmia (pernicious anæmia, chlorosis, Banti's disease, etc.), or from cachexia due to new growths or other wasting diseases. One of the commonest causes of ascites is obstruction to the portal return circulation. The obstruction is frequently located in the liver itself (cirrhosis, tumour in the portal fissure), or it may be due to pressure of tumours on the vein, or to thrombosis of the vessel. Obstruction in the course of the inferior vena cava (valvular disease of the heart, adherent pericardium, mediastinal tumours or aneurism) may also be responsible.

Ovarian cysts, hydronephrosis, pregnancy, or hydatids of the liver may be mistaken for ascites. The position of the area of dullness in the flanks, with a clear area round the umbilicus (the patient being in the recumbent dorsal position), the association of symptoms of one of the affections mentioned above, and the character of the fluid withdrawn, are usually sufficient to make the diagnosis clear.

Instead of the usual ascitic fluid, one finds at times blood-stained liquid (in cancer, in tuberculosis, and sometimes in cirrhosis) or milky fluid (chylous ascites). The latter condition may be due to obstruction or perforation of the thoracic duct (as in the remarkable case recorded by Whitt—*Brit. Med. Journ.*, 1885, vol. i., p. 1089—where occlusion of the thoracic duct occurring in the course of tuberculous peritonitis caused dilatation and rupture of the duct in its lower third); to filariasis; and in slightly marked cases with mere turbidity of the fluid, to a milk diet.

### ASPECT (Facies).

The aspect of a patient and the expression on his face may at times be of assistance in arriving at a diagnosis. The following types of **facies** may be borne in mind:

(a) **A dull and apathetic aspect** is characteristic of several conditions. When combined with a flushed, heavy appearance, a raised temperature, and, in severe cases, a low delirium, with sordes-covered gums and dry tongue, the aspect is significant of well-advanced typhoid fever. A somewhat similar aspect is observed in the typhoid state from any cause. A very different type of dull, apathetic facies is that of paralysis agitans, and is known as **Parkinson's mask** (the disease being often styled Parkinson's disease). Here the face is inscrutable and mask-like in its want of expression (see Fig. 47), but there is an absence of the signs of severe constitutional disturbance seen in the form just described. A third dull and heavy type of face may be mentioned, viz., that of mouth-breathers. The presence of nasal or naso-pharyngeal obstruction gives the child a dull, stupid appearance—the mouth remains open, the upper eyelids droop somewhat, the child is listless and wanting in the keenness of youth. In myxœdema a dull, unemotional aspect is found (see Fig. 7).

(b) **An alert and active aspect** is common to a variety of very different conditions. Pneumonia is an example: the restless, wakeful, flushed condition during the height of the fever is striking; a certain pained and anxious expression accompanies the animation of this aspect. Phthisical subjects in an advanced stage of the disease exhibit a bright but careworn and pathetic expression. In the early stage of typhoid fever, and in most feverish conditions which have not advanced to the point of prostration, the eyes are bright and the expression is animated.

(c) **An anxious aspect** is constantly seen in acute inflammatory affections generally, and especially in those of the abdominal organs. Dyspnoea from any cause gives rise to an anxious or agonized expression, which, combined with stridor, excessive respiratory efforts, and lividity or cyanosis, make up a striking picture.

(d) **A prematurely old, wrinkled, and elfish face** is seen in children the victims of congenital syphilis.

(e) **The nervous, self-conscious, and often affected aspect** of the neurotic or hysterical patient is characteristic.

(f) The expression produced by such conditions as facial paralysis, ptosis, exophthalmos, or mumps do not need further mention.

(g) **The sunken features, deeply-lined face, sharp nose, eyes**

deep in their sockets, pale, livid, or cyanosed skin, known as the **Hippocratic facies**, is seen in moribund cases, and in serious, but not necessarily moribund, affections of the abdominal organs, especially in intestinal obstruction, peritonitis, and cholera.



FIG. 7.—MYXŒDEMA.

The dull, apathetic aspect, swollen eyelids, thinned hair, and 'spade hands' are well shown.

**ASPHYXIA** (Gr. *ἀ*, privative; *σφύξις*, the pulse).

The condition produced in an air-breathing animal by depriving it of oxygen. Three stages may be recognized :

1. **Exaggerated Breathing.** — The efforts to respire are

laboured and excessive, the muscles of extraordinary inspiration and expiration being brought into use (**hyperpnœa**); the face becomes cyanosed, the expression anxious, the eyes prominent, the respiration noisy (**dyspnœa**, *q.v.*, p. 129). This stage lasts one minute, or longer if the air privation be incomplete, and is due to stimulation of the respiratory centre by blood becoming each moment more venous.

2. **Convulsive Stage.**—The violent breathing becomes convulsive and develops into general convulsions; this stage is shorter than the first.

3. **Exhaustion.**—The respirations grow much weaker; they occur at longer intervals and finally cease. The pulse has grown feebler, but the heart may continue to beat after respirations cease; it is obvious, therefore, that the term **asphyxia** is singularly inappropriate to this condition. This stage may last three minutes or longer, during which time the muscles have grown flaccid, the conjunctivæ insensitive, the pupils dilated, and general insensibility has supervened.

This clinical picture is not often seen in medical diseases; it may be observed in a modified form in laryngeal diphtheria, in asthma, in emphysema, in laryngismus stridulus, in œdema of the glottis, and in coal-gas poisoning. In these affections the symptoms differ considerably from the typical asphyxia, owing to the varied amount of air reaching the lungs, and to the presence of toxæmia produced by the diseases. The most familiar cause of this catastrophe is impaction of a foreign body (food, artificial teeth, etc.) in the pharynx or larynx.

A condition of venous stasis in the fingers, toes, ears, or nose occurs as one of the stages of **Raynaud's disease** ('symmetrical gangrene of the extremities'). This cyanosis was termed **local asphyxia** by Raynaud, and is due to a spasmodic contraction of the walls of the arteries, causing, in the first place, a white or dead appearance in the part affected (**local syncope**, 'dead fingers'). or if the condition goes a stage further it becomes local asphyxia; a still more severe degree may be found—viz., **symmetrical gangrene**.

### ATAXIC GAIT (Stamping Gait).

The imperfectly controlled movements of the limbs in walking, characteristic of muscular inco-ordination, and seen typically in locomotor ataxia. The subject is further considered in the article on Gait (p. 150).



**ATHETOSIS** (Gr. *ἄθετος*, without fixed position).

A condition, generally found in young persons, of involuntary choreiform and rhythmic movements of the limbs, and sometimes of the head and facial muscles; it is often active during sleep. The condition occurs as a sequence to hemiplegia and the birth palsies, and is really a symptom of these affections, though it is sometimes described as a disease. It is due to continuous stimulation of the cortex or basal ganglia in certain rare residual conditions of the cerebral lesion, the motor impulses finding their way through or round the obstructed motor path to the periphery.

**ATROPHY** (Gr. *ἀ*, privative; *τροφή*, nourishment).

Diminution in the bulk of organs or tissues is the result of—  
 (a) emaciation from excessive destructive metabolism, and from insufficient or unsuitable nutriment; (b) disuse of the parts;  
 (c) changes occurring in tissues after inflammation or injury;  
 (d) muscular wasting consequent on disease of the nervous system (as seen in the various atrophic forms of paralysis);  
 (e) diseases of the muscles (progressive muscular dystrophy).

The subject is further considered in the article on Trophic Disturbances (p. 487).

**AURA** (Gr. *αἶρα*, air in motion).

The term is given to the warning experienced by about 50 per cent. of epileptics immediately before the onset of the convulsive attack. This may take the form of some disturbance of sensation, movement, secretion, etc., by which they become aware of an approaching fit. A common signal is the feeling of a breath of air blowing upon some part of the body, most frequently on the hand or arm; hence the name **aura**. Other sensory phenomena are numbness, tingling, prickling, which may be felt anywhere, but most frequently in one hand or arm. Of motor symptoms, twitching or cramp in the facial muscles is frequently observed, and may occur in other parts of the body. Hallucinations affecting the special senses, and particularly that of sight, also those of hearing, smell, and taste, may be experienced. Vaso-motor disturbances may occur, such as perspiration, waves of heat or cold, and palpitation. Psychological disturbances sometimes form the warning, as, for example, a vague sense of fear or dread, and uncontrollable impulses to perform some involuntary act.

**AUSCULTATION SOUNDS : General Principles.**

The use of the term 'auscultation' is clinically restricted to the examination of the body by means of the stethoscope. It is possible to observe many of the phenomena of auscultation by simply laying the ear upon the surface of the patient's body, but that procedure offers no advantages over the use of one of the many forms of stethoscope; in some respects it is objectionable, and can only be recommended when an instrument is not available.

The choice of the instrument need not detain us, as it is largely a matter of individual taste and experience; but it may be stated that the simple binaural stethoscope, without any intensifying or resonating chamber, will be found most generally suitable. Occasionally an instrument with two chest-pieces is of assistance in timing or discriminating heart murmurs.

Care must be taken when placing the bell of the stethoscope upon the body that it presses evenly and firmly but lightly upon the surface; air must be excluded by this means, as otherwise the sounds will be obscured. Movements of the chest-piece upon the skin must be avoided, and the serious inconvenience caused by a hairy surface can be mitigated by anointing the surface with vaseline or other lubricant.

On listening over the thorax, the abdomen, and the larger bloodvessels of the body and limbs, certain sounds are to be heard, which may be modified by a variety of conditions, both in health and in disease, and sounds which do not occur in the healthy body may be perceived when the tissues and organs undergo morbid changes. In studying these phenomena at the bedside, we must endeavour to discover (*a*) by what means the sound is produced, and (*b*) by what means it is conducted to our ear.

Audible vibrations occur in the body from various causes. They may be the result of the rubbing of surfaces upon each other, an abnormal occurrence, or the vibration of elastic structures in the blood stream. They may be due to the production of a **fluid vein**. By this term is understood the passage of a fluid from one chamber into another through an aperture whose sectional area is either greater or less than that of the chamber into which the fluid passes. Should the current be sufficiently rapid, vibrations are generated in the fluid which are transmitted to the wall of the chamber, and under favourable circumstances may reach the ear of the observer (see p. 433).

By the production of this physical phenomenon many of the sounds resulting from the movements of both gases and liquids can be accounted for—*e.g.*, the sound heard over the larynx or trachea during respiration; possibly the vesicular breath-sounds heard over the large masses of spongy lung tissue; the sounds produced by movements of gas in the stomach and intestines; the murmur elicited by pressure of the stethoscope upon any large artery; the sound heard in aneurisms; the murmurs produced by the blood passing through damaged cardiac orifices, etc.

Sounds may also be the result of gas passing through liquids of different consistencies and densities, giving rise to an infinite variety of gurglings, bubblings, crepitations, or râles. The passage of liquids or gases through normal or abnormal channels causes vibrations in the different elastic structures met with. By this means are produced the voice-sounds, many of the so-called dry râles or rhonchi, the first sound of the heart (to a large extent), some of the murmurs due to cardiac lesions, etc. The concussion of solid structures may elicit audible vibrations: the second sound of the heart is a possible example. The 'muscular sound' is due to tetanic contractions of muscle fibres, or, in the case of the heart, to a series of single contractions of the fibres. In addition, the various sounds thus originated may be modified by the presence of resounding cavities or media of different degrees of elasticity, producing overtones and harmonics, and altering considerably the character of the sounds.

The sounds thus produced will only be audible if the vibrations are taken up by substances or tissues which form fairly good conductors of sound. The most efficient conductor to be found in the body is an unbroken column of air, such as is normally found in the trachea or bronchi, and abnormally in pulmonary cavities and in the pleural or peritoneal sacs. If the tissues intervening between the source of the sound and the surface are composed of a fairly homogeneous structure, such as bone, solid organs like the liver, or tissues rendered more homogeneous by consolidation from inflammatory infiltration, then the vibrations producing the sound are more readily conducted than through a heterogeneous structure, and so are more distinctly heard. While the larger air channels of the lung are excellent conductors, the spongy lung tissue, composed of a mass of small collections of air, separated from each other by cellular divisions of animal membrane,

conducts vibrations imperfectly. Water is a fair conductor, though a bad resounding medium; sounds may pass through it without great difficulty, but instead of amplifying or intensifying sounds produced in its neighbourhood, it dulls and weakens them. A thick layer of fat is also a bad conductor of sound, and the same may be said, though to a less degree, of muscle.

The article on Auscultation of the Thorax, at p. 407 *et seq.*, deals with the subject in detail.

### AUSCULTATORY PERCUSSION-SOUNDS.

A combination of auscultation with percussion gives in some instances useful results. Its use in producing the bell sound is referred to at p. 56, and it is considered by some observers to be a trustworthy means of delimiting the borders of both solid and air-containing organs or tumours. While differences of opinion as to its efficacy exist, it should be practised in all suitable cases.

The bell of the stethoscope is placed over the organ and well inside its limits. Whilst listening through the stethoscope immediate percussion is performed with one finger of the disengaged hand by short steps along a series of radiating lines whose centre is the stethoscope. As a rule, it is better to percuss from a point in the periphery well outside the limits of the organ, and progress inward toward the centre. The strokes of the finger should be fairly strong, and as they approach the stethoscope the sound they evoke grows louder. On reaching the margin of the organ over which the stethoscope rests, a sudden increase in intensity and often a metallic quality in the sound is observed, and the spot is marked on the skin. This having been done along a number of radii, the marked points are joined by a line, which, according to the advocates of the method, describes fairly accurately the boundaries of the organ.

It is especially in the examination of the stomach that this method is employed, but it is also found useful in other conditions, chiefly abdominal. The clearness and ringing quality of the percussion-note depend upon continuity of tissue between the structure lying below the spot struck and that under the stethoscope. It may, therefore, be possible to decide by this means if a tumour forms part of the organ over which one is listening, or whether, on the contrary, it is a separate structure: Figures 8 to 16 explain diagrammatically the results to be sought.

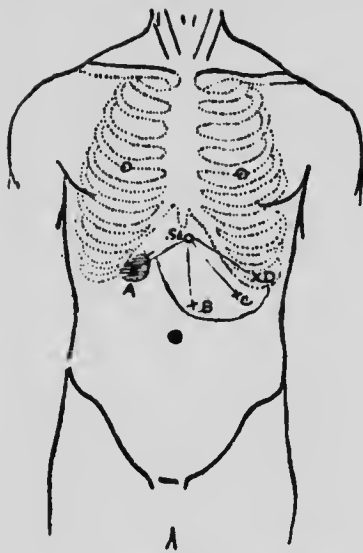


FIG. 8.—TUMOUR NEAR THE PYLORUS.

The chest-piece of stethoscope being placed at St, sounds elicited by percussion on the points B, C, and D are heard more distinctly than that produced by percussing over the tumour at point A.

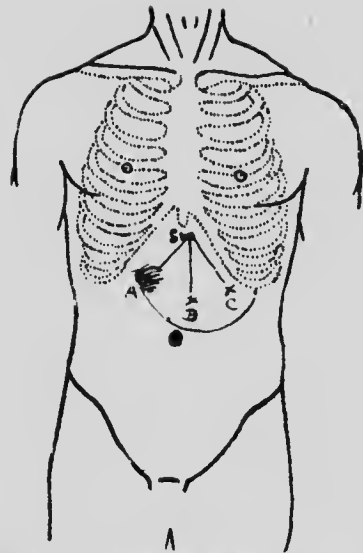


FIG. 9.—TUMOUR OF THE PYLORUS.

The sounds elicited by percussing over the points A, B, and C are equally distinct.

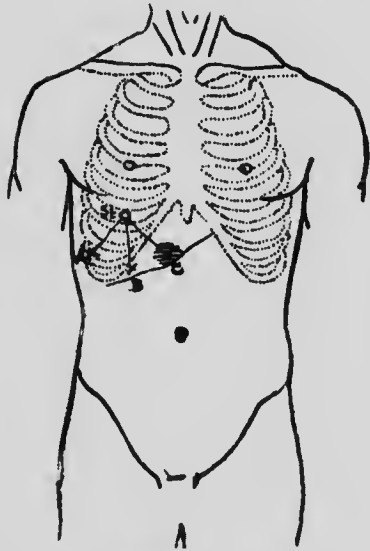


FIG. 10.—TUMOUR OF THE LIVER.

The sounds produced at A, B, and C are equally distinct.

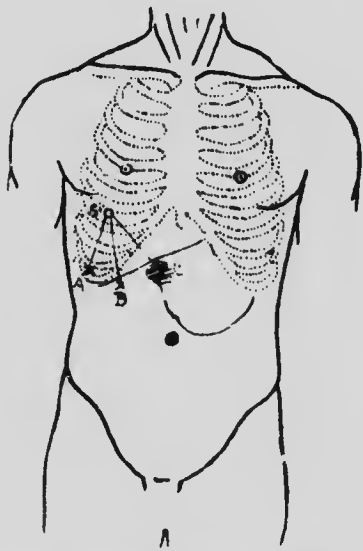


FIG. 11.—TUMOUR OF THE PYLORUS.

The sound produced at A (over the liver) is more distinct than those produced at B and C (off the liver).

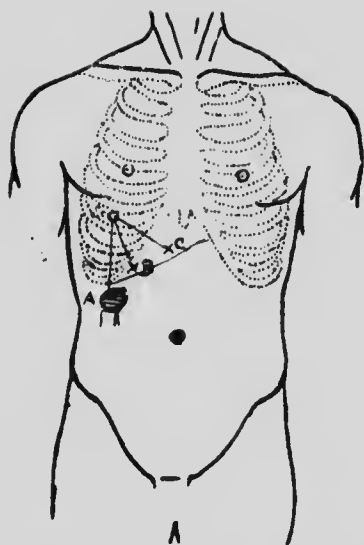


FIG. 12.—TUMOUR OF THE BOWEL.  
The sound produced at A (over the tumour) is less distinct than those at B and C (over the liver).

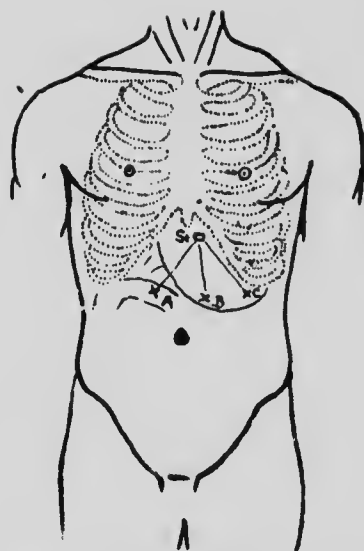


FIG. 13.—TO DISTINGUISH STOMACH FROM BOWEL.  
The sound produced at A (over the bowel) is less distinct than those at B and C (over the stomach).

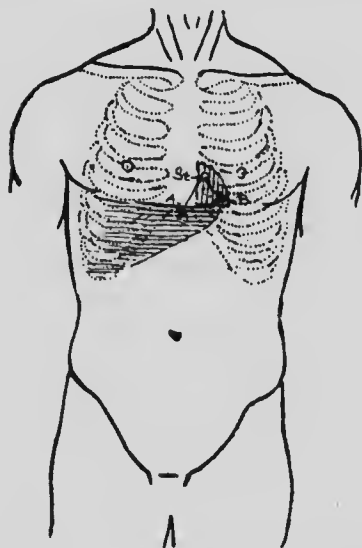


FIG. 14.—TO DISTINGUISH HEART DULNESS FROM LIVER DULNESS.  
With the stethoscope (St) over the heart, the sound at A (over the liver) is less distinct than that at B (over the heart).

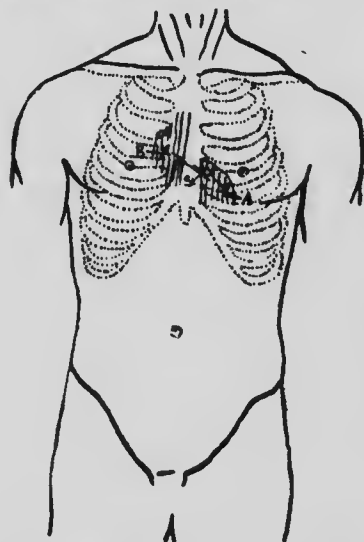


FIG. 15.—ANEURISM OR OTHER MEDIASTINAL TUMOUR.  
The sound at A (over the heart) is more distinct than that at B (over the tumour).

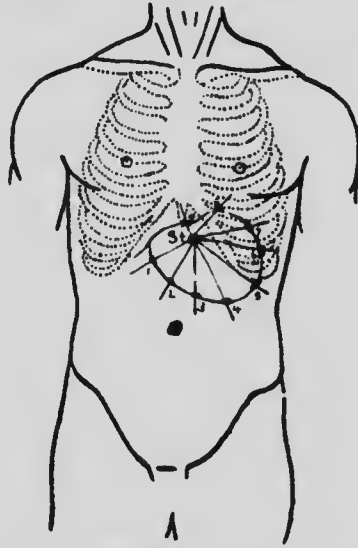


FIG. 16.—TO MAP OUT THE STOMACH.

With the stethoscope on point *St* percuss along a series of radiating lines from that point as a centre. The spot on these lines at which the percussion-sounds suddenly grow weaker is marked. The marked spots (1, 2, 3, 4, 5, 6, 7, 8, 9) are joined by a line which represents the boundary of the abdominal wall in contact with the stomach.

### AXILLARY REGIONS.

That portion of the side of the thorax on each side, bounded before and behind by the axillary folds, and below by a line drawn transversely at the level of the third costal cartilages. The abnormalities to be observed in this region are referred to in the articles on the Shape, etc., of the Thorax (p. 464), Percussion (p. 450), Auscultation (p. 407), and Pain (p. 268).

### BABINSKI'S SIGN.

In conditions of increased reflex action, and especially in lateral sclerosis, the following reflex may occur: the sole of the foot towards its inner side is irritated by pricking or gently scratching it with a moderately sharp instrument. The result is flexion of the great toe on to the dorsum of the foot, and flexion of the remaining toes towards the sole of the foot. (In normal cases the 'plantar reflex' causes flexion of all the toes towards the sole.)

### BACCELLI'S SIGN.

It has been pointed out by Baccelli that the whispered voice cannot be heard through purulent pleural effusion, though it may be distinguished through serous effusion.

**BACTERIURIA.**

The presence of bacteria diffused through a specimen of urine imparts to it a turbidity, and, on moving the fluid, a curious opalescent wavy movement of the cloud in the urine is seen. The opacity differs from that produced by urates, phosphates, pus, etc., in showing no tendency to settle in a few hours.

For the methods best suited to the clinical investigation of micro-organisms in the urine, see the article on Urine Examination (p. 528). The diagnostic significance of the observed bacteria is considered in the article on Urinary Abnormalities (p. 512).

**BARREL CHEST (Emphysematous Chest).**

The thorax seems to be in a condition of full inspiration—it is more capacious and more barrel-shaped than normal. This form of chest is due to long-continued coughing and dyspnoea, and is most frequently seen in chronic bronchitis and emphysema, hence its name. (See Thorax, Shape, etc., p. 468.)

**BEDSORES.**

If the nursing of a bedridden patient be efficiently carried out, he should never suffer from sores produced by pressure, unless his nervous system is injured in such a way as to interfere with the nutrition of the skin and subcutaneous tissues. (See Trophic Disturbances, p. 493.) On finding broken skin or sloughing tissues over the bony prominences which are exposed to pressure in a bedridden patient, one may safely assume that he is either the victim of bad nursing or he is a subject of a disease of the nervous system causing trophic changes in the tissues.

**BELL SOUND (Anvil Sound, Coin Sound, Bruit d'airain).**

A sound produced by placing the stethoscope over a large cavity containing air, while an assistant, using two coins as plessor and pleximeter, percusses on another spot overlying the same air cavity. A clear, ringing, bell-like sound is heard through the stethoscope. If the same process is carried out in the absence of a large air cavity, the sound has only a clinking, metallic quality, quite wanting in the ringing, musical character.

This symptom is almost pathognomonic of pneumothorax, but is at times heard over a large pulmonary cavity, or over the distended stomach.



**BELL'S PHENOMENON.**

In cases of facial paralysis (Bell's paralysis), the effort made to close the eye on the affected side results in the eye being rotated upward and outward under the unlowered lid.

**BENEDIKT'S SYNDROME.**

An oculo-motor paralysis of one side, with loss of power and tremor or ataxy of the opposite limbs. This may result from a lesion of the crus involving the red nucleus on the side of the third nerve paralysis.

**BIAL'S TEST (for Pentose in Urine).** See Urine Examination, p. 537.

**BIERMER'S SIGN.**

When pneumothorax is present, combined with fluid in the pleural cavity, the sound evoked by percussion over the cavity alters in pitch (in many cases) as the patient alters his position.

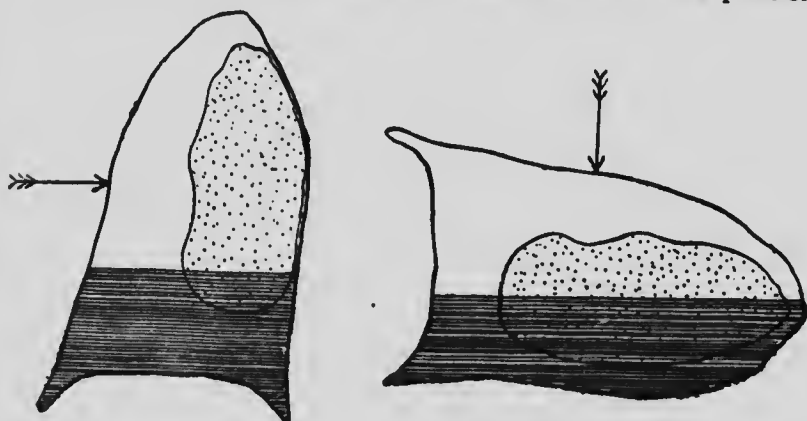


FIG. 17.—BIERMER'S SIGN.

Air and fluid in the pleural cavity. Change of posture, by altering the shape of the air cavity, causes an alteration in pitch of the percussion sound. The shaded area represents fluid, the dotted figure lung.

This, like the similar result of percussing over a pulmonary cavity (Gerhardt's Phenomenon, p. 152), is due to an alteration in the shape and size of the cavity, produced by movement of the contained fluid (see Fig. 17).

**BIERNACKI'S SIGN.**

The customary sensitiveness of the ulnar nerve behind the internal epicondyle of the humerus is absent in many cases of locomotor ataxia.

**BIOT'S RESPIRATION (Meningeal Respiration).**

In certain intracranial diseases, and especially in meningitis, also occasionally in other grave affections, the respirations cease periodically for an interval of a few seconds, or perhaps half a minute. (See *Dyspnœa*, p. 130.)

**BIURET REACTION (for Proteids).** See *Urine Examination*, p. 532.

**BLADDER REFLEX.**

The functions of the urinary bladder are performed by means of a series of reflexes, which are fully discussed in the article on *Micturition* (p. 132).

**BLOOD EXAMINATION.**

- I. The examination of the corpuscular elements of the blood :
  - (a) Enumeration of the red cells—Methods to be employed.
  - (b) Enumeration of the white cells—Methods.
  - (c) The hæmoglobin value of the blood (the hæmoglobin being, as a rule, entirely confined within the red cells).
  - (d) The colour index.
  - (e) The preparation and staining of blood films—Information obtained from the study of stained films. A. Red cells: poikilocytes, megalocytes, microcytes, polychromatophilia, basophile granulation, nucleated red cells. B. White cells: normal leucocytes, granular and non-granular; pathological leucocytes; differential count of normal blood; origin of the leucocytes; leucocytosis. C. Blood platelets. D. Blood parasites: plasmodium malarix, spirilla of relapsing fever, filarix, trypanosomata.
- II. The examination of the blood serum—Methods and instruments :
  - (a) Alkalinity of the serum.
  - (b) Saline concentration—Test of renal efficiency.
  - (c) Opsonic power.
  - (d) Agglutinating power of the serum—Widal's reaction.
  - (e) Wassermann's reaction.
  - (f) Appearance of the serum.
- III. Further methods of examining the blood :
  - (a) Specific gravity.
  - (b) Determination of the total volume of the blood by the method of Haldane and Lorrain Smith.
  - (c) The coagulability of the blood—Wright's method.
  - (d) Determination of the amount of calcium salts in the blood
  - (e) The viscosity of the blood.
  - (f) The isolation of micro-organisms from the blood.

The study of the blood frequently furnishes us with valuable help in diagnosis, and is now an essential part of clinical medicine.

Within recent years much has been added to our knowledge in this department of medicine, and many new methods have been devised which demand the attention of the physician.

The impetus given by Ehrlich to the study of the corpuscular elements of the blood has called forth an enormous number of observations on the variations in red and white corpuscles which occur in disease. From such work much that is useful in diagnosis has evolved.

The work of Haldane and Lorrain Smith has made it possible to measure approximately the total volume of the blood by the CO method, and their results have already thrown important light on the clinical features and pathology of the anæmias.

By the use of most ingenious methods A. E. Wright and his pupils have demonstrated that the 'opsonic' power of the blood serum may be accurately determined, and by their researches the whole subject of the treatment and diagnosis of the microbic infections has been placed upon an entirely new basis.

The researches of Bordet and Gengou have supplied a new method, known as the *fixation of complement*, for the diagnosis of infection.

The discovery and identification of the various blood parasites has literally revolutionized the diagnosis and study of tropical diseases.

Methods have also been devised for studying such properties of the blood as its viscosity, alkalinity, coagulability, etc., and such methods seem certain to largely increase the usefulness and scope of blood examinations.

It should be remembered, however, that some of the methods referred to require considerable manipulative power, and that even with most careful workers the results are more or less approximate and liable to error. Blood findings should, therefore, always be considered in conjunction with the other clinical features and symptoms of the disease in question.

The object of this article is to indicate how the blood should be obtained and examined. Only one method for each of the details will, as a rule, be described. The selection of the methods is not intended in any way as a criticism of other methods, which will be found described in the various text-books and papers on hæmatology. The method described is merely recommended as a valuable one for furnishing the required information, and as one that has proved useful in practical work of this kind. It must

also be borne in mind that the majority of these manipulations only require a little practice for their successful accomplishment. The examination is directed to the investigation of the condition of the corpuscular elements and of the serum; accompanying constituents and characteristics of the blood are at the same time to be studied.

## I. The Examination of the Corpuscular Elements of the Blood.

### 1. Enumeration of the Red Cells.

In health the cubic millimetre of blood contains from 4,500,000 to 6,000,000 red cells. Slight variations in the number of red cells per unit of volume occur as the result of physiological conditions—such as the time of the day, digestion, menstruation, pregnancy. A convenient rule is to regard 5,000,000 per cubic millimetre as the normal numbers, and to make our calculations from this figure. The instrument now generally in use for counting the red cells is that of Thoma, and may be bought from any instrument dealer. The apparatus consists of a mixing pipette and a counting chamber.

1. The pipette is a capillary tube graduated in ten equal divisions; above the pipette is a bulb, which has a capacity 100 times that of the pipette. To the pipette is attached a piece of rubber tubing with a mouthpiece. Thus when the tube is filled with blood up to the mark 1, and then is mixed with the diluting fluid drawn up to the mark 101, a specimen of blood is obtained in the dilution of 1 in 100. In cases of plethora a greater dilution is more convenient for counting, and the blood may in such cases be drawn up to the mark 0.5, thus giving a dilution of 1 in 200. The bulb contains a glass ball, to facilitate the mixing of the blood and diluting fluid.

2. The counting chamber consists of a slide upon which a glass frame with a circular opening is fixed. In the centre of this opening is a disc of glass, the surface of which is ruled in small and large squares. The frame extends exactly 0.1 millimetre above the surface of the glass disc, so that when a glass cover-slip is placed on the frame, the interval intervening between the ruled disc and the cover-slip is exactly 0.1 millimetre deep. Diagram 1 (see Fig. 18) explains the ruling of the disc of glass. The side of each small square is  $\frac{1}{20}$  millimetre. Thus the corpuscles seen on each square are deposited from a volume of fluid equal to  $\frac{1}{20} \times \frac{1}{20} \times \frac{1}{10}$ , or  $\frac{1}{4000}$  cubic millimetre.

3. Diluting fluids. Various diluting fluids may be used, and must be of such a concentration that they do not dissolve or alter the red corpuscles.

Toisson's mixture is a good one :

Sodium sulphate	..	..	..	..	8 grammes
Sodium chloride	..	..	..	..	1 ..
Glycerin pur.	..	..	..	..	30 ..
Aqua distillata	..	..	..	..	160 ..
Methyl viol.	..	..	..	..	0.025 gramme

The red blood-corpuscles are counted in the following manner: A drop of blood obtained by a puncture of the finger or the ear is drawn into the pipette to the mark 0.5 or 1. The excess of blood is removed by the finger, and the diluting fluid is then drawn up to the mark 101. No air must enter the pipette during this operation. The blood and diluting fluid are now thoroughly mixed by shaking, and after rejecting one or two drops, a drop is placed on the ruled glass disc. The size of the drop required to just cover the disc when the cover-glass is applied is easily learned by a little practice. When the cover-glass is applied it should be in close contact with the frame, and if Newton's rings are seen, this is the case. After allowing a short interval to elapse so that the corpuscles may have time to sink on to the ruled squares, the counting chamber is placed under the microscope. The corpuscles in 40 of the small squares are now counted, and it is best to adopt the convention of counting all the cells on or touched by the rulings forming the bottom and right side of the squares when proceeding from left to right, and those touched by the bottom and left rulings of the squares when proceeding from right to left (see diagram). The number of cells on 40 squares having thus been determined, the counting chamber is washed and dried, and a second drop is counted in an exactly similar way. If the results closely correspond, the mean of the results obtained may be taken as correct; otherwise another drop should be taken and a mean of the three results taken. If the red corpuscles are found to be massed together in clumps, the result will be very inaccurate, and

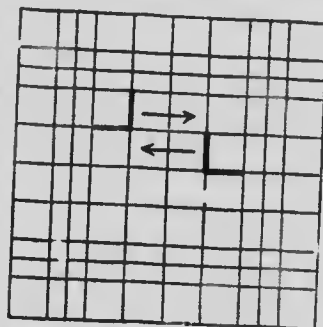


FIG. 18.—METHOD OF COUNTING RED CELLS.

the blood should be taken again and diluted as quickly as possible. Having obtained the number of red cells in 40 squares, the calculation is very simple.

Thus  $40 \times \frac{1}{20} \times \frac{1}{20} \times \frac{1}{10}$  cubic millimetres of diluted blood is found to contain  $x$  corpuscles. If the blood has been diluted 100 times then  $40 \times \frac{1}{20} \times \frac{1}{20} \times \frac{1}{10}$  cubic millimetres of undiluted blood will contain  $100x$ ; therefore  $\frac{1}{1000} \times 40$  or  $\frac{1}{100}$  cubic millimetres of undiluted blood will contain  $100x$  corpuscles. Therefore 1 cubic millimetre of undiluted blood will contain  $10,000x$  corpuscles.

In other words, if the number obtained by counting 40 squares be multiplied by 10,000, the result obtained is the number of corpuscles per cubic millimetre of the blood examined.

The determination of the number of red cells per cubic millimetre furnishes useful diagnostic information in all anæmic and polycythæmic conditions.

## 2. Enumeration of the White Cells.

The normal number of leucocytes per cubic millimetre of blood is from 6,000 to 8,000. Considerable variations occur in health from physiological causes, such as have been mentioned when considering the normal variations in the red cells. The white cells are subject to greater variations in number than the red cells under such conditions. The common sources of error are best avoided by taking specimens about four hours after a meal, and at the same hour each day. It is usual in counting the white cells to use the second pipette of the Thoma Zeiss apparatus, and to make use of a diluting fluid, which lyses or dissolves the red cells, so that the white cells alone are visible. The pipette in question has a similar construction to that already described for counting the red cells, but gives a dilution of 1 in 20 or 1 in 10. The diluting fluid generally used is 3 per cent. acetic acid tinged with gentian violet. A good plan for counting the white cells is to estimate the area (in small squares) of the microscopic field of the optical combination of the microscope generally used for this purpose. This is accomplished as follows (see Fig. 19): The ruled squares of the counting chamber are focussed, and the number of sides of the squares (each side =  $\frac{1}{20}$  millimetre) corresponding to the diameter of the field determined. If the diameter of the field does not exactly correspond with a definite number of sides, the draw-tube of the microscope can easily be altered until this occurs. For instance, supposing the diameter of the field is

found to correspond to the sides of 10 squares, then the formula ( $\pi r^2$ ) gives the area of the field—*i.e.*, the area of the field is equal approximately to 80 squares. Thus if the number of leucocytes in five fields of the microscope be counted, the result will be equivalent to the number in 400 squares. Since the dilution is in this case 1 in 10, the result multiplied by 100 will give the number of leucocytes per cubic millimetre. As a rule, several drops should be counted and a mean taken.

Variations in the number of leucocytes present in the unit

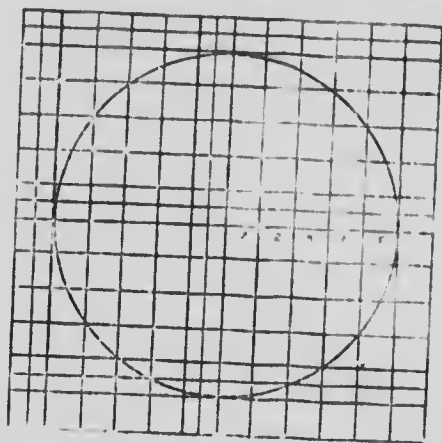


FIG. 19.—METHOD OF COUNTING LEUCOCYTES.

The microscopic field has the diameter of 10 squares; therefore its area  $=\pi r^2$  = about 80 squares.

volume of blood are often of great importance in diagnosis, but as deductions from these variations are not complete without a study of the varieties of leucocytes present, this question is best considered when dealing with the differential count of leucocytes (see below, p. 67).

Wright has shown that capillary pipettes, made by drawing out a piece of glass-tubing, can easily be substituted for the pipettes of the Thoma Zeiss instrument, and that the necessary dilutions can be easily and accurately made with such home-made pipettes. The method of making such dilutions is explained on p. 76.

### 3. Determination of the Hæmoglobin Value of the Blood.

This may be accomplished by the use of the instruments devised by Gowers, Fleischl, Sahli, or Haldane.

The great advantage of Haldane's method is that the standard

colour mixture is composed of blood. Most of the other methods in use have an artificial standard, such as the picro-carmin jelly used in Gowers' instrument, or the coloured glass of Fleischl's instrument. The colour of such a standard varies with the light, and it may be difficult or impossible to make the diluted blood exactly correspond with the standard tint. In Haldane's instrument the standard is a 1 per cent. solution of blood saturated with CO gas and sealed hermetically. The CO gas preserves the blood permanently. The instrument is graduated in such a way that when the tints of the standard solution and the diluted blood correspond, the percentage of hæmoglobin compared with the average normal is at once read off by the graduation on the tube. The blood must be saturated with CO gas, obtained by running in ordinary coal-gas by means of an apparatus supplied with the instrument. This method seems superior to all others, and gives most accurate results. The error is probably not greater than 2 per cent.

The determinations mentioned above should be made as soon as possible after the blood is taken. Several hours make little difference, but if the blood is kept overnight the determinations are always more or less inaccurate.

#### 4. The Colour Index.

By this is meant the relation between the percentage of hæmoglobin and the percentage of red cells. For instance, supposing it is found that in a given blood the number of red cells is 1,500,000 per cubic millimetre, and the hæmoglobin is 40 per cent., then the colour index is  $\frac{40}{30}$ , or 1.3. The normal number of red cells is here considered to be 5,000,000 per cubic millimetre, so that 1,500,000 per cubic millimetre is 30 per cent. of the normal.

This colour index is a point of great value in the differentiation of primary pernicious anæmia from chlorosis and from the various forms of secondary anæmia. In true chlorosis the colour index is always very low, often as low as 0.5. In secondary anæmias it is almost invariably below 1, while the colour index in pernicious anæmia is usually above 1.

This point will be found of great practical value in diagnosis, and seldom leads the observer astray if the determinations are accurately made. A high colour index is the most constant and most characteristic feature of progressive pernicious anæmia (See Anæmia, p. 26.)



### 5. The Preparation and Staining of Blood Films.

The most important part of the examination of the corpuscular elements of the blood is the study of stained films. Films may be spread on slides or cover-slips. Slides possess numerous advantages: they are more easily handled, forceps are not required, they are easily transmitted by post, and usually do not require to be mounted.

The slides must be thoroughly clean and free from grease. It is a good plan to polish them with absolute alcohol before use. The smear on the slide may be made with another slide, with a small triangular area filed off the corner in order to make the smear narrower than the receiving slide, or a piece of cigarette paper may be used for this purpose. A small drop of blood obtained in the usual way from the finger or ear is lightly scraped off with the end of the slide without the corner or with the strip of cigarette paper. This end of the spreader is then applied to the receiving slide, and when the blood has run along the angle between the two slides, the spreader is slowly drawn over the surface of the receiving slide. A film should thus be produced so thin that it dries almost instantaneously. With a little practice good films can be made in this way.

Such films may be wrapped in tissue paper and kept for months. It is best, however, to fix and stain them at once.

*Fixing and Staining.*—Films may be fixed by heat, alcohol, corrosive sublimate, or formalin. The best method to select depends on the stain used, and the full details will not be described here.

It is most convenient for ordinary blood examinations to use a stain such as Leishman's, which fixes and stains the preparation at the same time. The differential staining thus obtained is very good, and serves almost all clinical purposes.

**Leishman's Stain:** This stain is an eosinate of methylene blue dissolved in methyl alcohol. The methyl alcohol not only dissolves the stain, but acts as a fixative. The method is as follows: A few drops of the stain are run on to the films and allowed to remain, covering the film for about one minute. Double the quantity of distilled water is then added. This differentiates the ruby tint of the nuclei from the light blue of the cytoplasm. After four or five minutes' staining the film is thoroughly washed in distilled water and dried with blotting-

paper. The preparation is then ready for examination with an oil immersion lens, or may be mounted in balsam with a coverslip. In a properly stained Leishman's preparation the nuclei are of a deep ruby-red tint.

A stain of this sort serves the majority of purposes required in an ordinary blood examination. It differentiates the varieties of leucocytes, stains the blood platelets, and is one of the best stains for malarial and other blood parasites. For the study of the granulations of the leucocytes it is fairly good, but for this purpose better results are obtained by the use of Ehrlich's triacid stain.

Ehrlich's triacid stain is best obtained ready-made from Grüber. The attempt to prepare this mixture by an amateur is simply to waste time, as the result is usually far from satisfactory. The film for this stain should be fixed by heat. In ordinary work fixation by heat may be obtained with the flame of a Bunsen burner. The slide, specimen side up, is passed slowly through the flame until it is decidedly too hot for the hand to bear. At this temperature, which probably varies between  $110^{\circ}$  and  $150^{\circ}$  C., fixation is complete in from one to two minutes. In staining with the triacid mixture it is only necessary to flood the fixed film with stain for two or three minutes and hastily wash with water.

#### Information obtained from the Study of Stained Films.

A. **Red Cells.**—The normal red blood-corpuscles are biconcave discs without a nucleus. They are composed of a stroma containing hæmoglobin. Their average diameter is  $7\mu$  to  $8\mu$ . With Leishman's stain normal erythrocytes are stained a pinkish red, with Ehrlich's triacid stain an orange colour if the specimens have been properly fixed. In fresh unstained specimens they appear distinctly yellow under the microscope.

1. **Poikilocytes:** This term is applied to red cells of abnormal shape. They are not circular, but may be pear, spindle, dumb-bell, or kidney shaped. They appear in the blood in all severe anæmic conditions, but are specially common in pernicious anæmia. Very little diagnostic importance can be ascribed to such changes in form.

2. **Megalo- and Microcytes:** Changes in the size of the red cells are very important from a diagnostic point of view. In pernicious anæmia a large number of the red cells are larger than normal—*i.e.*, are megalocytes. In a doubtful anæmia, if many of

the red cells are megalocytes, the case is probably one of pernicious anæmia. The increased size can usually be made out without measurement by the experienced observer, but a beginner should measure the size of a number of the red cells with a micrometer scale. Normal red cells vary from  $6\ \mu$  to  $8\ \mu$  in diameter. Very small red cells are called microcytes, and are usually found in blood, together with poikilocytes. They are very frequent in pernicious anæmia.

3. Polychromatophilia: Red cells which have lost to a certain extent their affinity for acid dyes, and have a slight affinity for basic dyes, are termed polychromatophilic. They stain a bluish red or violet with Leishman's stain. They do not appear in normal blood, but are frequently seen in severe anæmias, especially in pernicious anæmia. When a red corpuscle does not stain at all it is termed a 'shadow cell.' Such cells are found in severe anæmias.

4. Basophile Granulation: In severe anæmias red cells may be found containing granules of varying size, which stain well with all basic stains, and are, therefore, blue with all staining methods. They are considered by many to be the remains of nuclei, and appear in severe forms of anæmia, especially the anæmia of lead-poisoning.

5. Nucleated Red Cells: These occur in two forms—(a) Normoblasts are red cells of the normal size, with a deeply staining homogeneous nucleus, usually eccentrically placed. They may occur in any severe anæmia, and are said to be indicative of active blood regeneration. When they occur in very large numbers a blood crisis is said to be present, and this often heralds a marked improvement in the anæmia. (b) Megaloblasts are red cells of a larger size than the normal, and possess a nucleus, which stains more faintly and has more of a reticular structure than that of the normoblast. Various nucleated red cells, transitional between normoblasts and megaloblasts, may be found. Typical megaloblasts are most characteristic of pernicious anæmia. Films in which are found several typical megaloblasts usually come from a case of pernicious anæmia. This rule, however, is not invariable, as megaloblasts may occasionally occur in other severe anæmic conditions.

B. White Cells.—The normal leucocytes of the blood may be divided into granular and non-granular. The granular leucocytes are: (1) polymorpho-nuclear leucocytes; (2) eosinophile leu-

cocytes; and (3) basophile leucocytes. The non-granular are: (1) small lymphocytes; (2) large mononuclear leucocytes; (3) transitional leucocytes.

*Granular Leucocytes.*—1. Polymorpho-nuclear Leucocytes: These are large cells, two or three times as large as the red cells. They are characterized by a polymorphous nucleus, which may take the form of an S, Y, E, or Z, and granular protoplasm. These granules stain red with Leishman's stain, while the nucleus is a deep ruby red. With Ehrlich's triacid stain the nucleus stains greenish to deep blue, the granules violet, and the protoplasm between the granules pink. These cells constitute the majority (about 75 per cent.) of the normal leucocytes.

2. Eosinophile Leucocytes: These cells vary in size, but are usually about the same size as the polymorpho-nuclears. Their nuclei are usually bilobed, and stain more faintly than the nuclei of the polymorpho-nuclears. Their most characteristic feature is the presence of large granules in their protoplasm. These granules stain red with eosin, the triacid stain, and with Leishman's stain. They constitute 1 to 4 per cent. of the total leucocytes of the blood.

3. Basophile Leucocytes or Mast Cells: The protoplasm of these cells contains coarse granules about the size of eosinophile granules or larger; these granules are often of different sizes and shapes. The granules have a marked affinity for basic stains, and stain a deep blue with Leishman's stain, but remain unstained with the triacid mixture. The nucleus is usually single, but may sometimes be polynuclear. They appear in very small numbers, if at all, in normal blood.

*Non-Granular Leucocytes.*—1. Small Lymphocytes: They are usually a little larger than the red cells, and possess a spherical nucleus, almost filling the cell, surrounded with a narrow homogeneous (non-granular) rim of protoplasm. With Leishman's stain the nucleus is ruby red and the protoplasm blue. The small lymphocytes constitute about one-fourth of all the leucocytes normally present in the blood.

2. Large Mononuclear Leucocytes: These are usually the largest cells seen in normal blood. Their nuclei are vesicular, and often eccentrically placed; they may be circular, horseshoe-shaped (transitional), or elongated. Their protoplasm is relatively much more abundant than that of the typical lymphocytes. With Leishman's stain the nuclei are ruby red and the protoplasm a faint blue, while with the triacid stain the nuclei are greenish or

blue and the protoplasm a faint red. All grades of transition between the small lymphocytes and the large mononuclears are found in pathological bloods. These cells occur in normal blood in 2 to 4 per cent.

3. The Transitional Leucocytes may be regarded as large mononuclear leucocytes, whose nucleus is more twisted than usual. It is often horseshoe-shaped. Apparently they sometimes contain a few neutrophile granules, and are regarded as transitional between the large mononuclears and the polymorpho-nuclears. They occur only in small numbers in normal blood (1 to 2 per cent.).

*Leucocytes in Pathological Blood.*—Besides the above forms of leucocytes, which alone are found in normal blood, other types of white cells may be found in the circulation in disease.

(1) Neutrophile Myelocytes: These are large cells with a single nucleus and granular protoplasm of the same type as that of the polymorpho-nuclear cells. The spherical nucleus usually occupies the greater part of the cell and stains faintly. The granules in the protoplasm are best demonstrated by the use of Ehrlich's triacid stain. These cells constitute the majority of the cells of the bone-marrow, but do not, in normal conditions, find their way into the general circulation. They are regarded as the antecedents of the normal polymorpho-nuclear leucocytes.

(2) Eosinophile Myelocytes: These are mononuclear eosinophiles. They are usually larger than the eosinophile leucocytes, and have a somewhat smaller nucleus than the neutrophile myelocytes. They occur normally in the bone-marrow, but not in the general circulation. They may be regarded as the antecedents of the eosinophile leucocytes of normal blood.

(3) Non-Granular Marrow Cells: These are very large, delicate cells with homogeneous protoplasm. The nucleus and protoplasm stain very faintly. Occasionally a few neutrophile granules may be seen in the protoplasm. They may be considered as transitional forms of myelocytes. They do not occur in normal blood.

The following may be regarded as a normal differential count:

Polymorphonuclear leucocytes	=	70	per cent.
Small lymphocytes	=	23	"
Large mononuclear leucocytes	=	3	"
Transitional leucocytes	=	2	"
Eosinophile leucocytes	=	2	"
Basophile leucocytes	=	0	"
Neutrophile myelocytes	=	0	"
Eosinophile myelocytes	=	0	"

The origin of these different types of leucocytes cannot be discussed here, but it may be mentioned that the bone-marrow is now regarded as the birthplace of the granular leucocytes of the blood, the polymorpho-nuclears arising from the neutrophile myelocytes, and the eosinophile leucocytes from the eosinophile myelocytes. The lymphocyte group of cells originate, for the most part, in the lymph glands and lymphatic tissues throughout the body. By many authorities the large mononuclear leucocytes are regarded as the representatives in normal blood of the parent blood cell. This cell is supposed, on the one hand, by a change of its nucleus and an acquisition of granules, to give rise to the polymorpho-nuclear and eosinophile leucocytes; in another line of development it is supposed to give rise to the typical lymphocytes. Although this view is probably correct in a developmental sense, the granular and non-granular cells have different origins in mature differentiated blood.

Variations in the numbers and differential count of the leucocytes are of great importance in the diagnosis of blood conditions (*cf.* 75).

1. *Polymorpho-nuclear Leucocytosis.*—This refers to an increase in the polymorpho-nuclear elements, occasioning also an increase of the total number of leucocytes. This is the most common type of leucocytosis. The total count of leucocytes may rarely rise above 100,000 per cubic millimetre, but is usually below 50,000 per cubic millimetre. Of these leucocytes the polymorpho-nuclears account for from 80 per cent. to 95 per cent. This form of leucocytosis occurs in—

(1) Acute inflammatory conditions, such as abscesses, serous inflammations, gangrenous inflammations, etc.

(2) In the majority of the acute infectious diseases, *e.g.* erysipelas, pneumonia, cerebro-spinal fever, suppurative meningitis, diphtheria, scarlatina. Leucocytosis of this sort is usually absent in uncomplicated tubercle, typhoid, malaria, measles, and influenza.

(3) In cachectic conditions, such as malignant disease, sometimes the leucocytosis is to be accounted for by septic processes occurring in the growth, but rapid dissemination without apparent septic changes will produce considerable leucocytosis of this sort.

(4) After large hæmorrhages there is a temporary leucocytosis, lasting for several days.

The presence or absence of a leucocytosis of this sort may be

of considerable help in diagnosis. One or two applications of its value may be mentioned.

In an acute febrile illness, where the diagnosis may be typhoid, influenza, malaria, or pneumonia, a marked polymorpho-nuclear leucocytosis will point to pneumonia rather than to any of the other conditions suspected.

In undoubted typhoid fever, the presence of a leucocytosis of this type points to some inflammatory complication, such as pneumonia, abscess formation, or suppurative peritonitis. If perforation be suspected from the clinical symptoms, the presence of a marked leucocytosis will confirm this diagnosis.

Where the signs of croupous pneumonia are well marked, and there is a total absence of leucocytosis, the prognosis is very grave, as such cases are almost invariably fatal. In a well-marked microbic infection, in which it is the rule for leucocytosis to occur, its absence points to a failure or exhaustion of the machinery of immunization, and is therefore of the gravest import.

In a case where a deep-seated abscess is suspected, a very high leucocytosis (30,000 to 40,000 per cubic millimetre) probably points to the presence of pus. With lower degrees of leucocytosis this diagnosis is neither justified nor negatived. If, on repeated examination, the leucocytosis is steadily increasing, the presence of pus is probable.

In a meningeal inflammation the presence of a high leucocytosis points to a suppurative inflammation, such as septic meningitis or cerebro-spinal fever, rather than tuberculous meningitis.

The presence of leucocytosis in an anæmia is in favour of the condition being secondary rather than primary pernicious anæmia or chlorosis.

2. *Lymphocytosis*.—In this condition the lymphocytes are present in increased numbers. Two varieties of lymphocytosis may be distinguished as absolute or relative lymphocyte leucocytosis :

(a) *Absolute Lymphocyte Leucocytosis* : In this condition the total number of leucocytes is increased, and the percentage of lymphocytes is higher than normal. This condition is found frequently in the anæmias of children. It reaches its acme in lymphatic leukæmia. In this disease over 90 per cent. of the white cells are usually lymphocytes. It also occurs in some cases of sarcoma with metastases in the bone-marrow.

(b) *Relative Lymphocytosis* : In this condition the total number

of leucocytes is normal or below normal, but the percentage of lymphocytes is increased. This is a very common condition in anæmias and in conditions with glandular hyperplasia. It occurs in pernicious anæmia, typhoid fever, small-pox, measles, malaria, Malta fever, etc.

The large mononuclears frequently partake in the conditions of lymphocytosis already mentioned. They occur in very large numbers in some cases of acute lymphatic leukæmia. In malaria a large increase of these cells is very common, although the total number of leucocytes is not increased. In a doubtful case, where malaria is in question, an abnormal number of these cells should encourage a prolonged search for parasites. These cells are also found increased in typhoid and Malta fever, and in other conditions with splenic enlargement. On this account they have sometimes been called splenocytes.

3. *Eosinophile Leucocytosis (Eosinophilia)*.—This is an increase in the percentage of eosinophile leucocytes. The total number of leucocytes may or may not be increased. It occurs in the following conditions: (a) bone affections: sarcoma, myelogenic leukæmia, osteomalacia; (b) diseases of the nervous system: neurasthenia, hysteria, etc.; (c) diseases of the skin: eczema, pemphigus, pityriasis rubra, etc.; (d) after splenectomy; (e) during convalescence from some acute infections of pneumonia, diphtheria, etc.; (f) it probably reaches its acme in parasitic invasions, e.g. trichina, bilharzia, ankylostoma, filaria, etc.

4. *Myelocytosis*.—Myelocytes are not present in normal blood, so that their presence in blood may be styled myelocytosis. Neutrophile and eosinophile myelocytes are very characteristic of spleno-medullary leukæmia. Any blood whose differential count contains 20 to 40 per cent. of myelocytes is splenic leukæmia.

Myelocytes are also found in other conditions, such as pernicious anæmia, von Jaksch's anæmia, diphtheria, etc. (See the articles on Anæmia, p. 28, and on Leucocytosis, p. 176.)

C. **Blood Platelets**.—These are often seen in stained films. Their study is not as yet of much diagnostic importance—authorities are at variance as to their origin and significance.

D. **Blood Parasites**.—Blood parasites are easily found and studied in stained films. Leishman's stain is one of the best for this purpose. They are of great importance in diagnosis.

The four most important varieties of parasites at present known



to occur in the circulating blood are—(1) the *Plasmodium malariae*; (2) the spirochæte of relapsing fever; (3) the varieties of filaria; and (4) the trypanosomata.

1. *Plasmodium Malariae*.—The examination of the blood from a suspected malarial patient should be made during the decline of the fever, or shortly after it. The patient should not have taken quinine for several days before the examination. Unstained fresh blood, allowed to spread in a thin layer between a cover-slip and slide, may be examined at once under an oil-immersion lens, and the parasites can frequently be made out in this way. The most information is, however, obtained from the study of stained films. Leishman's stain is, perhaps, the most convenient for this purpose.

There are three well-known varieties of the malarial parasite—the tertian, the quartan, and æstivo-autumnal. For a full description of the life-history and forms of these parasites, the reader should consult some special work on the subject.

The diagnosis of the different varieties of fever from the study of blood films is in some cases quite easy, in others extremely difficult or impossible. A double infection may frequently complicate the diagnosis. The examination for this purpose should go hand-in-hand with a study of the temperature chart of the case.

The following points may be mentioned: The tertian parasite is characterized by large pigmented rings, and by the enlargement of the red cells; the quartan by the band forms, and there is no enlargement of the erythrocytes; the æstivo-autumnal variety is characterized by crescents. If the parasites are found only in the ring forms, it may be impossible to make a diagnosis from the microscope examination alone, since the small tertian and quartan rings have much the same appearance, and may resemble the larger forms of the æstivo-autumnal rings.

2. *The Spirilla of Relapsing Fever*.—The spirilla are usually only found during the fever, so that the examination should be made at this time. The spirilla are detected, though with some difficulty, in unstained specimens by the fact that, in moving about, they displace the blood-corpuscles. They may be stained by Leishman's stain. These spirilla, discovered by Obermeyer, are highly motile very fine spiral threads, with pointed ends, 10  $\mu$  to 40  $\mu$  in length and 1  $\mu$  in thickness. They usually lie singly or a few side by side.

3. *Filaria*.—The embryos of several species of nematode worms

are found in the circulation. At least three species are well known: (a) *Filaria diurna* appears in the peripheral blood during the day, and disappears during the night; (b) *Filaria nocturna* appears at night only; (c) *Filaria perstans* are constantly present both day and night.

*Method of Examination.*—As the filaria are of considerable size and very motile, they can easily be detected in the fresh blood. Fresh specimens, prepared in rather thick layers, should be looked over with a low power of the microscope.

*Filaria sanguinis hominis*, with its embryo, *Filaria nocturna*, are responsible for certain tropical forms of chyluria, elephantiasis, and lymph-scrotum.

4. *Trypanosomata.*—These parasites are probably responsible for the disease known as African lethargy, or sleeping sickness. They also produce fever, general emaciation, and enlargement of the lymphatic glands.

In blood films they can be well stained with Leishman's stain. The trypanosoma is a long-shaped protozoon, containing a large nucleus centrally, and a vacuole, or contractile vessel, at the larger end. The single flagellum proceeds from a small mass of chromatin at the anterior end. The flagellum forms the edge of the undulating membrane which is observable from end to end of the organism, and continues in the same direction for some length as a free tail. It measures  $18.26 \mu$  by  $2.5 \mu$ .

The blood is best examined when the temperature is high, and, if none are found, the blood may be citrated and centrifugalized, as the trypanosomes accumulate in the leucocyte cream above the red corpuscles.

## II. The Examination of the Blood Serum.

For this purpose the blood is obtained in one of Wright's blood capsules, and after being allowed to clot, is centrifugalized, and the clear serum obtained from the top. In many of the methods of examination described in this and the following section the use of blood capsules and capillary pipettes of Wright's design is necessary. The following is a short description of the method of making and using these capsules and pipettes:

**Blood Capsules** of the form No. 4 in Fig. 20 are easily made from a piece of glass-tubing by heating parts of the tube in a

blowpipe flame and drawing it out as follows: a piece of tubing is first drawn out in two places into the form 1 in Fig. 20. The centre of this capsule (No. 2) is then heated in the flame and drawn out, and before the glass has cooled a Z-shaped bend is made, as in 3; from this form two blood capsules of the form shown at 4 are obtained.

These capsules are charged with blood as follows: The finger or ear is first punctured with an appropriate pricker. A good pricker can be easily made by drawing out a small piece of tubing into a very fine capillary tube and breaking it off, or a hypodermic needle may be used for this purpose. The blood capsule, with both capillary ends open, is now taken in hand, and the

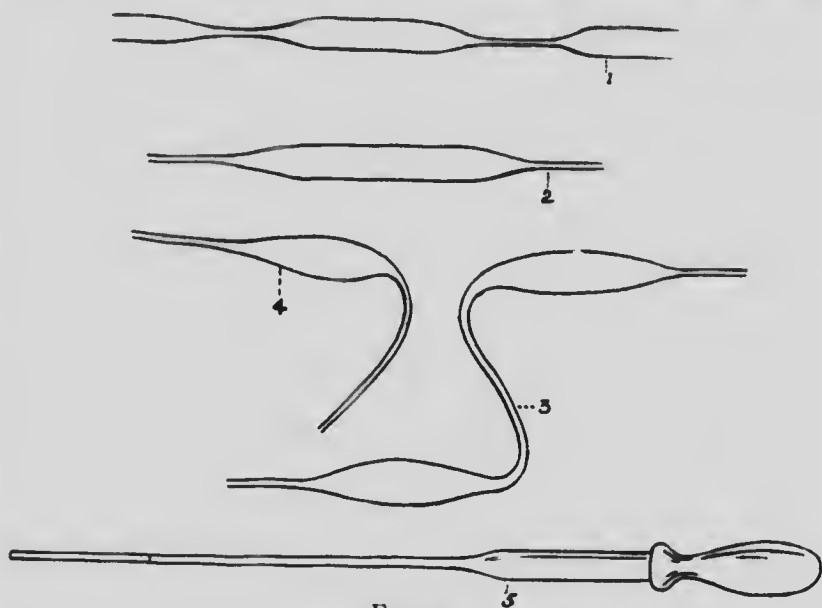


FIG. 20.

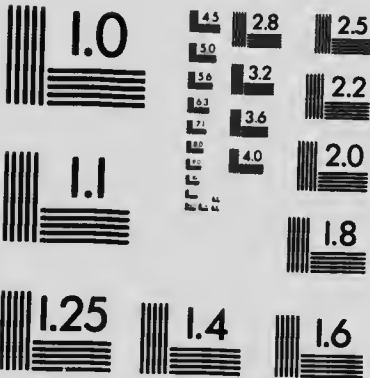
1, 2, 3, 4, blood capsules; 5, blood pipette.

recurved end of the capsule is applied to the drop as it exudes from the finger. The blood is allowed to flow into the capsule by capillary action until a sufficient quantity has been obtained. If the blood does not flow freely enough from the puncture, a handkerchief or piece of bandage can be wound round the finger above the puncture, and this will increase the flow. Care must be exercised not to allow the blood to flow into the straight end of the capsule, as if this is done the blood will be heated in sealing the capsule. The straight end of the capsule is now sealed in the



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flame. The sealing is found to be efficient by the blood receding further into the capsule, owing to the contraction of the air as it cools. If desired, the recurved end of the capsule can then be sealed without injury to the blood by heat.

The recurved end of the capsule furnishes us with a very convenient means of centrifugalizing the blood.

**Capillary Pipettes.**—These are made by simply heating a piece of glass-tubing and drawing it out into a capillary tube of appropriate thickness—two capillary pipettes of the form 5 in Fig. 20 result. These capillary pipettes are useful for numerous purposes, especially for measuring small quantities of blood or fluid, and for making any desired dilution of a given fluid.

The capillary pipette is provided with an accurately fitting rubber teat, and at a convenient distance from the capillary end of the pipette a mark is made with a glass writing pencil. The diagram explains the result obtained.

**Method of making Dilutions.**—By means of the action of the rubber teat we draw up into the capillary tube in succession:

1. A volume of the fluid to be diluted sufficient to fill the tube up to the mark.
2. A bubble of air to serve as an index.
3. The required number of volumes of the diluting fluid with a bubble of air between each.

Thus, supposing we require a dilution of 1 in 5: one volume of the dilutand and four volumes of the diluting fluid are taken. These may be mixed in the neck of the capillary tube, or may be blown out on to a slide and mixed by several aspirations.

If we wish a dilution of 1 in 100, it is best to do this by two successive operations, thus:  $\frac{1}{100} = \frac{1}{10} \times \frac{1}{10}$ —*i.e.*, a dilution of 1 in 10 is first made, and then  $\frac{1}{10}$  dilution of this dilution is made at a second operation, giving a final dilution of  $\frac{1}{100}$  of the original fluid.

To arrive at a dilution such as  $\frac{3}{5}$ , we take 3 volumes of the dilutand and 2 volumes of the diluting fluid.

### 1. The Alkalinity of the Serum.

This is accomplished by making use of a series of dilutions of decinormal sulphuric acid in 0.8 per cent. sodium chloride corresponding to  $\frac{N}{20}$ ,  $\frac{N}{30}$ ,  $\frac{N}{40}$ ,  $\frac{N}{50}$ , and  $\frac{N}{60}$ . These dilutions can be made up and stored in separate bottles, or the required dilution may at once be made by the use of Wright's pipettes. Sensitive litmus

is also required. A capillary pipette is taken, and a mark made on the stem about 1 inch from the end; the pipette is fitted with an indiarubber teat. By means of the rubber teat a quantity of serum is drawn into the capillary tube sufficient to fill it up to the mark; a bubble of air is then admitted to act as an indicator, and an equal quantity of one of the acid dilutions is then taken up into the same pipette. These two quantities of fluid are mixed by blowing them out on to a slide, and the mixture is then applied, by means of the pipette, to the sensitive litmus-paper, and the alkalinity or acidity of the mixture determined. Proceeding in this way the acid dilution is determined, which, as nearly as can be judged, exactly neutralizes the alkalinity of an equal quantity of serum. This dilution represents the alkalinity of the serum in question.  $\frac{N}{35}$  is found to be the normal alkalinity of the blood serum.

In scurvy the alkalinity of the blood serum tested in this way is greatly decreased ( $\frac{N}{100} - \frac{N}{200}$ ). There is, therefore, reason to believe that this condition is really one of so-called acid intoxication.

## 2. The Saline Concentration of the Serum.

This can be determined by the hæmolytic method devised by Wright. The method depends on the fact that when a drop of blood is placed in distilled water, the red cells promptly lose their hæmoglobin, or hæmolysis occurs; but when blood is placed in a solution of saline of a certain concentration, the red cells retain their hæmoglobin, or hæmolysis does not occur. The point at which hæmolysis occurs can be accurately determined by the use of capillary pipettes, and we have thus a method of estimating the saline concentration of blood serum or any solution containing salts.

This method finds its chief application as a practical *substitute for comparative cryoscopy*. The following paper explains the usefulness of this method:

'The presence or absence of albumin in the urine does not always furnish us with an adequate test of the efficiency of the kidneys, as the following considerations will show:

'1. In cases of granular kidney albumin may be absent from the urine, although the kidneys may be in a most inadequate condition, and the patient liable to all the serious consequences of this disease.

'2. There seems little doubt that so-called physiological albuminuria does not necessarily mean that the kidneys are inadequate, and may in some cases be due to the condition of the blood, and not to any abnormal kidney condition. Some cases may be good lives in every sense of the term, and ought not to be rejected by insurance companies. The condition may be curable, and may never develop into nephritis.

'3. Albumin may be present in various inflammatory conditions of the urinary tract, and yet the kidneys be functionally adequate.

'4. In surgical cases, where nephrectomy is in view, the presence or absence of albumin does not furnish the much needed indication of whether the other kidney has sufficient excretory power or not.

'To test the renal excretory power the estimation of the amount of urea in the twenty-four hours provides useful information; but this test leaves out of consideration the other salts of the blood, which are probably of even greater importance than the presence of non-excreted urea. We also have no convenient means of estimating the amount of urea formed in the system in varying conditions.

'Various drug tests have been suggested as tests of the renal excretory power, such as methylene blue and phloridzin. They have the advantage over urea estimations, that a known amount of the substance is administered, and the degree of retardation or prolongation of the elimination can thus be judged.

'Hitherto the most satisfactory contribution to this subject is to be found in cryoscopy. Koranyi and Kummel have been the pioneers of this method in its application to clinical work. The process is based on the fact that the greater the number of molecules in solution the lower the freezing-point. That is to say, the freezing-point shows the degree of concentration (or, as it is termed, the "osmotic" pressure) in the fluid. Adequate kidneys have the power of secreting urine of practically double the molecular concentration of the blood. When they are unable to do this the kidneys are inadequate. Comparative cryoscopic determinations of blood and urine will, therefore, furnish us with an accurate method of determining the adequacy of the kidneys.

'Such examinations, however, involving as they do a complicated and delicate apparatus and a large quantity of blood (3 or 4 drachms), are not practical in connection with ordinary clinical work.



'It remained, therefore, for the genius of A. E. Wright to devise a more excellent practical way. This he has done, as described in two recent communications to the *Lancet*.

'The method depends on the fact that when a drop of blood is placed in distilled water, the red cells promptly lose their hæmoglobin, and hæmolysis occurs; but when blood is placed in a solution of salt of a certain concentration, the red cells retain their hæmoglobin, and are not laked or hæmolyzed.

'The method in its simplest form is accomplished as follows:

'The following dilutions of decinormal saline are prepared by means of Wright's pipettes—*i.e.*, 2, 2½, 3, 3½, and 4. These solutions usually suffice, except in bloods of high concentration, when higher dilutions must be used.

'The patient must not have ingested fluid for two or three hours before the performance of the test.

'Two parts of each of these dilutions are mixed with one part of blood and respired into the capillary pipette, and the point at which hæmolysis is complete is noted.

'The same operation is conducted with urine, using in this case dilutions of 2, 4, 8, 16, and the hæmolytic effect noted. Supposing, for instance, dilution 16 shows hæmolysis and dilution 8 does not, intermediate dilution should be made. With a little practice the method is easily accomplished.

'The method of calculating the excretory quotient is best understood by an example: Supposing hæmolysis occurs only in the fourth dilution with decinormal saline, and only in the sixteenth dilution with the urine, then,

$$\frac{U}{16} = \frac{N}{40} \quad (N = 5.85 \text{ per cent.});$$

$$\therefore U = 2.340.$$

That is, the relation of the saline concentration of the urine to that of the serum is as 2.34 is to 1.

'I have been testing this method in a number of cases, with the following results:

(x)	} Three presumably healthy persons	{	2.4
(y)			2.3
(z)			2.3

1. M.M. Parenchymatous nephritis, albumin, and many casts (six months' duration) ... .. 1.4

2. J. Parenchymatous nephritis, albumin, and casts (twelve months' duration) ... .. 0.936
3. Heart case. Trace of albumin in the urine; patient delirious; probably uræmic ... .. 0.78
4. Sarcoma of kidney; no albumin ... .. 2.006
5. M.M. Pyuria. Irregular temperature; urine contains pus and trace of albumin. Specific gravity 1015 ... .. 1.04
6. T. M. Parenchymatous nephritis albumin casts. Specific gravity 1020 ... .. 0.88
7. Mrs. C. Eclampsia. Urine loaded with albumin. Specific gravity 1018. Convulsions ... .. 0.87
8. Patient with high tension pulse. Trace of albumin has been found; none at present. Specific gravity 1020. Average daily excretion between 40 and 50 ounces ... .. 1.87
9. Gout. Trace of albumin occasionally present; no casts. History of calculus. Specific gravity 1012. No albumin at present ... .. 1.6
10. A case of tuberculosis of the kidney. The right kidney had been enlarged and movable for some years; tubercle bacilli and pus were present in the urine. Removal of the right kidney was under consideration. With a view to determine the excretory power of the left kidney the left ureter was catheterized, and the urine was collected. It contained a trace of albumin, had a specific gravity of 1015, and gave the excretory co-efficient of 1.1. The operation was therefore deemed inadvisable.

'So far as these results go, this test seems to furnish a very useful clinical test of the renal excretory power. There are some theoretical objections that may be urged. I think, however, that these objections are merely theoretical ones, and that the test will be found invaluable.

'It might, I believe, give valuable information as to the proper diet for kidney cases, and will often mark a danger-signal in such cases. It should prove invaluable to the surgeon when he is meditating a nephrectomy, and may possibly cause insurance companies to alter their views of functional albuminuria.'

### 3. The Opsonic Power.

Opsonins are substances contained in the serum or plasma of the blood. They possess the power of so acting on micro-organisms as to make them an easy prey for the phagocytes. This power of the serum Wright has called the opsonic power, from the Latin *opsono*, I cook, I make ready a feast. It has been shown that for healthy subjects the opsonic power is practically constant, but is usually anomalous—*i.e.*, either lower or higher than normal—in patients infected with the micro-organism. The opsonin appears to be specific, and to be almost entirely destroyed by heat.

By the opsonic index is meant the relation between the opsonic power of the infected individual and that of a healthy person.

#### *The Method of Determining the Opsonic Power of the Serum.*

The following are required :

1. **Washed Corpuscles to provide the Phagocytes.**

2. **Sera**—(i.) The serum of the patient whose index is to be determined ; (ii.) the serum of a healthy person as a control, or, preferably, the sera of several normal individuals, to be mixed and used as a ' pool.'

3. **A Uniform Test Emulsion of the Micro-Organism concerned.**

1. Washed corpuscles are obtained as follows : Several drops of blood from the finger are received into several cubic centimetres of salt solution containing a little citrate of soda. The mixture is shaken, and the corpuscles separated by centrifugal action. The supernatant fluid is pipetted off. The corpuscles are then washed in several cubic centimetres of fresh salt solution, and the mixture again centrifugalized. The supernatant salt solution is again removed with a pipette. The deposit furnishes the leucocytes for the determination, the upper layers containing, as they do, a large number of white cells.

2. The sera are obtained by allowing small quantities of the bloods under consideration to clot in glass capsules, and centrifugalizing to separate the sera.

3. The preparation of the test emulsion varies somewhat with the micro-organism under consideration :

(i.) **Staphylococcus** : An agar culture of a twenty-four hours' growth is taken. The culture is removed from the surface of the agar with 1.5 per cent. salt solution,

- thoroughly mixed with the salt solution by shaking, and then centrifugalized to drive down any clumps, and diluted to an appropriate density with 1·5 salt solution.
- (ii.) Tubercle: A small quantity of sterilized tubercle growth is ground up in an agate mortar with 1·5 per cent. salt solution. This is removed into a glass capsule, centrifugalized, and diluted with 1·5 per cent. salt solution to an appropriate density.
- (iii.) *Bacillus coli communis*: The emulsion is made in a similar way to the emulsion of the staphylococcus; a two hours' growth of coli must be used.

Capillary pipettes are then provided; these are furnished with rubber teats, and a mark is made on the tubes about an inch from the ends. The following experiment will explain the procedure:

Washed corpuscles = 1 part	}	These are drawn into the capillary pipette with a bubble of air dividing each part.
Serum of patient = 1 ,,		
Test emulsion = 1 ,,		

These are thoroughly mixed on a slide and reaspirated into the capillary tube, the end is sealed, and the pipette placed in the incubator for fifteen minutes. With a second pipette a precisely similar operation is carried out, except that the control serum, or 'pool,' is substituted for the patient's serum.

Immediately after the fifteen minutes in the incubator has expired, blood films are made from the contents of the tubes, and these are appropriately stained. The micro-organisms in 40 or 50 polymorphonuclear leucocytes of the two preparations are then counted, and the relation between the results gives the opsonic index.

For instance, supposing the bacilli or cocci in 50 leucocytes of both preparations be counted, and in the preparation containing the patient's serum we find 100 bacilli in 50 leucocytes, while in the preparations containing the control serum we find 200 bacilli in 50 cells, then  $\frac{100}{200}$ , or 0·5, is the opsonic index in this case.

The following is an example of the classical experiment showing that the opsonic power is destroyed by heat:

- A 1. T. H.'s washed corpuscles = 2 parts.
2. Citrated serum = 2 ,,
3. Emulsion of staphylococci = 1 part.

Mixed and incubated for fifteen minutes.

Count of 100 cells = 218—*i.e.*, 2·18 per leucocyte.

- A<sup>1</sup>
1. T. H.'s washed corpuscles = 2 parts.
  2. Citrated serum (heated for twenty minutes at 60°) = 2 "
  3. Emulsion of staphylococci = 1 part.
- Mixed and incubated for fifteen minutes.  
Count of 100 cells = 28—*i.e.*, 0.28 per leucocyte.

The determination of the opsonic index has its chief application as an indication for the treatment of bacterial invasions by inoculation with the corresponding vaccines. It may, however, furnish most useful information from a diagnostic point of view. In almost all chronic infections without a temperature the opsonic index for the micro-organism concerned is below normal, while in acute infections the opsonic index varies, being as a rule above normal. It therefore follows that if, after a careful determination, the opsonic index for a given micro-organism be found anomalous, this result is an indication of infection with the micro-organism concerned. For purposes of diagnosis the determination must be made with the greatest care, and always repeated several times. The method is such that even the most careful workers are liable to obtain inaccurate results unless the determinations be repeated with a second or even a third example of blood. A few examples will make clear the application of this method to the diagnosis of bacterial invasions.

A patient had tenderness with some swelling over the sacro-iliac joint, and had suffered from lameness for a considerable time. The diagnosis on clinical grounds was doubtful. Two very careful examinations of the blood revealed an opsonic index on both occasions of 0.7 for tubercle. The condition was, therefore, diagnosed as tuberculous sacro-iliac disease, and treated with considerable benefit by tuberculin inoculations.

Another patient with ascites and pleural effusion was thought to be suffering from a tubercular invasion. Two separate determinations of the opsonic power of the blood revealed an absolutely normal opsonic index. A sample of the fluid from the pleural cavity had also a normal opsonic index for tubercle. The patient was afterwards operated on, and found to be suffering from malignant disease of the ovaries.

A patient was admitted to hospital with a high temperature, furred tongue, and swollen abdomen. Typhoid fever was sus-

pected. The blood gave a negative result to the agglutination test with typhoid bacilli, but a tuberculo-opsonic index of 3. The condition was therefore diagnosed as tuberculous peritonitis. The after-history showed that this diagnosis was correct.

A patient with symptoms of cystitis and pyuria. The *Bacillus coli communis* was isolated from the urine. The following indices for this micro-organism were found on three separate examinations: 1.2, 1.4, and 4. The patient was, therefore, actively infected with this micro-organism.

A patient in the North of Ireland, who had not been abroad for sixteen years, was suffering from undulatory fever, resembling that due to the *Microrhus melitensis*. On repeated examination, a varying opsonic index for this micro-organism—as a rule much above normal—was found. The patient was, therefore, believed to be suffering from Malta fever. This diagnosis was afterwards confirmed by the agglutination test.

#### 4. The Agglutinating Power of the Serum—Widal's Reaction.

This reaction can be applied to the blood of patients who are suspected to be suffering from cholera, typhoid, or Malta fever. The agglutinating power of the serum may also be of value in the diagnosis of other micro-organisms that are clumped by their specific sera. The technique of the reaction with reference to typhoid (Widal's reaction) will alone be described.

The serum of the patient is obtained as already described. By the use of capillary pipettes the following dilutions, with 0.8 per cent. sodium chloride solution, are made: 1 in 5, 1 in 10, 1 in 25, and 1 in 50. These dilutions may be made on a glass slide or in watch-glasses. A fresh culture (incubated about twelve hours) of typhoid bacilli, either on agar slope or in peptone-water, is obtained. If an agar growth is used, an emulsion of the bacilli in salt solution is made of an appropriate density. Equal quantities of this emulsion (or of the peptone culture) and of the various dilutions of serum are then taken and mixed with a capillary pipette, and each preparation of serum and bacilli is placed on a clean cover-slip in the form of a hanging-drop. Thus, a series of hanging-drop preparations, corresponding to dilutions of the original serum of 1 in 10, 1 in 20, 1 in 50, and 1 in 100, are obtained. Place the cover-slips on hollow glass slides, or on improvised hollow slides made by placing a ring of moist blotting-paper on an ordinary slide, and examine occasionally. A dilution

of 1 in 100 should show well-marked agglutination with motionless bacilli within two hours if the case is typhoid fever. The lower dilutions will usually be clumped in a much shorter time. The use of the lower dilutions is to determine if any of these dilutions clump when the higher dilutions are negative. This may give useful information as to the necessity for a second examination of the blood; but unless a dilution of 1 in 50 shows marked agglutination at the end of an hour, a positive result should never be recorded. A control hanging-drop, diluted with salt solution should be examined to exclude pseudo-clumping.

It is often of great value in cases where the reaction is indistinct, or only occurs in low dilutions, to examine a film preparation of the blood. The combination of an indistinct serum reaction, diminution in fibrin, absence of leucocytosis, with a relative lymphocytosis, especially if the large mononuclears are increased, is seldom or never found in the early stages of an obscure febrile illness, except typhoid.

**Value of Widal's Reaction in Diagnosis.**—Widal's reaction properly done is a most valuable test. Unless a distinct reaction occurs in a dilution of 1 in 50 in an hour a positive result should not be recorded. A negative result is of little value unless obtained at the height of the fever (in the third or fourth week) and on repeated examination. The agglutinative power of the serum is usually slight and transient in very mild cases, and marked and persistent in severe cases, but bears no constant relation to the gravity of the disease, and gives no certain prognostic indications.

### J. Wassermann's Reaction.

This test is generally spoken of as the *fixation of complement*. It was originally applied by Wassermann to the diagnosis of syphilis following the phenomenon of Bordet and Gengou. Foreign substances of an organic nature introduced into the animal body are termed antigens. The repeated injection of small doses of antigen results in a certain degree of immunity on the part of the animal to further injection of that substance. This immunity apparently depends on the production of antibodies in the blood of the inoculated animal. One of these antibodies is termed the immune body. This immune body has the power of linking the antigen to the complement which is found in normal blood, and thus neutralizes the effect of the

foreign substance upon the animal. Thus where the antigen and its immune body or amboceptor are in contact free complement is taken up or fixed. These three substances—antigen, immune body, and complement—constitute an *immune system*. The immune body is thermo-stable—that is, it is not lost when the serum is heated to 55° C. for half an hour. The complement is thermo-labile and destroyed by heat. If the red blood-corpuscles of one animal (*e.g.*, sheep's corpuscles) are used as an antigen and inoculated into another animal (*e.g.*, a rabbit) an amboceptor for the sheep's corpuscles is formed in the rabbit's serum. Consequently, sheep's corpuscles combine with this body (amboceptor) in the serum of the inoculated rabbit, and in doing so have the power of fixing free complement. This reaction is shown by the hæmolysis of the sheep's corpuscles. These three substances—sheep's corpuscles, immune body, and complement—constitute a *hæmolytic system*.

Further research has shown that Wassermann's reaction is of a nature entirely different from this phenomenon of Bordet and Gengou. The antigen which is used is not a true antigen, nor is the substance in the syphilitic serum with which it unites a true antibody. The test, however, still remains of great value in the diagnosis of the disease.

The original method requires (1) a suitable extract or antigen; (2) fresh complement; (3) a hæmolytic system. The original antigen used by Wassermann was an extract of syphilitic liver swarming with parasites. Complement was supplied by fresh guinea-pig's serum, and the hæmolytic system was obtained by inoculating an animal such as a rabbit with sheep's corpuscles. The heated serum from the inoculated rabbit has the power of hæmolysing the corpuscles of the sheep when fresh complement is present. The corpuscles subjected to the action of the heated serum are said to be 'sensitized.'

Wassermann's reaction depends on the fact that when the so-called antigen or extract comes into contact with the so-called antibody found in the serum of the syphilitic patient complement is fixed. Hence, when sensitized corpuscles are added no hæmolysis takes place, because the complement has already been used up by the union of the extract and so-called antibody. Several modifications of the original reaction have been introduced, and Fleming's is one of the simplest, and gives very accurate results. Fleming uses as an extract an alcoholic extract of heart muscle—1 gramme



of fresh heart muscle is pounded up in a mortar with 5 c.c. of absolute alcohol. The mixture is heated to 60° C. for one hour, and then put in the incubator for twenty-four hours. The clear fluid is pipetted off, and, after dilution with normal salt solution, furnishes the extract for the test. The best dilution of the extract is found by a trial test with a normal and syphilitic serum. Fresh complement is provided by the complement found in the specimen of blood to be examined. The fact that human serum hæmolyses sheep's corpuscles is used as a natural hæmolytic system.

The test is performed as follows :

Small glass tubes about 1 inch long and  $\frac{1}{4}$  inch in diameter are obtained. These are arranged in two series, one marked salt solution, the other extract. Four parts of saline are measured with a Wright's pipette, and added to all the tubes of the series marked saline. Then four parts of extract are added to the series marked extract. The sera to be examined are then taken along with a control serum from a non-syphilitic blood. One part of each serum is added to corresponding tubes in each of the series. These preparations are now put in the incubator for twenty or thirty minutes to allow the extract time to unite and fix the complement. One part of a 10 per cent. suspension of sheep's corpuscles in saline is now added to each of the tubes, and the preparations put back in the incubator for one and a half hours. The tubes are then examined, and, provided the control shows hæmolysis in both tubes, the corresponding tubes which show hæmolysis in the salt tubes and no hæmolysis in the extract tubes are read positive, while tubes showing hæmolysis in both are negative. If the two corresponding tubes show no hæmolysis in either, the result is indeterminate. The only difficulty about this method of doing the reaction is that a certain number of human blood sera produce no hæmolysis with sheep's corpuscles and indeterminate results may be obtained. The test in these cases must then be repeated, adding to each a small quantity of a known hæmolytic non-syphilitic serum. The value of the test is very great. It is practically never present in health, and is very uncommon in diseases other than syphilis; it does occur, especially in diseases (like syphilis) due to animal parasites, such as malaria or trypanosomiasis, and is said to occur occasionally in leprosy and scarlet fever. In primary and secondary cases of syphilis it occurs in 90 per cent, or more, and is present in the majority of patients suffering from tertiary syphilis and 'meta-syphilitic' affections.

### 6. The Appearance of the Serum.

When the blood has been centrifugalized in the blood capsule, it may be important to note the appearance presented :

(i.) In certain conditions, notably Bright's disease, the serum may have a muddy appearance.

(ii.) In cases of jaundice the serum is seen to have a yellow or green appearance. This is a most delicate test for the presence of bile in the blood, as by this means it is quite easy to demonstrate the presence of jaundice when it cannot be detected in the conjunctivæ.

(iii.) The presence of hæmolysis can also be detected in this way.

## III. Some Further Methods of Examining the Blood.

### 1. The Specific Gravity.

The methods of determining the specific gravity of the blood are rather cumbersome for clinical purposes. The specific gravity seems to vary at least roughly with the hæmoglobin value, and it is easier and more accurate to determine the percentage of hæmoglobin than the specific gravity directly.

**Hammerschlag's Method** is, perhaps, as good as any. A mixture of chloroform and benzol is taken of about 1055 specific gravity. Into this mixture a drop of blood is brought with a suitable pipette. If it tends to fall, chloroform is gradually added till, after mixing, the drop remains stationary. If the drop rises, the specific gravity is too great, and benzol must be added. When the drop assumes a fixed position, neither rising nor sinking, the benzol-chloroform mixture and the drop of blood are then of the same specific gravity. The specific gravity of the mixture is then determined. The benzol chloroform mixture may be filtered through a dry filter, and used for other examinations. The specific gravity of normal blood is 1055 to 1060.

### 2. Determination of the Total Volume of Blood by the Carbon-Monoxide Method (Haldane and Lorrain Smith).

This method of estimating the total volume of blood, although it cannot be applied for clinical purposes except by an expert, is

so interesting, and has thrown such important light on blood conditions, that a short account of the method explaining the principles on which it depends deserves mention here.

The subject breathes into a special apparatus designed for this purpose: he thus absorbs a measured quantity of CO gas. The percentage saturation of the subject's hæmoglobin with CO is then determined by the carmine method described below, and from this the total capacity of the subject's blood for CO (or O) is determined. Thus, suppose the subject absorbs 100 c.c. of CO, and his blood is found to be 20 per cent. saturated, his total capacity for CO (or O) will be  $100 \times \frac{100}{20} = 500$  c.c. By estimating the percentage of hæmoglobin, the subject's oxygen capacity per given volume (say 100 c.c.), or, what is the same thing, his CO capacity per given volume, is now determined, and from this the total volume of the subject's blood is calculated. For instance, suppose the oxygen (or CO) capacity of the subject's blood to be 20 c.c. per 100 c.c., and his total capacity for CO (or oxygen) has already been found to be 500 c.c.; therefore the volume of the subject's blood is:

$$500 \times \frac{100}{20} = 2,500 \text{ c.c.}$$

**Method of Determining the Percentage Saturation of the Subject's Blood by the Carmine Method.**—Shortly after the subject has absorbed the CO gas, a sample of his blood is taken and diluted until it equals in density a 1 per cent. solution of ox or human blood; a measured quantity of carmine solution is now added to the diluted normal blood (measured quantity, say, 5 c.c.), until the tints exactly correspond, and the amount of carmine solution is noted. The subject's blood is now fully saturated with CO by shaking it up with gas. Carmine solution is again added to the normal blood solution until the tints again correspond and the amount of carmine solution again noted. Thus, supposing, in the first instance, 0.5 c.c. of carmine solution has been added to the normal blood to make the tint correspond to the subject's partially saturated blood, and, again, 4 c.c. of carmine solution to make it correspond in tint to the subject's fully saturated blood, then the ratio of the amount of carmine needed to produce the partial saturation tint to the amount which produces the total (100 per cent.) saturation tint, gives the fraction of complete saturation in which the subject's blood was after absorbing the CO.

As we began with 5 c.c. of normal blood solution, the calculation is as follows :

$$\frac{0.5 \text{ c.c. (partial saturation)}}{5.5 \text{ c.c.}} = \frac{x}{100} \quad \text{or} \quad \frac{1}{11} \times \frac{9}{4} = \frac{x}{100}$$

$$\frac{4 \text{ c.c. (complete saturation)}}{9 \text{ c.c.}}$$

$$\therefore x = \frac{9 \times 25}{11} = 22.2 \text{ per cent.}$$

By means of this ingenious method it has been demonstrated that in various anæmic conditions, especially in chlorosis, the total volume of blood in the body is increased. In fact, it has been shown that in chlorosis the total amount of hæmoglobin in the circulation is not decreased, so that in this disease the total number of red blood-corpuscles must often be greatly increased. The disease is, in fact, a dilution or hydræmic plethora of the blood rather than an anæmia in the strict sense of the term. This observation accounts for many of the symptoms of the disease, such as œdema and flabbiness of the tissues, and a certain increase of weight due to increased fluid. An apparent loss of weight, due to loss of fluid, is, as a rule, one of the first symptoms of improvement under treatment.

The method of determining the percentage saturation of the blood with CO is a very useful method of determining the quantity of CO in the blood in cases of coal-gas poisoning.

For further information the reader is referred to the papers by Haldane and Lorrain Smith.

### 3. The Coagulability of the Blood.

The coagulability of the blood is a very important factor in the study of blood conditions. It has been shown that a pathological condition of the blood characterized by diminished coagulability frequently occurs. This condition has been styled by Wright 'serous hæmorrhage,' and may account for such symptoms as urticaria, physiological albuminuria, chilblains, lymphatic headache, and even hæmorrhage. On the other hand, increased coagulability is sometimes the cause of thrombosis, increased viscosity of the blood, etc.

The methods of Wright are much the best for determining the coagulation time of the blood. A certain amount of information on this point may be obtained from an observation of the ease or difficulty with which blood is obtained from a puncture of the

finger in making an ordinary blood examination. Thus, in hæmophilia and purpura hæmorrhagica it may require firm pressure to stop the bleeding from a small puncture. A rough estimation of the coagulation time may be obtained by putting on a clean slide a series of equal drops of blood by means of a capillary pipette, and noting the time when coagulation occurs by drawing a clean needle through the drops at intervals. A string of fibrin indicates the beginning of coagulation.

**Wright's Coagulometer.**—This instrument, when carefully used, gives reliable results. A series of tubes of standard calibre are partially filled with blood taken from successive drops from the finger. The exact time is noted when each tube is filled, and, by means of a water-bath, the determination is made at a constant temperature, either at blood heat ( $37^{\circ}$  C.), or at half blood heat ( $18.5^{\circ}$  C.). The coagulation time is determined by blowing out the tubes at different intervals from the time of filling. The tubes should be blown out on to blotting-paper when the first threads of fibrin are easily detected, and in this way the coagulation time determined.

Recently Wright has invented a new and ingenious plan for making coagulation tubes. The tubes are calibrated with mercury, and made to hold a definite quantity of blood in the calibrated part of the tube. The reader is referred to the original paper in the *Lancet* for further details of this method.

The recent work of Addis on blood coagulability throws considerable doubt on the accuracy of the above and other methods for its determination. The conditions required for Addis's method, however, render it hardly applicable as a clinical procedure.

#### 4. The Method of Determining the Amount of Calcium Salts in the Blood.

When normal blood is mixed with an equal quantity of a solution of neutral ammonium oxalate, of the strength 1 in 800 in 0.8 per cent. of sodium chloride, the blood is prevented from forming a clot.

If the calcium salts are deficient, a weaker solution of ammonium oxalate will be sufficient to prevent clotting.

By means of Wright's pipettes a series of dilutions of the oxalate solution with normal saline are made—for instance,  $\frac{1}{800}$ ,  $\frac{1}{1200}$ ,  $\frac{1}{1600}$ ,  $\frac{1}{2000}$  will at first be appropriate dilutions.

By means of a pipette, equal parts of each of these dilutions

and blood from the finger of the patient are taken and mixed. The drops are then re-aspirated into the tube, and the tube is sealed and allowed to stand for some hours. It is then examined and the dilutions in which any clot appears is noted. Thus, if a clot is found with  $\frac{1}{1000}$  dilution, and not with  $\frac{1}{1200}$  dilution, the dilution of oxalate solution required to just keep the blood fluid, lies between these two dilutions. Intermediate dilutions can then be tried if greater accuracy is required.

The coagulation of the blood is intimately connected with the quantity of calcium salts in the blood.

#### 5. The Viscosity of the Blood.

The coagulation time of the blood and its content in calcium salts is closely connected with its viscosity. The viscosity has to do with the ease or difficulty with which the blood flows through the small vessels and capillaries, and has, therefore, most important bearings in disease. An instrument has recently been invented by Du Pré Denning, and Watson for determining the viscosity of the blood directly. Their viscometer consists of a curved piece of capillary tubing with two arms. The long arms, 6 cubic millimetres in length, has been blown out at its free end into a cup-shaped receiver with a thin edge. On the short arm, which is only 2 cubic millimetres in length, there is a small elliptical bulb, and the point at which the capillary enters and leaves the bulb is etched on the glass. The lobe of the ear is first cleaned with ether and then punctured, and then the receiver of the viscometer—thoroughly dry and previously heated to the temperature of the body—is filled with blood. As the blood flows down the capillary tube it is carefully watched. By means of a stop-watch the time required to fill the elliptical bulb is accurately determined. This time is compared with the reading for water. This time value seems to be a fairly reliable, comparative indication of the viscosity of the blood under consideration.

#### 6. The Isolation of Micro-Organisms from the Blood.

The plan usually adopted is as follows: After thorough disinfection of the skin, 5 to 10 c.c. of blood are obtained by puncture of the median vein by an all-glass syringe, which has previously been boiled. If the vein is with difficulty seen, the arm may be

compressed with a bandage above the elbow, thus making the vein stand out prominently. If the vein is properly entered by the needle, no difficulty is experienced in obtaining the required quantity of blood, which, if the piston works perfectly smoothly, will flow into the syringe without suction. The blood thus obtained is immediately distributed into a series of flasks containing from 50 to 100 c.c. of sterile bouillon, 1 to 2 c.c. of blood to each flask. It is important to do this operation with the most thorough asepsis and as quickly as possible. The blood should be thoroughly mixed with the broth by shaking before any clotting takes place. The flasks containing the blood are placed in the incubator, and subcultured daily on to agar slopes. The length of time required for incubation depends on the micro-organism concerned.

This method should be adopted in all cases of general septicaemia, as by this means an accurate diagnosis of the micro-organism concerned in the process may often be arrived at.

To exclude the possibility of accidental contamination, an opsonic determination should be made with the blood of the patient, and the micro-organism found.

If the opsonic index is anomalous—that is, above or below normal—there is then positive evidence that the micro-organism found is actively concerned in the infection.

T. HOUSTON.

#### BOX NOTE.

The tone elicited on percussing the emphysematous chest has usually a peculiar reverberating quality, resembling that heard when a box is struck, and is known as the box note (see p. 454).

#### BRADYCARDIA (Gr. *βραδύς*, slow; *καρδία*, the heart).

A term employed to indicate abnormal infrequency in the pulse-rate from any cause. Of these may be mentioned cachexia, starvation, conditions of high arterial tension, aortic stenosis, intracranial pressure, certain poisons and drugs, some forms of arrhythmia—*e.g.*, Stokes-Adams disease. (See Arterial Pulse, p. 306.)

#### BREATHING, Disturbances of.

In the article on *Dyspnœa* (p. 125) the causes and nature of respiratory disturbances are considered; the movements of the

chest and of the abdomen in disease are discussed in the sections on the examination of those regions, at pp. 469 and 10.

**BREATHLESSNESS.** See *Dyspnœa*, p. 125.

**BREATH, Odour of.**

The sweet odour of acetone may be observed in the breath of diabetics for months, or even years, before the fatal termination. While, therefore, it must be regarded as a sign of serious disease, it is an error to assume that it is a warning of impending coma or collapse.

A somewhat similar odour may be temporarily perceived in the breath of persons, and particularly children, suffering from digestive disturbance.

The offensive odours arising from *ozæna*, gangrene of the lung, hæmoptysis, and hæmatæmesis, have only to be once experienced to be recollected.

Alcohol, ether (largely used as an intoxicant in certain localities in the North of Ireland), opium, and other poisons in the stomach give their characteristic odours to the breath.

The foetid odour due to indigestion and to constipation and that due to caries of the teeth are among the commonest of the abnormal odours of the breath.

**BREATH-SOUNDS.**

In auscultating the chest certain sounds may be recognized as those produced by the act of respiration, and modified either by the normal structures through which the vibrations are transmitted to the stethoscope or by various abnormal conditions the result of disease. The study of the breath-sounds, as distinguished from those fresh or adventitious sounds which have no counterpart in health, is of the utmost importance in the formation of a diagnosis, and the subject is considered in some detail in the article on Auscultation of the Thorax (p. 408).

**BRIQUET'S SYNDROME.**

A series of symptoms forming a type of hysterical affection—viz., shortness of breath; loss of voice; inaction of the diaphragm, causing the respiration to be of a panting, excessive, and thoracic character.



**BROADBENT'S LAW.**

Central or supranuclear lesions of the motor nerves do not necessarily cause complete paralysis of the muscles supplied by the injured nerve, even if the path from the cortex to the nucleus be completely interrupted. This is because of the fact that the motor nuclei in the medulla, pons, and crura, as well as those in the anterior cornual grey matter, receive cortical communicating fibres from both hemispheres. This bilateral innervation is especially well furnished to those nuclei which govern the action of muscles which habitually act in concert with their fellows on the opposite side of the body. It has been pointed out by Broadbent that (in consequence of this mode of nerve supply) lesions of the upper segment of the motor tract—*i.e.*, supranuclear lesions—have less paralyzing effect upon those muscles which habitually produce bilateral movements than upon those which more frequently act independently of the opposite side. For example, in hæmiplegia the arms are more powerless than the legs, while the trunk muscles commonly escape. The more frequent involvement of the muscles of the lower half of the face than those of the upper is another example. (See Movement, Decreased, p. 225.)

**BROADBENT'S SIGN.**

A systolic retraction of the tenth and eleventh interspaces below the left scapula has been shown by Dr. J. F. H. Broadbent to be an important sign of adherent pericardium. When the adhesion is extensive, the contracting ventricles pull the diaphragm away from its attachments to the posterior thoracic wall to a sufficient extent to cause the retraction referred to.

**BRONCHIAL BREATHING.**

Normally the breath-sounds heard by placing the stethoscope on those parts of the chest-wall near which the larger bronchi are situated have a harsh, blowing character, in which the expiratory portion is, if anything, of a more pronounced intensity and higher pitch than the inspiratory; in these respects bronchial breathing contrasts with vesicular breathing. Bronchial breathing is normally heard in the upper part of the chest, especially in the neighbourhood of the last cervical and four upper dorsal vertebræ; in front bronchial breathing is normally heard in the first and second interspaces, not far from the sternum, and most markedly

on the right side. The nearer to the trachea that one places the stethoscope, the louder the bronchial breathing; it is simply the sound of the air-current passing through the glottis, and no doubt sounds produced in the mouth and larger air-tubes contribute to the effect. The sound-waves are conveyed through the bronchi and tissues to the surface of the chest, and variations in the intensity and quality of the sound must depend upon differences in the production of the sound, and in its conduction to the surface. In diseased conditions these variations in the bronchial breathing and its appearance in unusual situations afford valuable information as to the state of the subjacent tissues. The subject is further discussed at p. 409.

### **BRONCHIAL FREMITUS.**

In bronchitis, bronchiectasis, phthisis, asthma, etc., the passage of air through fluids and through catarrhal and constricted channels causes a palpable vibratory movement of the chest-wall. It is best observed in children, but is of trifling diagnostic value, as the condition is more advantageously investigated by the stethoscope.

### **BRONCHOPHONY.**

An increase in the vocal resonance, indicating in most cases consolidation of the lung; if well marked it suggests that the consolidation is dense, and is in close relation to large open bronchi. Under these circumstances the voice-sounds are well conducted from the larynx to the surface, and give one the impression of emanating from the spot immediately under the stethoscope. If they are badly conducted, as is the case when the consolidation is imperfect, or is deeply placed in the chest, with intervening healthy lung tissue, the increased resonance is not distinctly heard. We find well-marked bronchophony, therefore, in pneumonia, phthisis, and compression or relaxation of the lung. Cavities in the lung, which are usually surrounded by inflammatory consolidation, may give rise to bronchophony, to which a cavernous or distinctly articulate quality may be added (see p. 415 *et seq.*).

### **BRONCHO-VESICULAR BREATHING (Transitional, Indeterminate, or Mixed Breathing).**

A combination of bronchial and vesicular breath-sounds, in which the inspiratory portion is either vascular or a mixture of

both forms, while the expiratory part is usually bronchial. This sound represents increased intensity of the breath-sounds in many cases, and may, as a rule, be regarded as the first step in the production of true bronchial breathing (see p. 410).

**BRUIT D'AIRAIN.** See **Bell Sound**, p. 56.

**BRUIT DE CUIR NEUF (New-leather Sound).**

A creaking sound sometimes heard in pleurisy, and more commonly in pericarditis; it replaces the rub which is usually heard in these conditions, and is due to similar causes.

**BRUIT DE DIABLE.** See p. 444.

**BRUIT DE DRAPEAU.**

A dry, crackling râle, heard in cases of plastic bronchitis. It is probably due to the vibration of a loosened portion of the fibrinous cast.

**BRUIT DE POT FELE.** See **Cracked-pot Sound**, p. 111

**BULIMIA** (Gr. βου, augmentative; λιμός, hunger).

An abnormal increase in the desire for food. It may be seen in diabetes, in hysteria, in idiocy, and in dementia. (See **Appetite**, p. 36.)

**CACHEXIA** (Gr. κακός, bad; ἕξις, a condition).

A weakened or depraved condition of the body, produced by a variety of diseases or by inanition. The condition is familiar as a result of cancer, tuberculosis, syphilis, anæmia, lead-poisoning, etc.

**CALMETTE'S TEST.**

Of a 0.5 per cent. solution of tuberculin, one drop is placed in the conjunctival sac at its inner part. If the patient be the subject of tuberculosis, the reaction occurs inside six hours, and consists of congestion and œdema of the conjunctiva, swelling,

redness, and perhaps a purulent discharge from the lachrymal caruncle. The test is found to be positive in most cases of obvious tuberculosis and in a fair proportion of individuals in whom other signs of tuberculosis are not to be discovered, but in whom possibly a tubercular lesion may exist. Calmette, however, states that the reaction indicates the existence of a focus of tubercle actually in evolution or incompletely cured. He admits that it fails with old caseated lesions, in old caecetic tuberculous patients, and in very extensive or virulent lesions. In no case of disease or injury of the eye or of the lids should this test be used.

### **CAPUT MEDUSÆ.**

An enlargement of the superficial veins of the abdominal wall, arranged in a more or less radiating fashion, with the umbilicus as a centre, is known as the caput Medusæ. This is due to obstruction in the portal outflow, whereby the tension in that vein is raised; the small veins in the round ligament, which form a communication between the superficial veins at the umbilicus and the portal veins, are thereby dilated, and the veins in the skin share in the enlargement. This form of enlargement of the abdominal veins—a comparatively infrequent occurrence—is seen in cirrhosis of the liver, where a long-standing hindrance to the return of blood from the portal distribution exists; it may occasionally be found in cases of heart disease (see p. 5).

### **CAPUT QUADRATUM.**

In rickets the head may assume a square shape, owing to bony overgrowth of the parietal and frontal prominences. The coronal and sagittal sutures, lying between the prominences, form grooves crossing the vertex. The enlarged prominences are known as *Parrot's nodes*.

### **CARDIAC DULNESS, Area of Superficial and of Relative.**

The superficial area of absolute cardiac dulness is the triangular portion of the chest's surface, bounded mesially by the left side of the sternum from the fourth to the sixth costal cartilage. The outer border of this area is a line drawn from the junction of the fourth left costal cartilage with the sternum, out in a curving line (convexity of the curve toward the left shoulder) to the outermost

limit of the apex-beat; the base of the area is a horizontal line drawn from the apex-beat to the inner end of the sixth left costal cartilage. Behind this area the pericardial sac lies directly, without the interposition of lung tissue, and therefore the percussion-sound is void of resonance. Outside this triangle there is an encircling area of relative dullness, behind which lies the heart, covered with the thinned-out borders of either lung. This area of relative dullness extends to the right about one finger's breadth beyond the right border of the sternum; upwards and to the left it extends one or two fingers' breadth beyond the area of absolute dullness.

The extent of the area of præcordial dullness varies from the above limits in disease; it may be diminished or abolished in emphysema, and increased or displaced in heart disease, pleural effusions, fibroid changes in the lung, etc. (see p. 459 and Fig. 67).

**CARDIALGIA.** See Heartburn, p. 162.

**CARDIO-HEPATIC ANGLE** is the angle formed on the anterior surface of the chest by the junction of a line representing the right border of the area of superficial cardiac dullness with another line marking the upper limit of absolute hepatic dullness. Normally these lines form a right angle, or less. In cases of pericardial effusion this angle becomes obtuse, by a sloping toward the right of the cardiac dullness, the fifth right interspace near the sternum losing its resonance (Rotch's sign, see pp. 353 and 461).

**CARPHOLOGY (Floccitation)** (Gr. *κάρφος*, chaff; *λέγειν*, to collect).

In conditions of severe prostration, in which a fatal termination is not far off, the patient makes movements as if he were picking up or grasping at imaginary objects. This may take the form of picking at the bed-clothes (floccitation or floccillation; from *floccus*, a lock of wool). (See Delirium, p. 121.)

### CASE-TAKING.

In order to arrive at a just estimate of the patient's condition, a methodical, and to some extent routine system of examining

the patient and recording the observations thus made should be adopted. For a student such a method is absolutely necessary, not only as a means of acquiring the necessary data upon which to form a diagnosis, but also as an educational process; and it is only after long practice that the physician can safely permit himself to omit those portions of the examination which his experience tells him he may dispense with.

The following is a brief outline of the essentials in taking notes of a medical case, but the student is strongly recommended to give this subject the utmost attention, as upon careful and methodical note-taking the capacity to form a well-considered diagnosis mainly rests. This is best accomplished by unremitting practice in the hospital wards, but much help may be gained by consulting a more detailed treatise on the theory of case-taking and physical examination.

A. The notes commence by stating the name, address, age, and occupation of the patient, and the date on which he was first seen, or, in case of hospital patients, the date of his admission to the wards or out-patient department.

B. From the patient, or from his friends, information is sought on the following points:

1. **Family History.**—Evidence as to the length of life of the different members of his family, and their freedom or otherwise from diseases which are known to exercise an hereditary influence—*e.g.*, tuberculosis in all its forms, diseases of the nervous system, cancer, syphilis, gout, rheumatism.

2. **Personal History.**—This is an account of all incidents and conditions in his body or surroundings which might influence his health—*e.g.*, his previous illnesses, his habits, and the nature and environment of his occupation. In cases of females the history of the reproductive system should be inquired into.

3. **History of the Present Affection.**—This includes the mode of onset, the dates upon which he first fell ill, left off work, or went to bed, and any cause which he can assign for the complaint. In brief, **how**, **when**, and **why** he has been suffering.

C. The patient is next questioned as to his **present condition**. Any pain or discomfort he may be suffering is to be noted, as well as any departure from health which he himself has observed.

The note-taker then proceeds to observe for himself the present state of the patient.

1. **A general survey** of his condition is first carried out, his pulse, temperature, respiration rate, and tongue being investigated; his aspect, posture, condition as to bodily and mental strength are observed. Lesions of the skin, hair, and nails are now to be noted.
  2. **Circulatory System.**—Examination of the heart, its vessels, and blood.
  3. **Respiratory System.**—The lungs are to be examined by the various means at our disposal (inspection, palpation, percussion, auscultation, etc.), and all abnormalities noted. These include changes in the shape, size, movements of the chest, in vocal thrill, in resonance; modification of the voice-sounds and breath-sounds; the presence of adventitious sounds and abnormal conditions of the sputum.
  4. The **Blood** in many cases is to be examined, especially as to the quantity and condition of the red and the white cells, and as to the amount of hæmoglobin present.
  5. The **Glandular System.**—Lymphatic glands, thyroid, spleen.
  6. The **Abdomen**, including the alimentary system and the pelvic organs.
  7. The **Urinary System.**
  8. The **Nervous System.**—Disturbances of the mental functions; defects of motility; sensory disorders; reflex disturbances; trophic and vasomotor disorders; electrical reactions.
  9. **Organs of Special Senses**, including examination of the eyes, ears, nose, fauces, larynx.
  10. **Locomotor System.**—Condition of the bones, joints, and muscles.
- D. The **treatment** adopted is to be noted, and records are to be made at short intervals of the **progress of the case**. The **result** is to be stated. In case of death, should a **post-mortem examination** have been made, the particulars of the condition found should be recorded.

## CAVERNOUS BREATHING.

A low-pitched bronchical breathing, with a reverberating quality, heard on listening over a large air cavity (see p. 412).

**CHARCOT-LEYDEN CRYSTALS.** See *Sputum*, p. 388.

**CHARCOT'S JOINT.**

This condition, which is to be looked upon as a symptom of disease in the nervous system rather than as a disease itself, is a rarefying osteitis affecting the ends of the bones entering into the formation of the affected joint. At the same time a tendency to overgrowth may be observed in the form of exostoses; fractures may occur into the joint, spontaneous or the result of slight injury, giving the characteristic features of the arthropathy. The clinical aspects and significance of Charcot's joint are discussed in the article on *Trophic Disturbances* (p. 494).

**CHEYNE-STOKES RESPIRATION.**

A periodical cessation of breathing, with a gradual and increasingly energetic return of the respiratory act. It is usually an indication of serious disease, and may occur in cerebral and renal affection. (See *Dyspnœa*, p. 130.)

**CHVOSTEK'S SIGN.**

A symptom of tetany. In this affection a tap over the facial nerve (best on or just below the zygoma) causes a spasmodic contraction of the facial muscles. An exaggeration of the excitability of the motor nerves generally is recognized in this affection.

**CLAVICULAR REGIONS.**

That portion of the thorax covered by the clavicle on each side. (See the articles on the *Shape*, etc., of the *Thorax*, p. 464; *Percussion*, p. 450; *Auscultation*, p. 407; and *Pain*, p. 270.)

**CLAW-HAND (Main-en-griffe).** See *Contracture*, p. 105.

**CLONIC SPASM.**

Involuntary increase of muscular contractility of a region of the body may take the form of a persistent or tonic spasm; or it may, on the other hand, be an intermittent or clonic spasm. The latter is usually the result of irritation of the cerebral cortex, and is the result of disease and injury of that structure, as well as



of toxic blood states which give rise to that irritation. The subject is considered in article on Increased Muscular Action, p. 252.

**COG-WHEEL BREATHING (Respiration saccadée).**

A jerky, intermittent, vesicular breath-sound, usually observed during inspiration only. It is sometimes found in bronchial catarrh, and in early or late phthisis; it may also be due to mere fatigue or to muscular weakness (see p. 412).

**COIN SOUND.** See **Bell Sound**, p. 56.

**COLLIQUATIVE DIARRHŒA** (L. *colliquesco*, to become liquid, to melt).

A profuse and copious watery diarrhœa, seen especially in cases of advanced phthisis.

**COLLIQUATIVE PERSPIRATION.**

The term is applied to profuse and copious sweating from any cause. (See **Sweating**, p. 401.)

**COLOUR INDEX.**

The subject is considered in the articles on **Blood Examination** (p. 64), and **Anæmia** (p. 25).

**COMA.** See **Unconsciousness**, p. 498.

**COMA VIGIL.**

In some cases of low delirium the patient lies with eyes open, apparently awake, and muttering disconnected or meaningless words. He is really quite unconscious, and the condition is therefore known as *coma vigil*, or wakeful unconsciousness. (See **Delirium**, p. 121.)

**CONJUGATE DEVIATION.**

A deviation of both eyes to either side, the visual axes retaining their normal relations to each other; occurs in certain intracranial affections. The lesion must injure the sixth nerve-centre in the pons and the association fibres from it to the third, or else the

cortical fibres proceeding to those centres (see p. 241 and Fig. 38).

### CONSONATING RÂLES.

Any variety of râle may acquire a clear, bright, resonating character, from the proximity of highly-conducting solidified lung in pneumonia or phthisis; the sounds are then spoken of as consonating râles. By some writers the term crepitant râle is used as a synonym for consonating râle; there are objections to the use of the former term employed in this sense (see p. 420).

**CONSTIPATION.** See *Defæcation*, p. 117.

### CONTRACTURES.

The fixation of a joint owing to contraction of the muscles which normally control the movements of that joint is termed a contracture. It occurs in two forms, the active and the passive contracture.

**Active contractures** result from an excessive tonicity of the muscles involved; the arms are flexed, but the legs tend to assume a position of extension. The condition may be a symptom of hysteria, but is often due to disease in the spinal cord, which has an exalting effect on the reflexes—*e.g.*, descending degeneration of the pyramidal tract or tracts, as a consequence of a lesion in the motor tract higher up—cerebral hæmorrhage, transverse interruption of the spine, etc. (see p. 209). Interruption of the inhibitory impulses from the brain to the reflex arc is the cause, according to most observers, of the increased reflex tone of the muscles which obtains in contractures (see *Reflexes*, p. 334); but active contracture only occurs when to the interruption in the upper segment of the motor tract is added descending degeneration of the lateral columns. That active contracture is due to increased muscular contractility, and not to an actual shortening of the muscles or fibrous adhesions, is shown by the fact that this form of contracture disappears under a general anæsthetic, and diminishes in a hot bath.

**Passive contractures** are due to the actual shortening of the muscle, which is brought about in one of two ways: A muscle or group of muscles being rendered powerless, the opposing muscles contract more extensively than is their custom when opposed by active muscles; the limb is therefore drawn toward the healthy

muscles, and in time becomes fixed in that position, owing to permanent shortening of the active muscles. This is what occurs when a limb has been retained in a splint for a considerable time, or when a localized paralysis destroys the contractility of certain muscles, as may be seen in cases of lesions of the lower motor neuron. Familiar examples are infantile para'lysis, progressive muscular atrophy, and lesions of the motor nerves. Another type of passive contracture is due to shortening of a muscle which has been paralysed and is undergoing degenerative and cicatricial changes. In this case the fibrous contraction in the muscle gradually overcomes the healthy but weakened muscles, and draws the limb in the direction of the affected muscles. Very similar in its results is the late rigidity affecting paralysed muscles, which is really an instance of overaction of the muscles that are beyond control, and is due to increased reflex action; it is therefore an instance of active contracture, which draws the limb in the direction of the affected muscles.

It will be seen that many varieties of contracture may occur, and this is especially the case in those of hysterical origin; the latter may closely simulate the contractures of one or more of the forms of paralysis—*e.g.*, of hemiplegia, of monoplegia, of paraplegia, of smaller groups of muscles. Inconsistency and incompatibility of the contractures, together with other symptoms of the neurosis, may be sufficient to distinguish the condition.

A few distinctive forms of contracture may be mentioned:

**Facial Contracture.**—Cases of facial paralysis in which the loss of power is persistent, but not complete, undergo a change in their condition owing to secondary rigidity or overaction. The previously smoothed and expressionless half of the face becomes contracted, and its naso-labial fold again becomes distinct; the mouth may be drawn somewhat over toward the paralysed side; voluntary movements are, however, still defective.

**Claw-hand (main-en-griffe).**—Flexion of the two distal phalanges of the fingers, and hyperextension of the metacarpophalangeal joints, with wasting of the interossei. It is typically seen in paralysis of the ulnar nerve (see p. 229), and is also found in progressive muscular atrophy, pachymeningitis cervicalis hypertrophica.

**Ape-hand.**—Inability to oppose the thumb, which lies extended beside the fingers. Due to paralysis of the median nerve.

**Talipes, or Club-foot.**—This is often a congenital deformity,

but many cases are of the nature of contractures, being of paralytic origin. The varieties most commonly observed are: **talipes equinus**, which is almost always an acquired deformity due to paralysis of the extensor muscles of the leg from infantile paralysis or lesion of the anterior tibial nerve; **talipes equino-varus**, from paralysis of the extensor and peroneal muscles (infantile paralysis usually), with shortening of the opposing muscles; **talipes calcaneus**, a rarer form, from paralysis of the calf muscles, also an effect of infantile paralysis, as a rule; **talipes valgus**, from paralysis of the tibial muscles, is less infrequently seen as an acquired contracture.

### CONVULSIONS.

By this term is understood an excessive and involuntary contraction of the limbs, face, and trunk, combined with loss of consciousness. The muscular contractions, or **spasms**, may be either persistent (tonic) or intermittent (clonic), and it is the clonic spasms which most commonly occur in convulsive attacks. The conditions in which this symptom may be observed are the following: Epilepsy, of the idiopathic and Jacksonian forms; meningitis; cerebral hæmorrhage; hysterical convulsions; infantile convulsions; convulsive tic; toxic blood states—*e.g.*, tetanus, strychnine-poisoning, uræmia, diabetes. The subject is further considered in the article on excessive muscular contractility (Movement Increased), p. 252.

### CORNEAL REFLEX.

A light touch on the cornea is enough to cause involuntary closure of the eye. (See Reflexes, p. 344.)

### COUGH.

Coughing is almost invariably a reflex act; it may occasionally be a neurotic or volitional act. The stimulus originates in the vast majority of cases in some region of the respiratory passages between the vocal cords and the terminal air vesicles; the mucous membrane between the arytenoid cartilages and that at the bifurcation of the trachea are probably the most sensitive spots, while irritation of the respiratory surfaces above the vocal cords more commonly gives rise to vomiting, retching, or sneezing. Pleural irritation does not, as a rule, cause much coughing unless there is

an accompanying bronchial disturbance. Stimulation of other peripheral regions may exceptionally produce cough; thus cough as a result of irritation of the external auditory meatus is a well-recognized phenomenon, the afferent impulses being conveyed by the auricular branch of the vagus. Enlargement of the tonsils, an elongated uvula, postnasal adenoids, rhinitis, nasal polypi, and excessive mucous secretion from these regions, may be the cause of cough; irritation of the skin, as by cold air, may also give rise to cough. The popular 'stomach cough' is probably a pharyngeal or a diaphragmatic cough; it is, however, possible that afferent impulses via the vagus nerve from the stomach may provoke the cough reflex. Obviously the object of cough arising from the respiratory apparatus is to rid the affected region of some source of irritation, and it is therefore in many cases a useful and necessary act. The readiness with which the cough responds to the peripheral stimulus depends on the intensity of the stimulus, the sensitiveness of the region stimulated, and the activity of the reflex. In certain individuals or in certain conditions of the bodily health, particularly in neurotic states, the readiness to respond with a cough to peripheral irritation is exaggerated, and a very slight, and perhaps unrecognized, source of irritation may suffice to produce the effect. This is probably the origin of the so-called hysterical cough, which is often regarded as an example of cough of central origin. Like most other hysterical phenomena which are almost always 'founded on fact,' there is a definite, though perhaps trivial, cause for the hysterical cough; the quality of the latter is influenced and its characteristics are modified by the hysterical condition, but the cough is not entirely due to hysteria.

A defective response, in the form of cough, to the peripheral irritation of the respiratory tract is in some cases due to deficient sensitiveness of the reflex—*e.g.*, in respiratory diseases accompanied by weakness, depression, or prostration the cough is sometimes absent or feeble. This may be seen in cases of intense pneumonic toxæmia, and in respiratory diseases occurring in patients debilitated by disease or drink.

The cause of such irritable stimuli in the respiratory passages is in the majority of cases either an inflamed or congested condition of the mucous membrane or an excessive quantity of mucous secretion. Less frequent sources are: hæmorrhage, either into the bronchi or extravasated into the parenchyma; inflammatory

or necrotic processes in the lungs (pneumonia, phthisis); ulceration or foreign body in the larynx or trachea; external pressure of the enlarged thyroid gland; enlarged lymphatic glands in the neck or in the mediastinum; aneurism of the aorta or of its larger branches; mediastinal new growths. Irritation of the recurrent laryngeal nerves, the left by aneurism of the transverse arch of the aorta, the right by aneurism of the right subclavian artery, by

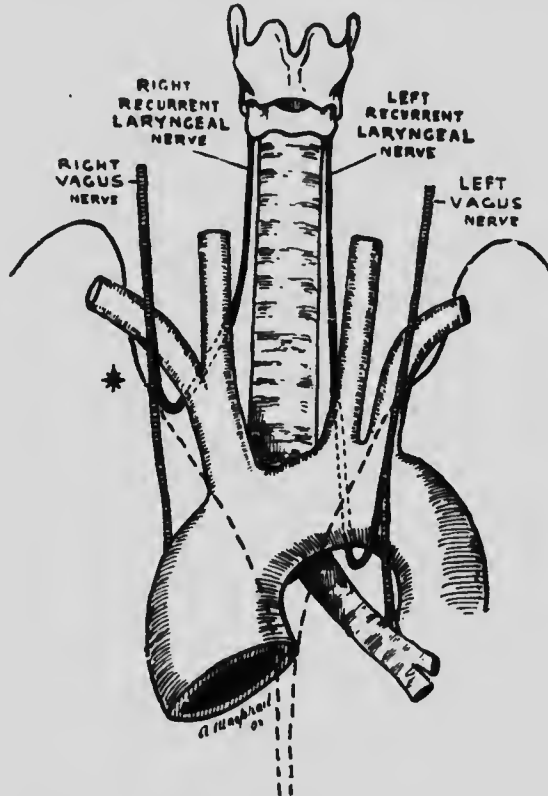


FIG. 21.—RELATIONS OF THE RECURRENT LARYNGEAL NERVES TO THE VESSELS OF THE THORAX (MONRO).

disease of the pleura, or by enlarged glands, may also give rise to cough by causing spasm of the vocal cords, or by provoking afferent stimuli in the coughing reflex arc (see Fig. 21).

The character of the cough varies with the cause and to some extent with the general state of the health. The most obvious distinction in the quality of the cough is that between the moist or loose cough and the dry or hard cough. The former indicates

the successful removal of mucus from the air passages, even though it may not be followed by expectoration; for it is not uncommon in adults, and it is the rule in children, that the secretion coughed up is immediately swallowed. The loose cough is therefore found in tracheal and bronchial catarrh, in excavating phthisis, and in resolving pneumonia. The dry cough indicates the presence of an irritant which the cough is unable to remove from the air passages; it may be tenacious mucus adhering to the surface of the membrane, ulceration, congestion, foreign bodies, pressure of tumours, etc., as mentioned above. The hard cough of the early stages of bronchial catarrh, while the mucous membrane is in an irritable, congested condition, is followed by the loose cough consequent on the appearance of a less viscid secretion.

A short, weak, frequently repeated, dry cough is that known as the **hacking cough**; the patient is often unaware of its presence, and it may become habitual. It is due to a mild irritation, generally in the upper air passages—*e.g.*, chronic catarrh of the trachea, larynx, or pharynx. It is frequently one of the earliest signs in phthisis.

Various grades of **harshness, hoarseness, hardness, or metallic quality** are to be observed in cases of irritation situated in the larynx. The hoarse, croaking, 'croupy' cough of laryngitis is familiar, but may be indistinguishable from that of diphtheria or foreign body in the larynx; the swelling of laryngitis often causes the cough to have a **barking** rather than a hoarse quality. Hysterical cough is also, as a rule, of a similar barking quality, and may be very loud and insistent. A similar type of cough often occurs at puberty in nervous children. An extreme grade of hoarseness, amounting finally to loss of tone in the cough, which has then a harsh, whispering character, is heard in cases where the vocal cords are ulcerated (syphilis, phthisis). The metallic ringing quality of cough may be heard as a result of any laryngeal irritation, provided the vocal cords are not too much damaged to vibrate freely; thus it may signify laryngitis, diphtheria, tubercular or syphilitic disease of the larynx before the ulceration has advanced, particles of food or other foreign body in the larynx, spasm of the cords due to irritation of the recurrent laryngeal nerve or nerves, by tumours, enlarged glands, abscess in connection with caries of the spine, and aneurism of the transverse arch of the aorta or of the subclavian artery. The

direct pressure of these structures on the trachea and bronchi also gives rise to cough (as already mentioned), but it is of a more spasmodic, paroxysmal character, and less metallic than that of laryngeal irritation.

**Paroxysmal cough** occurs in the conditions just mentioned, but it is most typically found in whooping-cough, which, besides being paroxysmal, presents the characteristic inspiratory crowing 'whoop'; it is also present in bronchiectasis, gangrene of the lung, and excavating phthisis. A night cough, paroxysmal in character, resembling whooping-cough in character, but not occurring, as a rule, during the day, and usually observed in children, is due to enlarged tonsils, adenoids, or other irritation in the nose and naso-pharynx.

A more **persistent cough** is observed in those cases where the irritation is constant and fairly acute; thus it is almost ceaseless in pneumonia, acute bronchitis, and laryngitis.

A cough **occurring at intervals** of variable length often originates in the accumulation of secretion in cases of mild bronchitis. If the catarrh is but slight, the cough may only appear in the morning, after the secretion has accumulated during the sleeping hours; if rather more severe, the irritation caused by the accumulating mucus wakens the patient at intervals during the night, and he coughs occasionally by day.

The patient may be **unable to cough**, or may only be capable of emitting a weak sound and soft expulsion. This may be the result of abdominal distension preventing the proper descent of the diaphragm, so that a sufficient preparatory inspiration cannot be taken; it may be due to pleurisy, especially of the diaphragm, which also impedes respiratory movements; destruction of the vocal cords by ulceration abolishes cough; total bilateral paralysis of the vocal cords, or total paralysis of one cord, renders coughing impossible; bodily weakness, paralysis of the muscles of respiration (advanced progressive muscular atrophy), or grave prostration and depression in the course of pulmonary disease abolish cough. The cessation of a previously active cough and expectoration in a case of lung disease which does not show other signs of distinct improvement is of ominous significance.

While cough is undoubtedly the most constant symptom of affections of the respiratory tract, its absence is occasionally noted even in cases of serious lung disease; as stated above, it may be absent owing to prostration, and it is often absent in cases of



early phthisis, though some of the cases which are reported by themselves, or even by their friends, as free from cough may have the slight hacking cough referred to above.

**Summary.**—Cough usually arises by reflex action in consequence of a stimulus applied to some portion of the respiratory tract.

Hysterical cough is an ordinary reflex cough arising from trivial causes in a neurotic subject.

A loose cough is a successful and fruitful cough; a hard or dry cough accomplishes nothing.

A harsh, hoarse, or metallic quality of cough points to the larynx as the seat of the irritation.

A paroxysmal cough is also the result of laryngeal irritation, or may be due to the pressure of tumours.

Coughing at intervals indicates that the irritating mucus is scanty in quantity, and requires some time to accumulate sufficiently to cause the cough.

Inability to cough may result from abdominal distension, from pleurisy, from paralysis of the vocal cords, from paralysis of muscles of respiration, and from grave prostration.

The cessation of a previously active cough and expectoration in a case of lung disease, which does not show other signs of distinct improvement, is of ominous significance.

### CRACKED-POT SOUND (Bruit de Pot fêlé).

On percussing over a pulmonary cavity which communicates with a bronchus, the sound elicited may be observed to have an indefinite metallic or *chinking* quality, such as may be produced by striking the closed hand filled with coins which have barely room to move, or by clasping the hands so that a little air is imprisoned between the palms, then smartly striking the back of the hand upon the knee so as to drive the air from between them. This 'cracked-pot sound' is due to the sudden expulsion of air under pressure from the cavity and through the narrow orifice of the bronchus.

While in most cases it is, if not pathognomonic, at least corroborative of a phthisical cavity, it may also occur under normal circumstances. It is often heard on percussing the chest of a healthy infant while crying; it may be heard on forcibly percussing an adult at the upper part of the thorax near to the

trachea while he is speaking, and if his chest-walls happen to be very yielding. In both these instances the air is being suddenly driven from the chest by the stroke through the glottis, which is narrowed for the purpose of phonation (see p. 462).

**CRANIOTABES** (*L. cranium*, the skull; *tabes*, wasting).

In cases of rickets or of syphilis, portions of the skull-bones in young children may be felt to be thinned and softened, so that on pressure they may be easily indented, and give a gentle crackling feeling. The condition is most likely to occur in the occipital or parietal bones.

**CREMASTER REFLEX.**

The skin on the inner side of the thigh is stroked or pinched, with the result that the testicle of that side is retracted. (See Superficial Reflexes, p. 343.)

**CREPITANT RÂLES** (Crepitation).

The finest variety of râle, or crackling sound, to be heard on auscultation of the thorax in the earliest stages of pneumonia, also in the stage of resolution of the same disease, in œdema of the lung, in hæmorrhagic infarction. The sound is said by some authorities to be an ill-defined friction sound. The use of this term as a synonym for consonating râles should be avoided (see p. 418).

**CRISIS** (*Gr. κρίσις*, the issue).

In certain fevers the temperature falls abruptly (within twenty-four to thirty-six hours) to or below normal. The sudden defervescence is accompanied by a sense of improvement, and the patient's condition may be observed to have been relieved. Thus his pulse is less frequent and is stronger, he perspires, the quantity of urine passed increases, and his respirations become less hurried.

This type of termination of a fever is less common than the more gradual defervescence (lysis), and may be seen in lobar pneumonia, measles, chicken-pox, malaria, tonsillitis, relapsing fever.

**Pseudo-crisis** is the premature fall of temperature which some-

times occurs in the course of these affections, and especially in pneumonia. After remaining near normal for a short period the temperature again rises, and may finally disappear in due time. (See *Temperature of the Body*, p. 404.)

The word is also employed in quite a different sense, as signifying the onset of a disturbance in the functions of a patient suffering from spinal disease. It is almost invariably in locomotor ataxia that the symptom is seen. The commonest form for it to assume is the **gastric crisis**, an attack of vomiting and abdominal pain. **Laryngeal crises**, **cardiac crises**, **rectal crises**, occur less frequently in the same disease.

**Blood crisis** indicates the appearance of a large number of nucleated red cells in the blood, usually in a case of pernicious anemia. (See *Blood Examination*, p. 67.)

### CROSSED-LEG GAIT.

In certain diseases of the spinal cord, owing to a spastic condition of the muscles of the legs, and of the adductors in particular, the legs are crossed in front of each other at each step. (See *Gait*, p. 149.)

### CROSSED PARALYSIS (Alternate Paralysis).

It may occur that in a case of hemiplegia the limbs of one side of the body are paralysed, while the face or eye muscles of the opposite side have lost power. This results from a lesion interrupting the fibres proceeding from the cortex down to the ganglion cells in the anterior horns. The fibres have not yet crossed over to the opposite side, so the paralysis is in the limb opposite to the lesion. At the same time, should the injury be situated in the medulla, pons, or crura, it is probable that not only will the supranuclear fibres be damaged, but the nerve cells in the various nuclear groups to be found in those regions will also be injured; in the latter case the paralysed muscles will, of course, be on the same side of the body as the lesion. The occurrence of paralysis of certain of the muscles supplied by cranial nerves on one side of the body, with an opposite-sided paralysis of the limbs, is then highly suggestive of a lesion of the pons, of the medulla, or of the crus. (See the article on *Decreased Movement*, p. 239.)

**CURSCHMANN'S SPIRALS.** See *Sputum*, p. 388.

**CYANOSIS** (Gr. *κράσις*, blue).

A bluish discoloration of the skin, seen in various shades from leaden white to purple. It is due to defective oxidation of the blood, which is consequently poor in oxygen and dark in colour. In addition, the imperfect oxygenation is usually associated with obstruction to the venous return to the heart, so that the veins generally become enlarged and the skin contains more blood than normal. The blue tint is most marked, as a rule, in distant regions (*e.g.*, hands or feet), where venous stasis is at its worst; it is also very pronounced in places where the skin is thin, such as the lips, cheeks, nose, or ears.

The conditions which give rise to the defective oxygenation may be grouped into the following classes :

**A. Respiratory Causes.**

(a) Obstruction to the entrance of air into the lungs. The most important causes of this condition are: (i.) pharyngeal obstruction (foreign bodies, tumours); (ii.) laryngeal obstruction (œdema, laryngitis, diphtheria, tumours, foreign bodies, stenosis, paralysis, or spasm of the vocal cords); (iii.) tracheal obstruction (foreign bodies, pressure of tumours or aneurism, stragulation); (iv.) obstruction of the bronchi (foreign bodies, bronchitis, asthma).

(b) Conditions in which the amount of lung tissue available for aeration is diminished—*e.g.*, phthisis, pulmonary embolism and infarction, atelectasis, œdema and passive congestion of the lungs, pneumonia, capillary bronchitis, emphysema, pneumothorax, pleural effusion, mediastinal and other thoracic tumours causing pressure.

(c) Conditions which diminish the activity of respiration: paralysis of the muscles (bulbar paralysis, diphtheritic paralysis, progressive muscular atrophy); spasm of the muscles (tetanus, strychnine-poisoning, epilepsy); severe pain in the chest (pleurisy, fractured ribs).

(d) Incompetence or stenosis of the mitral orifice of the heart, even when compensation is fairly well established, may give rise to cyanosis, owing to high tension in the pulmonary circulation; this causes changes in the lung tissue (brown induration, bronchial catarrh), which interfere with the efficient aeration of the blood.

### B. Circulatory Causes.

(a) Valvular affections in which compensation has failed or has never been established. Here the defective aeration is not due to pulmonary disease, but to inability on the part of the organs of circulation to pass the requisite amount of blood through the lungs.

(b) Weakness of the heart muscle, especially of the right side. This may be observed in the valvular affections just mentioned; in the myocardial changes occurring in the course of acute disease (*e.g.*, typhoid fever, pneumonia, scarlet fever); in fatty degeneration of the heart consequent on anæmia or arteriosclerosis; in dilatation of the heart from strain or in advanced kidney disease; in pericarditis.

(c) Vaso-motor changes may cause cyanosis—*e.g.*, the bluish colour of paralysed limbs. The same condition occurs in hysteria. The cyanosis due to cold is an example of vaso-motor disturbance, as is also the condition known as Raynaud's disease.

(d) An intense form of cyanosis is that termed 'morbus cæruleus' seen in children, and due to congenital defects in the heart. It is due not only to valvular disease, but may also be the result of the mixture of arterial and venous blood; this occurs in those cases in which there is a defective interauricular or inter-ventricular septum.

(e) Certain poisons, especially some of the coal-tar derivatives (antipyrine, antifebrin), cause a darkened colour of the skin, due to the formation of methæmoglobin in the blood.

(f) The pressure of tumours in the mediastinum may impede the venous return through the venæ cavæ, and so give rise to cyanosis. Pressure on venous trunks elsewhere may produce localized areas of cyanosis—*e.g.*, cyanosis of the face from pressure on the veins of the neck.

### CYCLOPLEGIA.

Defective or lost power of the ciliary muscle to contract, shown by inability to see near objects distinctly. It may be due to the action of poisons, such as the diphtheria toxin, or certain drugs; or it may occur as part of a more general oculo-motor paralysis (see p. 215).

### CYTODIAGNOSIS.

The study of the cells contained in inflammatory exudates or circulatory transudates has of late years occupied the attention of

observers, and has already in the opinion of many yielded useful results. It has been chiefly the pleural effusions which have proved instructive, but investigations as to the cellular constituents of fluids in most of the serous cavities have been carried out. The name 'cytodiagnosis' has been applied to the method, which may be briefly described as follows :

The fluid is withdrawn from the pleural cavity with the usual aseptic precautions, and placed in a sterilized tube ; if coagulated, it is well agitated and stirred until the clot has shrunk and can be removed. The fluid is thoroughly centrifugalized, and a stained film is prepared from the sediment.

According to Widal and other investigators, the following deductions may be made from the findings by this method :

(a) A predominance of lymphocytes indicates a tubercular effusion.

(b) A predominance of polymorphonuclear cells indicates an effusion of an acute infectious origin.

(c) A large number of endothelial cells indicates a mechanical effusion or transudate.

If the tubercular effusion be due, not to a primary pleural tuberculosis, but to phthisis, with secondary pleural involvement, the sediment is found to be largely composed of broken-down cells and detritus ; such cells as can be identified are mainly polymorphonuclears of inflammatory origin.

**DEAD FINGERS.** See *Asphyxia*, p. 48.

### DEFÆCATION.

Types of abnormal defæcation—Mechanism of the act—Effects of disease of the spinal cord—Constipation : causes ; symptoms.

Intestinal obstruction : causes—impaction of the bowel contents ; changes in the bowel wall ; volvulus and intussusception ; external pressure—Situation of the obstruction.

Diarrhœa : causes—Enumeration of forms of diarrhœa.

Departures from the normal regular evacuation of the bowels convey in many cases important information bearing on the diagnosis of the case. There may be a total inability to pass faeces (**intestinal obstruction**) ; the evacuations may be accomplished at unduly long intervals and with difficulty, the motions being hard and comparatively dry (**constipation**) ; the bowels may be too frequently moved, the act being often accompanied by

gripping pain in the abdomen, and the evacuations watery (*diarrhœa*); a sense of irritation and straining, with a desire to empty the bowel, which is not satisfied by the act (*tenesmus*); the motions may pass involuntarily from the patient (*incontinence of fæces*).

The act of defæcation, like that of micturition (see p. 195), is largely a reflex; the maintenance of the tone of the sphincter depends on the integrity of a reflex arc, whose central path lies in the lumbar enlargement of the spinal cord. It may be inhibited by volitional or reflex influences, and at the same time the peristaltic movements of the sigmoid flexure and rectum are active, while the abdominal muscles contract reflexly and voluntarily, the glottis being forcibly closed. By this means the contents of the rectum are pressed out through the relaxed sphincter. The reflex mechanism governing this act is less sensitive than that of micturition; hence it is less readily disturbed by diseases of the nervous system, and disturbances of the function of defæcation are less frequently an indication of nervous abnormalities than of affections of the alimentary canal itself. The main effect produced upon the action of defæcation by disease of the nervous system is incontinence of fæces. This may occur as a consequence of disease or injury of the lumbar cord, by which the central portion of the reflex arc maintaining the tone of the sphincter is destroyed. A lesion higher up in the cord may leave the sphincter reflex intact, but may block the inhibitory impulses, and may in addition impede the abdominal muscles in their expulsive efforts; the result will be, therefore, constipation or intestinal obstruction.

Three abnormalities of defæcation may now be separately considered—viz., constipation, intestinal obstruction, and diarrhœa.

**Constipation (Costiveness).**—The retention of the bowel contents for an unduly long period. Its causation is thus described by Monro: 'Constipation may result from deficiency of food, deficiency of intestinal secretion, or deficiency of action on the part of the intestinal muscle. The causes may be local or general. Among the local causes are obstruction of the bowel by disease of its wall; atony of the bowel, which, it is to be noted, is itself a result of habitual constipation; pressure on the intestine—e.g., by a tumour; and weakness of the abdominal muscles. The more acute or general causes include a family tendency; sedentary habits; errors of diet; lead-poisoning; functional and

structural diseases of the nervous system (hysteria, meningitis etc.); diseases of the stomach; and various acute fevers. With many people, especially women, constipation is habitual, and this is largely due to want of regularity in attending to the calls of nature. Constipation is sometimes troublesome in little children without obvious reason.'

Certain symptoms are frequently associated with, or in some cases directly due to, constipation. Chlorosis has been attributed to constipation (Clark), but the absence of constipation in many cases of chlorosis minimizes the probability of these conditions being cause and effect. Headache, flatulence, lassitude, a foul tongue and bad breath, loss of appetite, and mental depression, are symptoms which may, in part at least, be attributed to constipation, and in part to affections of the organs of digestion whereby the constipation is also caused. Chronic constipation may cause catarrh of the bowel, with many consequent disorders—*e.g.*, catarrhal jaundice, appendicitis, colitis, etc. It may give rise to atony of the intestine, with dilatation, fæcal accumulation and intestinal obstruction, ulceration, and in extreme cases perforation of the bowel and piles. Uterine displacements, dysmenorrhœa, and other pelvic disorders may also be fairly attributed to constipation.

**Intestinal Obstruction** differs from constipation in the fact that the muscle of the bowel may be active enough; there may be no deficiency in the intestinal secretion nor in the amount of food ingested; there is, however, some impediment to the free evacuation of the bowel, usually of a mechanical nature. There may be periodical passage, defective in quantity, but commonly there is either a complete cessation of defæcation or an irregular, frequent, scanty, watery, or bloody discharge from the bowel. In acute cases pain, vomiting, rapid pulse, and prostration are the most prominent symptoms; in chronic cases there may be merely constipation, or diarrhœa and constipation may alternate. Pain, distension of the bowels above the stricture, visible peristalsis of the intestine, the presence of scybala in one or more regions of the abdomen, and eventually fæcal vomiting and other symptoms of acute obstruction, are among the commonest features of chronic obstruction of the bowels.

The causes of the condition are to be found in (*a*) impaction of the bowel contents; (*b*) constriction in the lumen of the gut, owing to changes in the bowel wall; (*c*) twists or invagination of



portion of the bowel; (d) pressure of bands, hernial constrictions, tumours, etc., outside the bowel.

(a) **Impaction of the Bowel Contents** is frequently seen as a result of chronic constipation, the scybalous masses forming an accumulation, which produces obstruction. With this condition is sometimes found diarrhœa, due to the irritation of the impaction acting on the portion of bowel below the obstruction. This condition is commonest in elderly women, but may occur at any age. The impaction is occasionally composed of gall-stones; this is also commonest in elderly females. A rare cause is an accumulation of round worms, occurring in children; foreign bodies which have been swallowed (false teeth, fruit-stones, hair), or fœcal concretions (**coproliths**), may also be mentioned as infrequently occurring instances of obstruction from impaction.

(b) **Changes in the Bowel Wall**—Cicatrical contraction from healed ulceration (tubercular, syphilitic, dysenteric, simple); new growth in the bowel, commonest in the colon, and near its flexures. This often forms a hard, slowly-growing, and contracting ring, of but small bulk.

(c) **Volvulus**, a twist of a loop of bowel, is most likely to occur at the sigmoid flexure; the cæcum, or a portion of the small intestine, may, however, be the region affected. It is most likely to occur in at or beyond middle life, and gives rise to symptoms of obstruction. **Intussusception**—*i.e.*, the invagination of one portion of the gut into the part below—is caused by an excessive activity and irregularity of peristalsis. It occurs chiefly in children, and is commonest at the junction of the small with the large intestine, the ileo-cæcal valve being propelled into the large intestine. It gives rise to a sausage-shaped tumour, usually lying somewhat transversely across the middle of the abdomen, and its lower end may at times even extend so low down as to be palpable in the rectum. Acute obstruction is the result.

(d) **External Pressure from Bands, Hernial Constriction, Tumours, or Displaced Organs**, may give rise to either acute or chronic obstruction. The gut may be strangulated by slipping below a band of adhesion or Meckel's diverticulum, the tip of which has become fastened to an adjoining structure by inflammatory adhesion; a similar pressure may be exerted by an adherent appendix, or by the passage of the intestine through an abnormal opening in the omentum, mesentery, or abdominal walls. In all these conditions the symptoms will generally be those of acute obstruction.

Compression of the bowel by tumours and by enlarged or displaced organs occurs less frequently, and may cause acute or chronic obstruction.

**Situation of the Obstruction.**—Careful abdominal and rectal examination may disclose the nature of the stoppage: cancerous or other stricture of the rectum, intussusception, or fæcal impaction may be detected by digital examination of the rectum. A sense of resistance of the abdominal muscles over one area of the abdomen, visible peristalsis, tenderness, may all point to some region of the abdomen where the obstruction may be located. Careful examination of all the possible hernial apertures is of the utmost importance. The distended large bowel may often be discernible above the stricture; if this be near the lower end of the colon, the horseshoe shape of this bowel may be clearly defined, and its pouches may even be visible if the contractions of the gut be active and the abdominal walls thin. If the stoppage be at the neighbourhood of the ileo-cæcal junction, the swelling is chiefly observed in the umbilical and hypogastric regions, and when the bowel is contracting forcibly it may show up on the surface as a pattern of transverse or sloping bars (**the ladder pattern**). With the obstruction in this region, or higher in the intestine, vomiting occurs early, and is distinctly fæcal in character, while the urinary secretion is quickly diminished; collapse and prostration are not long delayed. Tenesmus and bloody or mucous stools, and a less acute type of obstructive symptoms, suggest the large intestine as the seat of the obstruction. The groin and umbilicus must always be thoroughly explored in view of the possibility of strangulated hernia.

**Diarrhœa.**—This is a symptom of a great variety of affections, chiefly, but not exclusively, of the alimentary tract. There is an unduly frequent and usually urgent call to stool; the motions are loose, and may be scanty with each motion, but at times are copious. It is caused by conditions the opposite to those which produce constipation—namely, increased activity of the peristaltic movements of the bowel, and increased copiousness of the intestinal secretion; both of these factors are at work in most cases of diarrhœa. Irritation of the lining of the bowel, from any cause, is the commonest origin of the condition: certain purgative drugs (*e.g.*, scammony, croton oil); bacterial and other poisons (*e.g.*, decomposing food); ulceration and catarrh of the intestine (*e.g.*, tubercular and dysenteric ulceration, typhoid inflammation, gastro-

enteritis); local irritation caused by faecal accumulation, new growths, or foreign bodies. The diarrhoea of amyloid disease, cirrhosis of the liver, uræmia, cholera, and that which occasionally occurs in the course of other infectious diseases, is the result of the toxic condition of the blood in those affections. A copious extraction of fluid from the bloodvessels is the cause of the looseness produced by concentrated saline solutions.

That the peristaltic activity, and possibly the secretory activity as well, can be stimulated by functional disturbances of the nervous system is obvious; examples are seen in the familiar tendency to diarrhoea among persons in a state of nervous suspense, and in hysterical subjects.

The following forms of diarrhoea may be enumerated:

The 'pea-soup diarrhoea' of typhoid fever.

Lienteric diarrhoea (Gr. *λείος*, smooth)—the passage of food but little changed by digestive action, as if it had simply passed through a smooth tube. It is chiefly observed in infants suffering from serious digestive disturbance; it is also found among adults who neglect to masticate their food properly, and who suffer from severe dyspepsia.

The 'rice-water diarrhoea' of cholera.

The 'meat-juice diarrhoea' of dysentery.

The 'colliquative diarrhoea' of advanced phthisis.

The 'straining diarrhoea' (tenesmus) of severe intestinal irritation.

## DELIRIUM.

The consciousness of the individual is disturbed and irritated, but not abolished; he exists in a dream-like condition, in which he is subject to hallucinations and illusions.

Two forms of delirium are noticed—a **low, quiet, or muttering** delirium, and a **noisy, active, or wild delirium**, which in extreme cases becomes maniacal.

Low delirium is observed in cases of serious and prostrating illness, as in the later stages of typhoid fever, in the second week of typhus fever, or in the typhoid state from any cause. This form of delirium is often accompanied by other signs of prostration—*e.g.*, **carphology** or **flocitation** (picking or plucking at the bedclothes); **subsultus tendinum**, a jerking of the tendons due to irregular muscular contractions; and **coma vigil**, a condition of apparent wakefulness, but really a muttering unconsciousness.

Active delirium is the form commonly seen in acute febrile states, in alcoholic delirium (delirium tremens), in that produced by belladonna or opium, in post-epileptic conditions, in hysteria, and in acute mania.

There may be an alternation of quiet and noisy delirium, the latter often supervening without warning on the muttering state.

In pneumonia the delirium frequently persists for some time after the critical fall of temperature, or may even first appear in the period following the crisis. The same late occurrence of delirium is at times noted in typhoid and other fevers.

**DELIRIUM CORDIS.** See *Palpitation*, p. 275, and *Arrhythmia*, p. 41.

**DESQUAMATION.** See *Skin Eruptions*, p. 362.

**DEVIATION, Primary and Secondary.**

Primary deviation is a departure of the visual axes from their normal relation one to the other; instead of meeting at the point of fixation, they may cross in front of or behind that point, or may not meet at all (squint).

Secondary deviation is the exaggeration of a paralytic squint observed on attempting to bring the paralysed muscle into action (see p. 217).

**DEXTROCARDIA.**

Transposition of the heart from the left to the right side of the thorax—a congenital malformation consistent with perfect health.

**DIARRHŒA** (Gr. *διά*, through; *ρέω*, to flow).

An unduly frequent evacuation of the bowels, the motions being liquid. (See *Defæcation*, p. 120.)

**DIAZO REACTION (Ehrlich).**

In some pathological conditions of the urine the red colour produced by the action of diazo compounds upon certain aromatic substances may be observed. This reaction may be regarded as an indication of abnormal decomposition of proteids, and is found in a variety of diseased conditions. It is nearly always found in

typhoid and typhus fever; very frequently in other fevers, such as scarlet fever, measles, diphtheria, pneumonia, phthisis, erysipelas; very rarely in meningitis and articular rheumatism. In non-febrile affections it is commonly absent, but may be observed in advanced cardiac disease, in cancer, especially of the pylorus, in leukaemia, in the cachexia of malaria, in cold abscesses; the reaction may also be obtained after the administration of certain drugs—*e.g.*, opium, heroin, carbolic acid, creosote, guaiacol, naphthalin, chrysarobin, etc.

The test is to be performed as follows: Prepare two solutions:

1. A 0.5 per cent. solution of sodium nitrite in water.
2. Sulphanilic acid .. .. . 0.5 gramme  
Hydrochloric acid .. .. . 5 c.c.  
Water .. .. . 100 c.c.

For each test shake up a couple of drachms of Solution 2 with two drops of Solution 1, added to an equal volume of urine in a test-tube; add an excess of ammonia, and the fluid turns a deep red colour. The froth on shaking the tube is a rosy-red colour. In normal urine the result of these reagents is to produce a brownish yellow colour.

### DIETL'S CRISES.

The term is applied to periodic attacks which may afflict persons suffering from movable or floating kidney. The symptoms are sickness, vomiting, severe pain in the abdomen, scantiness of urine, and possibly hæmaturia. The paroxysms are probably due to temporary strangulation of the pedicle of the abnormally movable organ, by twisting or kinking of the structures of which it is composed.

### DIPLEGIA (Gr. *δίς*, twice; *πληγή*, a stroke) (Cerebral Paraplegia; Double Hemiplegia).

Lesions occurring in the brain, interrupting the motor tract, usually cause paralysis of one side of the body only (hemiplegia), and that commonly on the opposite side of the body to the lesion. In some cases—*e.g.*, lesions of the crus, pons, or medulla—efferent fibres proceeding to both sides of the periphery may be damaged, causing a bilateral paralysis, which is therefore known by the terms diplegia, cerebral paraplegia, or double hemiplegia (see p. 243).

**DISSOCIATED ANÆSTHESIA (Dissociation).**

A symptom found chiefly in syringomyelia, in which affection the tactile sensibility is normal, but the patient may be insensitive to pain, heat, or cold. This is the consequence of the characteristic lesion of the disease—namely, the formation of cavities in the grey matter of the cord. The sensory paths for pain, heat, and cold are believed to traverse, in part at least, the grey matter, while the paths for common sensation are mainly in the posterior columns; the latter usually escape injury in this affection, hence the dissociation. It is the cervical and upper dorsal cord that is involved as a rule, and as a result the upper limbs are often the seat of this anomalous sensory condition.

A loss of power to distinguish heat from cold is occasionally observed in locomotor ataxia, multiple sclerosis, and hysteria.

**DITTRICH'S PLUGS.** See *Sputum*, p. 388.

**DOUBLE VISION (Diplopla)** (Gr. *διπλός*, double; *ὄψις*, sight).

In strabismus from any cause the faulty position of the visual axes results in the transmission to the visual centres of two separate images of the object looked at. These may or may not be perceived by the higher centres; in the former case two objects are seen instead of one. The varieties and causes of double vision are considered at p. 217.

**DROPPED FOOT.**

Inability to dorsally flex the foot or extend the toes on the dorsum first phalanges on the dorsum is produced by a lesion of the external popliteal nerve, a branch of the sciatic. The mode of walking is characteristic: the steppage gait (see p. 147). The nerve may be damaged by intrapelvic injury, or by neuritis from any cause (see p. 231).

**DROPPED WRIST (Dropped Hand).**

Paralysis of the extensors of the wrist and fingers in consequence of a lesion of the musculo-spiral nerve causes the hand to fall in a flexed position (see p. 258.)

**DULNESS.**

The sound produced by percussing some portion of the body which is inelastic, and which is not in immediate proximity to a

resounding air space, is of a dull, non-musical character. This is known as **absolute dulness**, as distinguished from the **relative dulness** elicited by a stroke over tissues in close proximity to air cavities. (See p. 281 and p. 451; also see Figs. 39 to 46.)

### DUROZIEZ'S DOUBLE MURMUR.

In some cases of aortic regurgitation carefully regulated pressure of the stethoscope over the femoral artery elicits a diastolic murmur, as well as the systolic murmur which one hears normally in pressing the stethoscope on any fairly large artery. (See Examination of Arteries, p. 43.)

### DYSPNOEA (Gr. *δύς*, bad, difficult; *πνέω*, to breathe).

Nervous mechanism of respiration—Meaning of the term 'dyspnœa'  
—Subjective dyspnœa—Circumstances under which it may be absent—Objective dyspnœa.

Frequent respiration, due to diminution in lung tissue, deficiency of hæmoglobin, excessive venosity of blood, impure and toxic blood, obstruction in the upper air passages, painful or paralytic conditions of the chest—Infrequent respiration.

Forcible respiration—Weakened respiration—Arrhythmical respiration—Inspiratory dyspnœa—Expiratory dyspnœa—Irregular breathing—Biot's respiration—Cheyne-Stokes respiration—Cyanosis—Stridor—Stertorous dyspnœa.

The act of respiration is presided over by a centre situated in the medulla, which is influenced by—(a) impressions reaching it from the periphery, and chiefly from the air vesicles. Distension of these evokes expiratory efforts, and their contraction calls for inspiratory movements; this reflex is an important factor in normal breathing. (b) The quality of the blood which circulates in the medulla influences the centre; blood containing a larger proportion of carbon dioxide than normal is a powerful stimulant to respiratory activity. (c) Stimulating or inhibiting influences reach the centre from the higher cerebral centres, whereby voluntary modifications of respirations are effected. A variety of organic diseases and functional disturbances interfere with the efficient performance of the act of respiration; this gives rise to certain changes in the type of breathing, generally in the direction of increased activity, accompanied as a rule, but not invariably, by sensations of respiratory discomfort on the part of the patient. The term 'dyspnœa' is employed to indicate such abnormalities in breathing as may occur in consequence of these disturbances,

and in that wide sense may be taken to comprise all departures from the normal type of breathing.

A distinction is usually drawn between those cases of dyspnœa in which the subject experiences distress, the so-called **subjective dyspnœa**, and those in which the signs of difficult breathing are obvious to the observer; the latter are known as **objective dyspnœa**. The diagnostic value of this distinction is slight, as the two types are very frequently combined, and subjective dyspnœa may be observed in conditions of such complete contrast as hysteria and advanced stages of pulmonary and cardiac disease.

**Subjective Dyspnœa.**—The sensation of 'shortness of breath' requires no description here. It may be produced in anyone by excessive and too rapid exercise, and denotes a need for more oxygen. In disease, even when the patient is at rest, the necessity for more air may assert itself. It usually accompanies the objective signs of dyspnœa to be mentioned presently, but may in chronic cases disappear as the organism grows accustomed to an imperfect aeration (*e.g.*, pneumothorax); or the increased respiratory efforts may suffice to meet the wants of the organs, in spite of the defect which gave rise to the dyspnœa (*e.g.*, moderate obstruction of the upper air passages); or the respiratory centre may become so dulled and the sensorium so benumbed (as may be observed in ante-mortem dyspnœa) that the abnormal respiratory action is unaccompanied by subjective dyspnœa.

The occurrence of the sense of want of air without some objective sign of dyspnœa is hardly conceivable. As soon as the patient experiences the oppressive sensation of breathlessness, he endeavours voluntarily or involuntarily to remove it by adding force or frequency to the respiratory acts, unless he be prevented by painful conditions of the breathing apparatus, or by mechanical impediments to the act of respiration. In hysteria and in nervous individuals there is often an undue preponderance of the subjective over the objective symptoms. The patient feels keenly a want of air, and expresses her feelings freely; there is, however, no cyanosis or other evidence of deficient aeration.

**Objective Dyspnœa.**—A variety of features and incidents are combined in greater or less profusion to make up the clinical picture of dyspnœa; different types of the disturbance of breathing may be seen, according to the prominence of one or other element in the symptom-complex, and a certain diagnostic infer-



ence is attached to the respective types of dyspnoea observed. The symptoms enumerated in the following types of disturbance of respiration may, occasionally singly, but more commonly in groups, form the evidence of dyspnoea :

1. **Increased Frequency of Respiration.**—This is by far the commonest form presented by dyspnoea, and is the simplest means at the disposal of the organism to correct deficient aeration, this being the cause of dyspnoea in almost every instance. Some authors, indeed, restrict the use of the term 'dyspnoea' to those cases of disturbed breathing in which respiration occurs too frequently; this, however, leaves out of consideration an important group. The cases, then, in which the respiratory acts occur too frequently are: (a) All those conditions in which the amount of lung tissue available for aeration is diminished—*e.g.*, phthisis, pulmonary embolism and infarction, atelectasis, œdema and passive congestion of the lungs, pneumonia, capillary bronchitis, pneumothorax, emphysema, pleural effusion, mediastinal and other thoracic tumours causing pressure. The pressure of abdominal tumours, ascites, and other causes of upward pressure may also diminish the area of lung surface, but the dyspnoea which so commonly results from these conditions is rather due to mechanical interference with the respiratory act. The blood state in these pulmonary affections has, however, to be reckoned with. (b) The blood may be insufficient in quantity or in hæmoglobin; the dyspnoea of chlorosis, pernicious anæmia, and that following hæmorrhage is to be referred to this cause. Rapid, sometimes shallow, but more commonly sighing and deep respirations are observed in these conditions. (c) Owing to non-compensated valvular disease of the heart, to pulmonary or other disease, the blood is imperfectly aerated; the blood which reaches the respiratory centre is, therefore, more of the venous quality than normal. Venous blood is a powerful stimulant to the centre. (d) Blood which has not been efficiently deputed, or which is of a higher temperature than normal, or which contains bacterial or other toxins, is probably an agent to increase the activity of the respiratory centre. Pneumonia may be included in this group, as well as in (a), (c), and (f); kidney affections, diabetes, and fevers of all descriptions also furnish instances of dyspnoea due to this cause. (e) Frequent respirations may be the result of obstruction in the upper air passages. Lesions of this nature are, as a rule, characterized by inspiratory dyspnoea, and often by

infrequent deep breathing (see below). (*f*) Shallow and defective breathing may result from painful conditions of the chest (pleurisy, pneumonia, pericarditis, wounds and fractures), or from paralysis of the intercostals and diaphragm; more frequent acts of respiration are therefore necessary to compensate for the deficiency.

It must be remembered in observing the patient that in many cases comprised in the six groups just mentioned the supply of blood-aeration is quite equal to the demand so long as the patient is at rest; it is only on attempting some act of bodily or emotional exercise that the objective or subjective signs of dyspnoea appear.

2. **Decreased Frequency of Respiration.**—This form of dyspnoea is much less commonly found than the foregoing; it may occur in obstruction of the upper air passages, inspiration being then prolonged, laboured, and noisy. At times it is observed in emphysema and asthma, but in these affections the expiratory act alone may be prolonged. A slow, sighing type of breathing is seen in the dyspnoea of shock, syncope, hæmorrhage, and in hysteria and other emotional states. The terminal dyspnoea in the dying is slow, irregular, and intermittent. Poisonous doses of opium, chloral, chloroform, and aconite produce slow breathing.

3. **Increased Force of Respiration.**—As a rule, slow breathing is deeper than normal; in many cases of rapid breathing, however, the breathing is energetic and deep—*e.g.*, the dyspnoea of anæmia, diabetes, heart disease, and sometimes that of pneumonia. The last-named affection is characterized by rapidity rather than by depth of breathing, and in its early and pleuritic stages the breathing is hurried and shallow. The slow breathing of shock, hysteria, and intracranial lesions (see Irregular Breathing) may be unduly forcible.

4. **Decreased Force of Respiration.**—Shallow breathing is, as a rule, associated with increased frequency.

5. **Altered Rhythm of Respiration.**—Normally the act of expiration lasts a little longer than inspiration, in the proportion of about 6 to 5, and a slight pause ensues after expiration, unless the breathing be hurried. It must be borne in mind that *audible* inspiration lasts three or four times as long as the expiratory sound. (See Auscultation of the Thorax, p. 407.) Three types of arrhythmical dyspnoea may be mentioned—

viz.: (a) inspiratory dyspnoea, (b) expiratory dyspnoea, and (c) irregular breathing.

(a) **Inspiratory Dyspnoea** occurs almost exclusively in conditions which cause obstruction to the free entrance of air to the lungs, among which may be mentioned: laryngeal obstruction (œdema, laryngitis, diphtheria, tumours, foreign bodies, stenosis, paralysis or spasm of the vocal cords); tracheal obstruction (foreign bodies, pressure of tumours or aneurism).

The most striking evidences of dyspnoea are, perhaps, displayed in cases of this description. The following quotation from Lindsay's work on Diseases of the Lungs is to the point: 'The essential fact is the existence of some mechanical obstacle to the free entrance of air into the larynx and trachea. The characteristics of this form of dyspnoea are stridor and either an increase or a diminution of the rate of respiration. If the former, the respirations will be superficial; if the latter, they will be deep, the organism seeking to compensate for the difficulty in the entrance of air, either by an increased frequency or an increased amplitude of respiration. In consequence of the incomplete entrance of air into the thorax, there will be more or less recession of the supraclavicular and jugular fossæ, the epigastrium, and the lower intercostal spaces in the lateral regions. The downward movement of the diaphragm during inspiration is diminished. Stridor is usually a marked feature in these cases, and is a point of great diagnostic significance. It is usually at first an exclusively inspiratory stridor, but as the case proceeds—*e.g.*, in œdema of the glottis—it may become expiratory also. The urgency of the dyspnoea in this class of case is often great, but depends not only upon the degree of obstruction, but upon the rapidity with which it is developed. Thus, a foreign body suddenly gaining entrance to the larynx or trachea (a cause always to be borne in mind when inspiratory dyspnoea suddenly develops in an individual, especially a child, previously healthy), will cause a much more urgent dyspnoea in proportion to the amount of obstruction than such a cause as the gradual pressure of a tumour or aneurism upon the larynx or trachea from without. Œdema of the glottis is another example of a cause which may develop rapidly, and speedily produce a very intense form of dyspnoea, chiefly inspiratory. A membranous laryngitis (diphtheria) often causes a form of inspiratory dyspnoea, which may be for a time slight or moderate, but is liable at any time to become urgent,

and to give rise to alarming symptoms. In laryngismus stridulus we have a form of dyspnœa which develops very suddenly, and is characterized by a temporary arrest of respiration, followed by a series of deep, noisy, stridulous inspirations. . . . The "laryngeal crises" of tabes dorsalis are an important example of inspiratory dyspnœa. They depend upon spasm of the glottis, and are accompanied by signs of impediment to the entrance of air into the larynx, and by a cough resembling that of whooping-cough.'

(b) **Expiratory Dyspnœa.**—The abdominal muscles are seen to be unusually active, and there may be bulging of the supra-clavicular and intercostal spaces during expiration; want of elasticity of the lungs and thoracic walls is the chief cause, and the necessary conditions for the production of this symptom are furnished in emphysema, chronic bronchitis, and asthma.

(c) **Irregular Breathing.**—Irregularity in the force of the respirations and in the intervals separating them is seen in hysterical and emotional states, in heart failure, in collapse, in cerebral hæmorrhage, brain tumours, meningitis, etc. A more methodical form of respiratory irregularity is sometimes seen in cerebral meningitis and occasionally in other grave disorders, and is known as **Biot's respiration**. At intervals of variable duration the breathing ceases for perhaps half a minute, then proceeds as before.

**Cheyne-Stokes respiration** is another example of a somewhat systematic type of irregular respiration; it bears some resemblance to Biot's breathing. For a short period the patient does not breathe, then slowly begins the shallow respirations at considerable intervals; the respiratory acts increase in force and frequency till they reach a maximum, then gradually diminish till they cease again for a time. The symptom is frequently seen in unconscious subjects, and often in sleep, or when the patient is breathing quietly and without self-consciousness; in some cases the patient loses consciousness during the period of respiratory pause, regaining it when the increased breathing supervenes. The symptom is produced by a want of excitability of the respiratory centre, but the exact origin of the augmenting and diminishing processes is uncertain; it may depend entirely on the degree of venosity of the blood (Traube), or on vaso-motor influence combined with the condition of the blood (Filehne), or may be a question of fatigue (Rosenbach). The varying quantities of carbon dioxide in the blood are probably important factors in

this, as in normal respiration. If the pressure of carbon dioxide in the alveoli fall below the amount which will stimulate the respiratory centre (the 'threshold value'), apnœa, or cessation of respiration, is the result. As the carbon dioxide accumulates, the respiratory action grows more forcible.

This symptom is, as a rule, an ominous sign, indicating in many cases a fatal termination. It is occasionally a transient sign, passing off as the patient improves; rarely it may persist for long periods, even for months, and this especially in cardiac and renal cases. Serious intracranial disease, heart affections, kidney disease, and arterio-sclerosis are the usual causes of the symptom; poisonous doses of morphia may originate Cheyne-Stokes breathing, and medicinal doses of the drug may intensify the symptom if it be already present.

A few other characteristics of dyspnœa merely require recognition. **Cyanosis** (see p. 114) is frequently present, but may be relieved by the active respiratory efforts; the same may be said of venous engorgement. **Stridor** has already been referred to; stridulous dyspnœa is an almost infallible indication of obstruction to the entrance of the air into the air passages. In addition to the exaggerated movements of the trunk muscles concerned in respiration, and to the recessions or bulgings of the thorax and epigastrium already mentioned, the **alæ nasi** are often seen to move vigorously with each act of breathing, and this at times when other signs of dyspnœa are ill marked. While it is usual for the patient to experience distress commensurate in some degree with the objective dyspnœa, this may be replaced by **apathy**, which in cases of organic disease is a formidable sign. **Stertorous dyspnœa**, caused by noisy vibrations of the soft palate while breathing through the mouth, rarely occurs, except during states of unconsciousness; it may be only sleep, but it is commonly observed in comatose conditions—*e.g.*, cerebral apoplexy, uræmic, diabetic, alcoholic coma, ante-mortem coma, and that due to opium or other narcotic poisons. It is constantly noted in children with enlarged tonsils and post-nasal adenoids, owing to their unavoidable mouth-breathing.

**DYSTROPHY** (Gr. δύς, bad; τροφή, nourishment).

Changes in the structures of tissues may result from some interference with the normal trophic control exercised by certain nervous organs over the state of nutrition of the remaining tissues

of the body. The most important of the trophic changes so brought about may be grouped into three classes: (a) trophic affections of the skin and its appendages, (b) of the muscles, and (c) of the bones and joints. The subject is further considered in the article on Trophic Disturbances (p. 487).

### **ELBOW-JERK.**

The triceps muscle is stretched by flexing the elbow-joint, the forearm hanging vertically; a tap on the triceps tendon now causes the forearm to be extended. This is often seen in health, and constantly in cases showing exalted reflexes of the upper extremity. (See Reflexes, p. 339.)

### **ELLIS'S LINE (Garland's Line).**

The upper boundary of the area of abnormal dulness in the pulmonary region, due to the presence of pleuritic effusion, is not a horizontal or fluid level line. It usually presents a curved outline, known as Ellis's line, which rises highest in the scapular or mid-axillary line, falling as it passes forward, and to a less extent as it approaches the median line behind. In hydrothorax, the result not of pleurisy, but of circulatory defects, as seen in heart disease or kidney affections, this curved line of dulness is less frequently observed. The position of free fluid in the pleural cavity is influenced by gravity, by pleuritic adhesions, and by intrathoracic pressure. The condition is more fully considered with the Percussion-Sounds of the Thorax, at p. 457.

### **EMACIATION.**

Wasting is observed in so many affections of a different nature that it cannot be said to be a symptom of much diagnostic value; variations in the weight are more valuable as an index of the progress of a case during treatment.

Loss of flesh in a young patient without obvious signs of disease suggests a possible tubercular affection. Emaciation combined with a hearty appetite may indicate diabetes. A urinary examination will, of course, in this case settle the question.

The cachexia of cancer, the wasting of fevers, the emaciation of progressive muscular atrophy, and of the affected regions in infantile paralysis, are striking instances of this condition, and are to be reckoned with in forming the diagnosis.

**EMBRYOCARDIA.**

Instead of the halting normal rhythm of the heart-sounds, one hears at times a rapid, regular beat, with an equal duration of pause between each sound; the rhythm is that of the 'pendulum beat' (*q.v.*, p. 428), but the latter is slower, and is produced by different causes. It resembles the sound of the foetal heart, and is found in conditions of heart exhaustion, as in fevers, in diphtheritic or other paralyses affecting the heart, and in the terminal stages of heart disease.

**EMPHYSEMATOUS CHEST.** See **Barrel Chest**, p. 56.

**EMPROSTHOTONOS** (Gr. *ἔμπροσθεν*, forwards; *τόνος*, a stretching).

A tonic spasm of the muscles of the trunk, in consequence of which the body is curved forwards; the anterior trunk muscles in this condition overcome those of the back. This symptom is sometimes observed in tetanus.

**ENTEROPTOSIS.**

An undue mobility and displacement of the intestines and of most of the other organs of the abdomen, due to a relaxation of all the supporting structures of these organs (see p. 10 and Fig. 22).

**EPIGASTRIC PULSATION.**

Pulsation in this region may be due to a strongly acting or enlarged right ventricle, to an unduly pulsatile but otherwise healthy abdominal aorta, to a tumour transmitting the aortic pulsations to the surface, to a pulsating liver, or to an abdominal aneurism (see p. 12).

**EPIGASTRIC REGION.**

That portion of the abdominal surface bounded above by the sternum, on each side by the costal margins, and below by a horizontal line drawn from the lowest point of one tenth rib to a similar point on the opposite side.

Retraction of this region, producing the **scaphoid** or boat-shaped abdomen, is seen in cases of meningitis and cerebral tumours, and in colic especially when due to chronic lead-poisoning. Stenosis of the larynx causes marked retraction of the epigastrium, as well

as of the intercostal spaces and supraclavicular regions. Swelling or bulging may be due to flatulent distension of the stomach or intestine; to tumour of the liver, gall-bladder, or pylorus; to abdominal aneurism. Pulsation, if systolic in time, is usually due to a strongly acting or dilated right ventricle; if post-systolic, to an 'irritable aorta,' or to aneurism of the abdominal aorta or its branches (see p. 12). Pain in this region may be the result of



FIG. 22.—A CASE OF ENTEROPTOSIS ARISING FROM AN UMBILICAL HERNIA. The hernial sac contained practically all the intestines.

stomach disorders. If it occur immediately or very soon after a meal, it may be due to ulceration of the stomach, either malignant or peptic; if an hour or two elapse between the meal and the onset of pain, it is more likely to be a catarrhal condition, or possibly duodenal ulcer; it frequently happens, however, that the pain of gastric ulcer is not felt till a considerable time has elapsed after eating. Gastralgia due to a neurotic condition causes a similar pain, but it must be admitted that the gastric neuroses are more frequently diagnosed than the condition warrants; many cases of so-called gastric neuralgia are in reality the results of gastric



ulcer—that is, adhesions or cicatricial contractions, occurring in some instances in neurotic subjects. The pain of gall-stones is also experienced in this region at times, though, like that of duodenal ulcer, it is more frequently located in the right hypochondriac or the umbilical region. In attacks of appendicitis, and especially at the beginning of the attack, it is not uncommon to observe the pain to be epigastric, and the same may be said of pneumonia, especially in children. Diaphragmatic pain, the result of pleurisy or of violent coughing or vomiting, is felt chiefly in the epigastrium.

**EPISTAXIS.** See *Disorders of the Nose*, p. 259.

**EPISTERNAL PULSATION.** See *Arteries, Examination of*, p. 43.

**ERB'S SIGN.**

An increased excitability of the muscles to electrical and mechanical stimulation, which may be observed in cases of tetany.

**ERRONEOUS PROJECTION.**

In consequence of muscular weakness, the effort required to fix the eye upon a given object may be misjudged, the 'strength sense' being at fault. The result is a miscalculation as to the exact position of the object (see p. 220).

**ERYTHEMATOUS RASH.** The condition in which one finds a red eruption are enumerated at p. 362.

**EWART'S SIGN.**

In cases of pericardial effusion, one may find an area of the posterior chest-wall over the base of the left lung, where the normal vesicular breathing is absent, and voice-sounds are feeble; the percussion note is wanting in resonance. This area lies below and internal to the left scapula, and is stated by Ewart to be due to an altered dorsal relation of the liver.

**EXOPHTHALMOS** (Gr. ἐξ, out; ὀφθαλμός, the eye).

Protrusion of the eyes is due to either of two conditions—viz., weakness of the structures which retain the eyeball in its seat, as

may be seen at times in paralysis of all the ocular muscles (third nerve paralysis), or the more commonly observed cause of protrusion by pressure from behind without obvious defect in the muscles. It is in exophthalmic goitre that this symptom is most frequently observed. Here the excessive blood-supply in the orbit, combined with increased quantity of fat behind the globe, are probably the causes of the protrusion. Tumours or inflammation of the orbit cause the eye to project. A rare but characteristic instance of the former is chloroma (see Fig. 23). Other swellings in the orbit may be borne in mind—*e.g.*, enlarged lachrymal gland, exostoses, œdema.



FIG. 23.—EXOPHTHALMOS FROM CHLOROMA.

The photograph was taken a couple of days before the child's death. The projecting eyes and swollen temporal regions gave the characteristic frog-like appearance. A post-mortem examination was unobtainable. (Dr. Reid's patient; photographed by Dr. Fullerton.)

The condition is most frequently bilateral, but in local affections is often unilateral.

In exophthalmic goitre several well-known accompanying symptoms are due largely to exophthalmos—*viz.*, (*a*) **Graefe's sign**. On directing the patient to lower his gaze slowly from the ceiling to the floor, his upper lid is observed to lag behind the eyeball in the movement; an increasing width of sclerotic is seen above the cornea as the eye falls. This sign is not strictly dependent on the degree of protrusion, and may often be observed before the exophthalmos is well developed, or it may disappear in the course

of the disease while the protrusion still persists. (b) **Stellwag's sign**: incompleteness and infrequency of the involuntary winking. (c) **Moebius's sign**: insufficiency of convergence in the act of looking at a near object; here one eye only is used to fix the object, the other diverging.

### EXTRA SYSTOLE.

A common cause of intermission and other irregularities of the pulse is the occurrence of an extra systole. The subject is considered under Arrhythmia (p. 39).

**EYES, Projecting.** See **Exophthalmos**.

**FACIAL CONTRACTURE.** See **Contracture**, p. 104.

### FACIAL PARALYSIS.

Injury or disease of the facial nerve or of its nucleus causes loss of power of one side of the face. The condition is fully discussed in the article on Diminished Movements, at p. 223.

### FÆCES, Abnormalities in their Condition and Constituents.

Examination of the fæces—Shape and consistency of the motion—Colour—Odour—Constituents of the motion—Mucus—Blood—Melæna—Pus—Fragments of tumours—Sloughs of bowel tissue or polypi—Calculi of various descriptions—Gall-stones—Foreign bodies—Undigested food (lientery).

Animal parasites: *Amœba dysenteria*—Intestinal worms—Round-worms: *ascaris*, *oxyuris*, *ankylostomum*, *Trichocephalus dispar*—Tapeworms—The proglottis—The strobilus—*Tania saginata*—*Tania solium*—*Bothriocephalus latus*.

In this article are considered the changes introduced by disease which are to be observed in the condition and constituents of the fæces; abnormalities in their mode of evacuation are separately discussed in the section on Defæcation (p. 116).

**Examination of the Fæces.**—Inspection of the evacuation is in many cases all that is necessary; by this means one observes the shape, colour, consistency, odour, and the presence of gross changes in the constituents of the motion. It may, however, be necessary to search carefully for abnormal substances (*e.g.*, worms, gall-stones), in which case the solid contents of the stool (if there are any) must be washed thoroughly through a sieve. Special forms of strainers can be obtained, which are designed to mitigate

the offensiveness of the operation. Under exceptional circumstances it is desirable to examine portions of the stool under the microscope. A minute quantity is placed on a slide; if solid it is softened with a drop or two of normal saline solution (0.6 per cent. solution of common salt), and a cover-glass applied. The film is then examined with a low and medium power lens.

**Shape and Consistency of the Motion.**—During its course through the large intestine the faecal mass, which was fluid in the ileum, gradually loses water, and arrives in the rectum in a semi-solid state. Owing to affections of the rectum or anus, the normal cylindrical shape of the motion may be altered: (a) Grooved, flattened, or ribbon-shaped stools indicate pressure at the outlet. This may be caused by piles, spasm of the sphincter ani (possibly produced by anal fissure, or by tonics, such as strychnine), cicatricial narrowing of the orifice, as a result of healed ulcers, ischio-rectal abscess, cancer or syphilis of the rectum, if situated at a low level in the bowel. (b) All varieties of rounded and irregular masses (*scybala*), from the size of green peas to huge collections, may be observed; these shapes are usually the effect of constipation. (c) Instead of the formed condition of the stool, it may be soft and liquid, without form, or with a certain proportion of solid masses floating in the fluid motion—the condition seen in diarrhoea. In certain forms of diarrhoea the motion is entirely fluid, being, in fact, little more than the serum withdrawn from the blood; this is found in cholera—both the Asiatic and the English diseases—and in the effects of poison (*e.g.*, arsenic and antimony). In Asiatic cholera the motion is a dirty whitish opalescent fluid, and is commonly known as the *rice-water stool* (see p. 120).

**Colour of the Motion.**—The normal colour (from light yellow in infancy to dark brown of adult life) is replaced or modified in a variety of conditions.

The ingestion of certain articles of food and of drugs changes the colour of the stools: a dark slaty grey or black colour results from the administration of bismuth, iron, or charcoal, and may be mistaken for altered blood in the motion. This is of diagnostic importance, as the commonest cause of blood appearing in the motions (gastric or duodenal ulcer), is frequently treated with these very drugs. Red wine, if consumed in considerable quantity, and fruits with dark juice (*e.g.*, blackberries, black cherries) darken the faeces. Starchy or milk foods cause the stools to be light in colour; when milk is the sole nourishment the motions

are pale yellow. On the other hand, a diet mainly of flesh meat causes the stools to be dark brown. An olive green motion may be the result of eating a large amount of green vegetables. A commoner instance of green stools is that seen in the digestive disturbances of infancy and childhood. This colour is often observed to follow the administration of mercury, which acts as an antiseptic, preventing the change of biliverdin into urobilin; the green colour seen among infants may be due to a similar failure of the biliverdin to be transformed into urobilin.

The altered colour may be due to the presence of blood in the motion. When this arises from the lowest regions of the bowel, it is red in colour (unless it has been retained for some time in the rectum). When it escapes from the upper portions of the intestine or from the stomach, it is changed into a black or dark tarry mass, the black discharge being known as *melæna* (see below, p. 140). The importance of this symptom is very great, but, as already indicated, one must make certain that the colour is really due to blood, and not to one of the drugs referred to.

Variations in the quantity of bile which finds its way into the duodenum are a common source of change in the colour of the stool. Excess of bile causes an unusual darkness of the motions; the contrary condition, in which there is a deficiency of bile in the intestine, results in the appearance of the so-called clay-coloured stools, a greyish or whitish shade, with very little of the yellow tinge. This deficiency of bile may be a deficiency of bile-production (*acholia*), as may be observed in some cases of cirrhosis, or cancer of the liver, or in the rarer condition acute yellow atrophy of the liver; it may also occur in anæmia and chronic lead-poisoning. Under these circumstances the fæces may have little or no bile-colouring, but there is no jaundice. This distinguishes acholia from those conditions where there is abundant formation of bile, which is prevented by some obstruction from reaching the duodenum; in this case the fæces are also pale, but the skin and urine are bile-stained. The obstruction is often due to catarrh of the common or other bile-ducts, whereby the mucous lining of the bile-passages is thickened, and blocks the canals; it is the common result of the passage of a gall-stone, which may become impacted in the common duct, or cicatricial contraction of the duct may occlude it at its entrance into the duodenum. Pressure on the duct from the outside is another fairly frequent cause; it may be the result of inflammation or new growth in the head of the pancreas, or in the portal fissure.

The colour of the stool in typhoid fever is somewhat characteristic; it is usually described as the **pea-soup motion**, owing to its consistency as well as its colour. The greyish colour of cholera stools has already been referred to.

**Odour of the Fæces.**—The infantile stool, which is usually almost odourless, may become offensive in conditions of intestinal irritation. The odour of the typhoid stool is offensive, and may often be recognized. In dysentery the fæcal odour may disappear, and the same may be observed in cancer of the rectum.

**The Constituents of the Fæcal Discharge.**—By inspection and, if necessary, by microscopic examination a number of abnormal substances may be detected in the fæces—viz., mucus, blood, pus, fragments of tumours or sloughs of the bowel itself, fat, calculi of various descriptions, foreign bodies, undigested food, and animal parasites.

(a) **Mucus.**—The slimy appearance of stools containing mucus can rarely be mistaken; a small quantity is a normal constituent, a large amount indicates catarrh of the intestine. If the motion be coated with mucus, or if the motion be composed of strings or ribbons of mucus floating in watery fluid, the catarrh is seated in the large bowel. If the mucus be well mixed through the stool, or be found chiefly as shreds and small transparent lumps evenly distributed throughout the motion, the small intestine is the catarrhal region. The catarrh is the result of superficial inflammation of the mucous membrane, usually of micro-organismal origin—*e.g.*, colitis, proctitis, dysenteric and other inflammations and ulceration, injury to the mucous membrane from contact with foreign bodies or impacted fæces, strangulation of a portion of the bowel by intussusception. In several of these affections blood is also passed (dysentery, colitis, intussusception); shreds or casts of coagulated mucus are seen in the condition known as membranous entero-colitis or mucous colitis, a disease chiefly affecting women, in which the excessive mucous secretion depends mainly on a functional or neurotic increase of activity of the secreting glands.

(b) **Blood.**—As stated above, blood, if it originate high up in the alimentary canal, is dark and tarry in appearance (*melæna*); if it has been shed in the lower part of the ileum or in the colon, it will show a red colour, bright or dark, according to the amount of blood and the duration of its stay in the bowel. It has to be carefully distinguished from the dark motions of those who have been taking iron, bismuth, or charcoal; in these instances the blackness is of a slaty or greyish hue, while the bloody stool has

often a slightly purplish tinge. In case of doubt a small portion of the fæces may be examined chemically by the guaiacum test (see p. 533).

The causes of blood in the stools are numerous. If the blood be dark and tarry, it may come from the stomach, into which cavity it may have been swallowed (adenoid operation, œsophageal varix, aneurism), or it may have been shed there as a result of gastric ulcer, venous stasis (cardiac, cirrhosis of the liver), or malignant disease. Dark blood is seen in consequence of duodenal ulceration, and bleeding from the upper regions of the ileum may occur in hæmophilia, purpura, leukanæmia, dysentery, and sometimes in typhoid fever.

Red blood, if in large quantity, may also originate in the regions and from the affections just enumerated; it is, however, more likely to arise in the colon or lower part of the ileum. Thus the blood from typhoid ulcers is usually a distinct, though often a dark, red, coming as it does from the neighbourhood of the ileo-cæcal valve; it is frequently very copious in this disease. Bleeding in the rectum is a common cause of hæmorrhage. Here the blood is not only bright in colour, but may be smeared on the surface of, or only partially mixed into, the motion, while that which is shed high up in the gut is more evenly mixed with the other constituents of the stool. Piles, cancerous, syphilitic, dysenteric, or simple ulceration of the rectum, fissure of the anus, and rectal polypi, are the chief causes of bleeding from this region. Ulcers of the colon (simple, malignant, syphilitic, dysenteric, typhoid, tubercular) are the usual causes of bleeding in this portion of the bowel; here also the blood is distinctly red, unless it have remained an excessively long time in the bowel. Aneurism opening into the bowel may be mentioned as an occasional cause of bloody stools. The blood from an intussusception is usually free from fæcal matter, and may be mixed with mucus; the discharge from dysentery may be sero-purulent, or may be serum mixed with blood and scraps of fibrinous or mucoid material (the 'meat-juice motion').

(c) Pus.—When in large quantity pus in the stool is readily recognized by inspection, and is invariably due to the opening of an adjacent abscess into the bowel. If the amount of pus be small, it may be with difficulty demonstrated by the microscope, as the pus cells soon undergo changes in the bowel; but if from the rectum, even a small quantity of pus may be identified. A

very small amount may be present in catarrhal conditions of the bowel, but, as a rule, when pus is distinguishable in the stool, it indicates some degree of ulceration, if not an abscess.

(d) **Fragments of tumours** may possibly be found and recognized by the microscope in cases of cancer of the lower regions of the bowel.

(e) **Sloughs of the bowel** of greater or less extent may be occasioned by found in cases where necrotic processes are at work; intussusception is sometimes the cause of such loss of tissue. Tubercular dysentery, or acute phlegmonous inflammation of the intestine may cause the appearance of portions of the mucous or even muscular coat of the gut. Portions of sloughing polypi may be occasionally found.

(f) **Fat.**—Stools containing fat present a greyish, and often a glistening or greasy, appearance. A deficiency of bile in the intestine (obstructive jaundice) is the chief cause of this symptom. Owing to the loss of the emulsifying power of the bile, the fat is not absorbed fully from the bowel. In pancreatic disease, whereby the pancreatic juice is defective (cancer, obstruction of the pancreatic duct), the stools are also likely to contain undigested fat.

(g) **Calculi.**—The only stones of any diagnostic interest to be found in the fæces are gall-stones. Calculi derived from the salivary glands, from the tonsils, from the pancreas, and the so-called intestinal sand, of uncertain origin, may very rarely be detected in the stools, but their appearance there is extremely exceptional.

**Gall-stones** may be found by thoroughly washing the motion through a sieve. They are generally composed of cholesterin, and float in water. Some, however, the darker specimens, are largely formed of lime salts (calcium bilirubin), and are heavier than water. They should be sought for perseveringly after an attack of biliary colic (hypochondriac and epigastric pain, rigors, moderate pyrexia, vomiting, possibly jaundice), and the search should not be given up for a fortnight. The failure to find the concretions by no means disproves the attack to be the passage of gall-stones, as the latter are often soft, and dissolve or become unrecognizable in the course of their transit through the intestine. They are, as a rule, easily identified as yellowish to brown or olive-green bodies, ranging from the size of a pin's head to that of a pigeon's egg; the surface is smooth, and may be rounded (when only one stone was present in the gall-bladder or duct), but



is more commonly faceted by mutual pressure of several or many stones in the gall-bladder, where they may have rested for long periods without giving rise to symptoms.

The so-called **intestinal sand** often consists of large quantities of minute gall-stones.

(h) **Foreign bodies** are of infrequent occurrence, and are the result of accidental or voluntary swallowing of objects which are not articles of food—*e.g.*, coins, artificial teeth, marbles, fruit-stones, etc. In some cases (among children or the insane) the foreign body has been inserted into the rectum.

(i) **Undigested food** is sometimes found in the motion; the stool may resemble food that has been vomited, with some addition of faecal appearance and odour. The condition, known as **lenteria**, or **lenteric diarrhoea**, occurs most commonly in infants whose digestive function is seriously impaired; it may also be observed in some cases of acute or severe dyspepsia in adults, and especially among those who fail to masticate their food properly.

(j) **Animal Parasites**.—Some examples of the protozoa may occasionally be found in the motion; of these the *Amoeba dysenteriae* is the only form of diagnostic importance, as it has been identified as the cause of certain cases of dysenteric diarrhoea. Of greater interest are the **intestinal worms**. They may give rise to no symptoms, or only those of intestinal irritation, or in some instances of anæmia. The following varieties may be enumerated:

(i.) **Round-worms (Nematoda)**—*Ascaris Lumbricoides* (*Common Round-worm*).—The parasite resembles the earthworm in appearance, and is from 6 to 12 inches long; it inhabits chiefly the small intestine. As a rule, there are only one or two worms present in the bowel, but large numbers have occasionally been observed, and they may even form a cause of intestinal obstruction.

*Oxyuris Vermicularis* (*Threadworm*).—It resides mainly in the large bowel, and, like the *ascaris*, is chiefly found in children. It is less than  $\frac{1}{2}$  inch long, and may be passed in large numbers after an aperient or owing to an enema.

*Ankylostomum Duodenale*.—A small worm inhabiting the duodenum and jejunum chiefly; the female is about  $\frac{1}{2}$  inch long, and the male half that length, and it adheres by four teeth to the mucous membrane. It occurs in the tropics and in Southern Europe, and gives rise to the condition known as **ankylostomiasis**, of which the chief feature is anæmia. The worms may be found in the fæces after the administration of suitable

anthelmintics (e.g., thymol). An allied species, the *Uncinaria Americana*, is common in the Southern States of North America, and produces similar symptoms.

*Trichocephalus Dispar* (Whip-worm).—This parasite is common in France, but rare in this country. It infests the cæcum and large intestine, and may cause diarrhœa or other symptoms of intestinal irritation; the ova may be detected in the fæces.

(ii.) **Tapeworms (Cestoda).**—Three varieties are encountered fairly frequently: *Tania saginata*, *Tania solium*, and *Bothriocephalus latus*. They consist of a head, by which the parasite attaches itself to the mucous membrane of the small intestine, and a long series of segments or **proglottides**, each of which is bisexual, and contains numerous eggs. The mature worm of combined segments is termed a **strobilus**. As the worm develops, the segments separate and are passed *per anum*, when they may be examined by placing a segment between two slips of glass gently pressed together; they are identified by their characters mentioned below. The diagnosis of tapeworm can only be made by discovering the parasite in the fæces; the objects declared by the non-medical public to be tapeworms must be always carefully scrutinized, as strips of mucus are often mistaken for the worm. The worm, as a rule, is solitary, and so long as the head is retained in the bowel the case cannot be regarded as cured. The following points will serve to distinguish the different varieties:

*Tania Saginata* (*T. Mediocanellata*, *Unarmed or Beef Tapeworm*).—An adult proglottis measures about 18 millimetres ( $\frac{3}{4}$  inch) by 10 millimetres ( $\frac{2}{5}$  inch); the uterus is seen as a median canal, with numerous (fifteen to thirty-five) well-divided lateral branches. The head is square, about 2.5 millimetres in diameter, and is furnished with four sucking discs; it has no hooklets, hence its name 'unarmed.' This tapeworm may reach a length of 18 feet.

*Tania Solium* (*Pork Tapeworm*).—The proglottis is smaller than the last named—viz., about 10 millimetres by 5 or 6 millimetres. The lateral branches of the median uterus are less numerous (eight to fifteen) and coarser than those of the beef tapeworm. The head is smaller and rounded, and is armed with a ring of hooklets at its pole, surrounded by four sucking discs. The total length of the worm may be 10 feet.

*Bothriocephalus Latus*.—The adult proglottis is nearly square, measuring about 5 millimetres in each direction. The uterus is a median convoluted tube arranged with three or four branches

as a rosette. The head is elongated, about 2 millimetres in diameter, and has neither sucking discs nor teeth, but is provided with a median groove on each side of its head, which may act as a sucker. This is the longest of the tapeworms, and may reach a length of 25 feet. It is acquired by eating imperfectly-cooked fish, and is common in Switzerland and the East of Europe. It gives rise to a severe form of anæmia, identical in many respects with pernicious anæmia.

**FEHLING'S TEST (for Sugar).** See *Urine, Examination of*, p. 534.

**FESTINATING GAIT (Festination, Propulsion).**

A type of progression sometimes seen in cases of paralysis agitans. The patient's tendency is to fall forward, and in order to keep upright he is obliged to walk faster and faster until he is brought up by some obstacle. The condition is further discussed in the article on Gait (p. 150).

**FEVER.**

This word is often used synonymously with pyrexia, or elevation of the body temperature above the normal height. It is, however, more correctly employed to indicate, not only the rise of temperature, but also the various signs of constitutional disorder which, equally with the pyrexia, are the result of very many bacterial and other disturbing influences. The more important of these are malaise, pain, weakness, disturbance of the digestion, with loss of appetite, thirst, diminution in the quantity of urine, emaciation, rapid breathing, and often temporary mental disturbance (delirium). (See *Temperature*, p. 404.)

**FINGERS, Clubbed.**

An enlargement of the terminal phalanges of the fingers, the nails being curved laterally and longitudinally (see Fig. 24). The swellings are to be seen in cases of chronic heart and lung affections, and particularly in empyema and bronchiectasis. As a rule, a considerable time is required to produce the deformity, but in some cases the condition may come and go inside a few weeks. It seems to be mainly the result of sustained congestion in the capillaries.

**FIXATION POINT.**

The spot in the field of vision on which the gaze is fixed, the image of which is formed upon the macula lutea. The visual axes cross at this point (see p. 216).

**FLASHES OF HEAT.**

In neurasthenic conditions, and especially in the nervous disturbances occurring at the menopause, women are frequently annoyed by various sensory disturbances. Of these a common one is the sensation as of a hot wave passing over the body.



FIG. 24.—TORPID FINGERS.

**FLASHES OF LIGHT** may be perceived by patients suffering from acute indigestion, and from migraine. They may form the aura in epilepsy.

**FLATULENCE.**

The presence of an excessive quantity of gas in the alimentary canal, giving rise to abdominal pain, increased peristalsis, belching, eructation, and the passage of flatus *per anum*.

The symptom is usually the result of delayed and imperfect digestion, with the evolution of gas mainly by a fermenting process in the stomach and bowels; it is chiefly observed in adults about or past middle age, who lead a sedentary life. The ingestion of an excessive proportion of starchy food is largely responsible for flatulence, especially if it have not been sufficiently masticated and mixed with saliva, as is commonly the case with soft and farinaceous foods. It may in some cases be in part caused by a habit of **swallowing** air, and this is by some observers looked upon as the chief source of the gas in cases of neurotic flatulence, which are not uncommon; it is, however, doubtful if any large bulk of air can be rapidly passed by this means into the stomach.

A more extreme degree of flatulent accumulation of gas in the stomach and intestines is termed **meteorism** or **tympanites** (see p. 7).

### FLINT'S MURMUR.

A diastolic murmur, heard best at the apex of the heart. It is found in cases of aortic regurgitation, with dilatation of the left ventricle (see p. 439).

### FLUID VEIN (Veine fluide).

This physical phenomenon accounts for the majority of endocardial and vascular murmurs. The conditions necessary for its production are: a current flowing with sufficient swiftness; the passage of the fluid through a channel which suddenly widens—*i.e.*, it flows through an aperture which is of less diameter than that of the chamber which receives the fluid; the contrary condition of a stream flowing from a larger channel into a narrower may also at times have the same effect. The result is a series of vibrations set up in the fluid, which are transmitted through the walls of the chamber and intervening tissues to the ear of the observer, and form a murmur. Should the vibrations be sufficiently ample and not too frequent, they may be **felt** as a **Thrill**. (See the article on the Auscultation of the Thorax, p. 433.)

**FLUOROSCOPY.** See **X-Ray Diagnosis**, p. 565.

**FRICITION FREMITUS.**

Movements imparted to the surface of the body and palpable by the observer's hand, caused by the rubbing together of two roughened surfaces in the vicinity of the spot palpated. It is usually a sign of pleurisy, when it is synchronous with respiration; if felt over the lower part of the front of the chest, synchronously with the heart-beat, it indicates pericarditis; felt over the regions of the liver or spleen, it signifies peritonitis of the serous coating of those organs (perihepatitis, perisplenitis). A similar grating felt over the limbs may indicate synovitis or tenosynovitis.

**FRICITION SOUNDS.**

On listening over the thorax or abdomen rubbing sounds may, under certain morbid conditions, be heard, which are due to the friction of abnormally roughened surfaces upon each other. These surfaces may be the pleuræ, the pericardium, or the peritoneum. In addition, one hears at times various extraneous rubbing or grating sounds, originating in the shoulder-joint, the scapula, or the skin. The subject is considered in more detail at p. 421.

**FRIEDREICH'S SIGN.**

On percussing over a pulmonary cavity the sound elicited is observed to be higher in pitch during inspiration than during expiration (Friedreich's sign). The same difference, but to a less marked extent, may be observed in percussing the normal chest. It depends on a difference in tension in the wall of the cavity and of the lung tissue.

The same observer's name has been attached to a sign of adherent pericardium—viz., a diastolic collapse of the jugular veins.

**FUNNEL CHEST (Trichterbrust).**

A chest presenting a depression or groove at the lower end of the sternum; it may be congenital, or may appear in childhood, or may even be produced in adult life by pressure long continued on the region. (See Thorax, Shape, etc., p. 468.)

**GAIT.**

The patient's mode of progression should, when possible, be observed; this is particularly necessary in diseases presumably

of the nervous system, but may be useful in other morbid conditions. In examining the patient the legs, as high as the knees, should be exposed, and he should be directed to walk away from and toward the observer, and across the line of vision; he may be requested to look straight in front or up to the ceiling while walking, and his mode of raising, putting down, and carrying forward his feet must be narrowly scrutinized; the ease or difficulty with which he turns should be observed.

The following types of gait may be distinguished:

1. **The Spastic Gait.**—Stiffness in the movements is the chief feature; the muscle tone is exaggerated, the extensors of the knee being especially affected, with the result that the joints are flexed with difficulty; hence the legs are brought forward in a very stiff manner, the toes scraping on the ground. The adductors of the thighs may in some cases show the chief spasm, causing the legs to be crossed in front of each other at each step—the **crossed-leg gait**.

The spastic gait is a marked feature in those diseases in which the patellar reflex is increased, and is most frequently produced by interruption to the passage of nerve impulses from the cerebrum to the anterior cornual ganglion cells. This commonly occurs in cases of sclerosis of the lateral columns (provided the patient is able to make some attempt at walking), and occurs in the conditions enumerated in the article on the Reflexes (see p. 342)—viz.: (a) **Transverse interruption of the cord** by compression (vertebral caries, tumours, pachymeningitis, aneurism, trauma), myelitis, or hæmorrhage. (b) **Sclerotic changes in the upper segment of the motor tract**: primary lateral sclerosis; hereditary spastic paraplegia of children; lateral sclerosis, accompanied by other affections of the cord (amyotrophic lateral sclerosis, ataxic paraplegia, multiple sclerosis, syringomyelia); intracranial lesions of the upper segment of the motor tract (hæmorrhage, embolism, thrombosis, tumours, birth-palsies, general paresis). (c) **Intoxications**: tetanus; strychnine-poisoning. (d) **Functional disturbances of the nervous system**: hysteria; 'railway spine.'

Of the above fairly comprehensive list certain spinal affections most frequently give rise to spastic gait, which in these symptoms is commonly bilateral, like most other spinal symptoms. These affections are: multiple sclerosis, amyotrophic lateral sclerosis, primary and secondary or descending lateral sclerosis. The intracranial lesions mentioned under (b) are more likely to produce

a one-sided descending degeneration, with a corresponding one-sided spastic gait.

2. **The Ataxic Gait.**—Several varieties of gaits due to defective muscular co-ordination may be distinguished; of these one is so typically ataxic in character that the name ataxic gait is commonly reserved for it alone. It is characteristic of locomotor ataxia. The patient walks with his eyes fixed on the ground; he brings forward his leg in an uncertain manner; in some cases he seems to feel for the ground with his foot; or, again, he advances it with unnecessary force, raises it unduly, and brings it to the ground with a stamp (**stamping gait**). He keeps his feet widely apart in walking, and his obvious difficulty in maintaining his balance is demonstrated by requesting him to restrict his footsteps to one plank in the floor, which he may be unable to accomplish; the act of turning may cause him considerable difficulty. These evidences of defective equilibrium are more marked in the **reeling gait**, which is seen in disease of the cerebellum, disturbances of the internal ear, and the various conditions referred to in the article on Vertigo (p. 539). Here the mode of progression is of the staggering, reeling type seen in cases of alcoholic intoxication. A gait that is partly ataxic and partly paretic is the so-called **steppage gait**, or **pseudo-ataxic gait**. This is observed in some cases of peripheral neuritis, in which the external popliteal nerve supplying the anterior tibial muscles is involved, and **dropped foot** results. In such cases some interference with the cutaneous and muscular sensory nerves is also present, giving rise to some degree of inco-ordination as well as paralysis. An uncertainty in advancing and placing the foot may be noticed, never, however, to the same extent as in the true tabetic gait; the hanging foot tends to catch the ground, and in order to clear it the knee must be raised excessively, giving rise to the 'heather step,' or high-stepping mode of progression. Looking at the patient's feet as he walks from one, more of the sole of his feet is seen than under normal circumstances.

3. **The Festinating Gait**, or gait of propulsion and retropulsion. Cases of paralysis agitans in standing (see Posture, p. 292) and in walking have a tendency to project their centre of gravity a little in front of their feet; the consequence is that in walking they are obliged to increase their speed in order to avoid falling forward; they must, in other words, hasten after their centre of gravity. The reverse condition is occasionally seen: if they attempt to



walk backward they may be under a similar necessity to increase their pace, owing to a tendency to fall backwards.

4. Disease of the hip-joint and sciatica give rise to a mode of progression which avoids movement at that joint, the leg being carried forward mainly by a swinging movement of the pelvis.

5. A curious **waddling, rolling gait** is seen in cases of large abdominal tumours, ascites, pregnancy, obesity, and in pseudo-hypertrophic paralysis; the shoulders are thrown well back, or the whole body inclines backward, and the lumbar spine is arched forward (lordosis).

6. Every variety of **lameness** may be seen in painful, weak, or damaged conditions of one or both legs.

### GALLOP RHYTHM.

A triple rhythm of the heart-sounds, one of the elements being possibly an indistinct murmur. It may be heard both at the base and at the apex of the heart, and is a sign of failing strength of the myocardium. It may also occur in conditions causing excited action of the heart—*e.g.*, excessive exercise, emotional excitement, Graves' disease (see p. 430).

### GALL-STONES.

The presence of gall-stones in the gall-bladder is not as a rule attended with symptoms; it may be only the passage of a stone into the cystic or common bile-duct that gives rise to signs and symptoms of disease. There may then be observed vomiting, rigors, pyrexia, pain in the right hypochondrium, radiating over the abdomen or toward the right shoulder, tenderness in the neighbourhood of the gall-bladder, and in more than half the cases (Monro) jaundice appears. The discovery of the gall-stones in the *fæces*, while not essential to the diagnosis, is to be attempted in all cases. (See the article on *Fæces*, p. 142.)

### GANGRENE.

As a symptom gangrene occurring in an elderly subject will suggest the presence of arterio-sclerosis; it is also a characteristic feature of diabetes. In Raynaud's disease, or symmetrical gangrene of the extremities, we have an instance of gangrene produced by excessive vaso-motor excitability; the fingers, ears, nose, etc., become white, livid, cyanosed, or even gangrenous.

**GARLAND'S LINE (Ellis's Line).**

A line drawn on the chest corresponding to the upper boundary of the area of dullness resulting from the presence of pleuritic effusion. (See Thorax, Percussion-Sounds, p. 457.)

**GASTRODIAPHANY.** See Examination of the Stomach, p. 394.

**GASTROPTOSIS** (Gr. *γαστήρ*, the belly, stomach; *πίπτω*, to fall).

A downward displacement of the stomach in the abdomen. See the articles on the Examination of the Stomach (p. 392), and the Shape and Movements of the Abdomen (p. 10).

**GASTROSCOPY.** See Examination of the Stomach, p. 394.

**GERHARDT'S PHENOMENON.**

On percussing over a pulmonary cavity containing both air and fluid, it is noticed that the pitch of the resulting note may vary



FIG. 25.—GERHARDT'S PHENOMENON.

A pulmonary cavity containing air and fluid. On changing the posture of the patient the percussion-sound may vary in pitch, owing to change in the shape or of the cavity.

with a change in the patient's posture. The consequent change of position of the fluid in the cavity so alters the shape and character of the cavity that the pitch and quality of the percussion sound changes. A similar phenomenon is noticed in cases of pneumothorax, and is known as Biermer's sign (*q.v.*, p. 57).

**GIRDLE PAIN (Girdle Sensation).**

An illusory sensation, as of a constricting belt round the body, is known as the girdle sensation. It may be a disturbance of

tactile sensory impulses, or is of the nature of painful impressions. It may be caused by a transverse irritative and destructive lesion of the spinal cord, in which case it may be associated with anæsthesia and paralysis of the regions below the lesions. Irritation of the posterior nerve roots will give rise to the symptom. This may occur in spinal meningitis, hæmorrhage, tumour of the cord or vertebræ, caries of the vertebræ (Pott's disease), injury of the spinal column, aortic aneurism. Diseases of the sensory tract in the cord may be the cause—*e.g.*, locomotor ataxia, myelitis.

A somewhat similar sensation may be the result of violent coughing or vomiting, from strain of the diaphragm.

### GLITTERING SCOTOMATA.

Patches of obscurity surrounded by a bright margin are seen in migraine, in irritative lesions of the cortex, and of the cerebral meninges.

### GLUTEAL REFLEX.

On stroking or otherwise irritating the skin over the buttocks the glutei muscles may contract. (See Reflexes, p. 343).

### GLYCOSURIA.

The presence of sugar in the urine is detected by means of the various tests described in the article on Urine Examination (p. 534), while the clinical significance of the symptom is discussed in the article on Urinary Abnormalities (p. 521).

**GMELIN'S TEST (for Bile).** See Urine Examination, p. 538.

**GRAEFE'S SIGN.** See Exophthalmos, p. 135.

### GRANULAR DEGENERATION of the Red Corpuscles of the Blood.

In cases of severe anæmia this change in the staining capacity of the red cells is sometimes seen. It consists in the appearance in a number of the red cells of bluish (basophilic) granules. The change is frequently observed in pernicious anæmia, and is especially characteristic of the anæmia of chronic lead-poisoning; it is stated not to occur in chlorosis. The exact nature of the change is not settled; some authorities regard it as an evidence of degeneration, others of regeneration. It seems to be related to and associated with polychromasia (see p. 67).

**GROCCO'S SIGN (Grocco's Paravertebral Triangle).**

In cases of pleural effusion an area of diminished resonance may be found on the opposite side of the chest to that which contains the fluid. It is triangular in shape, with its base at the level of the lower border of the lung, close to the vertebral column, and its apex about the height of the upper level of the fluid. The diagnostic value of this sign is doubtful, as a similar dulness has been found in cases of subphrenic abscess, lumbar abscess, and pneumonia, and it is stated to be present in health. It may be due to compression of the lung, but there is good reason to suspect that the dulness is merely due to the presence of the vertebral column and the large muscles filling up the costo-vertebral grooves, which, increasing in bulk from above downwards, cause a pyramidal area of diminished resonance in the pulmonary region behind.

**GUMS, Affections of the.**

The condition of the gums may be altered, not only by local disease—*e.g.*, dental caries, epulis—but by disease of more general distribution.

Congested, spongy, or ulcerated gums may be an evidence of scurvy, of mercurialism, of stomatitis, of digestive disorders, of phthisis, and other local inflammations.

A bluish or dark line on the gums at their junction with the teeth is seen in lead-poisoning. A greenish line in the same situation may indicate copper-poisoning. A red line at the margin of the gums is commonly supposed to be a sign of tuberculosis, but it is more probably the result of defective cleanliness in any subject whose resisting power is lowered by debility.

Bleeding from the gums may be seen in hæmophilia, purpura, leukæmia.

**GÜNZBURG'S REACTION.**

A means of detecting the presence of free hydrochloric acid in a fluid. (See Stomach Examination, p. 396.)

**GURGLING RÂLES.**

The coarsest type of râle, produced by the passage of air through a collection of fluid in a phthysical cavity, or in the dilated tubes of bronchiectasis (see p. 419).

**HÆMATEMESIS** (Gr. αἷμα, the blood; ἐμέω, to vomit).

The act of vomiting blood, or the stomach contents mixed with blood.

The appearance of the vomited matter is usually sufficient to indicate the presence of blood, but in some cases it may be necessary to examine the vomit more closely. The method best suited for its detection is the formation of hæmin crystals and their recognition by the microscope (see p. 533).

It is not in every instance easy to say definitely that the blood has come from the stomach, as sometimes there is a combination of coughing, retching, and vomiting in the act of bringing up the blood. It is a common experience that patients who have undoubtedly coughed up the blood from their respiratory passages—*i.e.*, who suffer from hæmoptysis—frequently believe they have vomited it. The distinction may be made by attention to the following points (Osler):

Hæmatemesis.	Hæmoptysis.
1. Previous history points to gastric, hepatic, or splenic disease.	1. Cough or signs of some pulmonary or cardiac disease precedes in many cases the hæmorrhage.
2. The blood is brought up by vomiting, prior to which the patient may experience a feeling of giddiness or faintness.	2. The blood is coughed up, and is usually preceded by a sensation of tickling in the throat. If vomiting occurs, it follows the coughing.
3. The blood is usually clotted, mixed with particles of food, and has an acid reaction. It may be dark, grumous, and fluid.	3. The blood is frothy, bright red in colour, alkaline in reaction. If clotted, rarely in such large coagula, and muco-pus may be mixed with it.
4. Subsequent to the attack the patient passes tarry stools, and signs of disease of the abdominal viscera may be detected.	4. The cough persists; physical signs of local disease in the chest may usually be detected, and the sputa may be blood-stained for many days.

It must be remembered that blood may be shed in the respiratory passages, or in the œsophagus, and swallowed, to be afterwards ejected from the stomach.

Hæmatemesis may occur under the following circumstances:

(a) In ulcer of the stomach or ulcer of the duodenum. Here the blood is usually vomited at considerable intervals, and may be copious.

(b) In cancer of the stomach. In these cases the blood is, as a rule, less copious, and only appears at a late period of the disease,

when cachexia is well advanced. It may be vomited frequently and at short intervals.

(c) Gastric catarrh occasionally causes hæmatemesis, and streaks of blood may be found in the vomit after any prolonged attack of vomiting.

(d) Obstruction to the venous return in the portal vein causes congestion and varicosities of the capillaries and venules in the stomach, as well as elsewhere in the portal circuit. This often gives rise to fairly free hæmorrhage into the stomach, causing hæmatemesis. The condition is furnished by cirrhosis of the liver, by cardiac disease (nutmeg liver), and by cancer of the liver.

(e) Diseases of the spleen.

(f) Local injuries, such as corrosive-poisoning, the passage of the stomach-tube, etc.

(g) Aneurism of the aorta, opening into the œsophagus or stomach.

(h) Blood states giving rise to hæmorrhages—*e.g.*, purpura, scurvy, septic inflammation, acute yellow atrophy of the liver.

(i) Leukæmia.

Of the above-enumerated causes, the commonest are ulcer of the stomach and cirrhosis of the liver.

### HÆMATURIA.

The passage of blood from the urinary channel imparts to the urine in many cases a smoky, dark hue; this is characteristic of bleeding from the kidneys, especially if the quantity of blood lost be small. Hæmorrhage from the lower urinary passages is more commonly brighter in colour, and is often clotted. It may be recognized by microscopical examination, the red corpuscles being seen as discs without a nucleus. Sometimes the cells are spherical and sometimes shrunken and crenated.

The methods best adapted for the detection of blood in the urine are described in the article on the Examination of the Urine, at p. 533. The clinical significance of the symptom is considered at p. 505.

**HÆMIN TEST.** See *Urine Examination*, p. 533.

### HÆMOGLOBINURIA.

The presence of blood pigment in the urine, free from the red cells, which may or may not be also found. The methods of

detecting the hæmoglobin are described in the article on the Examination of the Urine, at p. 533, and the diagnostic significance of the symptom is discussed in the article on Abnormalities of the Urine (p. 505).

### HÆMOPTYSIS (Gr. *αἷμα*, blood; *πίσις*, a spitting).

A bloody expectoration is due to disease or injury in some portion of the respiratory tract. As a rule there is no difficulty in recognizing blood when it appears in the sputum. Doubtful cases may in some instances occur, and the reader is referred to the article on the Sputum (p. 385) for a description of the methods of detecting hæmoptysis.

In all cases a careful investigation of the upper air passages, as well as of the chest, is to be made, and the sputum is to be subjected to microscopical as well as naked-eye examination. At times it may be difficult to decide if the blood come from the air passages or from the stomach. A comparison of the distinguishing points of hæmoptysis and hæmatemesis (Osler) may be consulted at p. 155.

**Causes.**—Naso-pharyngeal affections (polypi, rhinitis, adenoids, pharyngitis, ulceration, etc.); laryngeal disease (laryngitis; tubercular, syphilitic, malignant ulceration; foreign bodies); phthisis; heart disease; pneumonia; aneurism opening into the bronchi or trachea; tumours and injuries of the lung.

**Copious, bright, frothy blood** is usually due to the erosion of a vessel in a phthisical cavity. It may be the result of the rupture of an aneurism into the air passages. In the latter event the blood may not be especially frothy, but often it is shed with overwhelming rapidity, causing a rapidly fatal termination. In the early stage of phthisis, before excavation has occurred, a sharp hæmorrhage may take place—indeed, it may be the first sign of the disease. Here the activity of the localized tubercular inflammation has caused the erosion of a vein.

A mucous sputum, tinged throughout with blood or copiously streaked with blood, is commonly due to increased tension in the pulmonary circulation, or to a hyperæmia of one or more regions of the lung. The former condition is seen in disease of the mitral valve, and especially in stenosis of that orifice; also in bronchitis, emphysema, pneumonia, phthisis, œdema of the lung. Hyperæmia is responsible to a large extent for the hæmoptysis of some of the complaints just mentioned, and others—viz., phthisis,

pneumonia, bronchitis, œdema, infarction, gangrene, bronchiectasis. Owing to these disturbed conditions of circulation, the capillaries in the lung readily allow small quantities of blood to escape into the alveoli and bronchioles, whence it is discharged with the excess of mucus thrown out by the activity of the over-nourished mucous membrane.

The **character** of the blood-stained sputum varies. If dark-coloured, or even black, it has been extravasated for some time. This is seen in the dark sputum which persists for some days after a sharp bout of bleeding in phthisis, and in cases of infarct. In croupous pneumonia the sputum is infiltrated with blood in many cases, especially in the early stage of the disease. This is often of a brownish colour—the **rusty sputum**. A similar type of hæmoptysis may be observed in acute tubercular consolidation of the lung, and in gangrene of the lung. The so-called **prune-juice** sputum, a watery, dark, blood-stained expectoration, is seen in cases of œdema and hypostatic congestion of the lungs, in low forms of pneumonia, in cancer, and in gangrene of the lung.

Blood-stained sputum, or a spit composed apparently entirely of blood, may also originate in the larynx, pharynx, nose, or mouth; it is only by careful examination of these regions that the source of the bleeding can be ascertained.

In by far the greater number of instances a fairly abundant hæmoptysis is due to phthisis, either in the initial stages, when it may be copious, but rarely dangerous, or in advanced and excavated lungs, when the bleeding may be sufficient to cause death.

**HÆMORRHAGIC ERUPTIONS.** See **Skin Eruptions**, p. 368.

#### **HANDS, Abnormalities of.**

Reference is made elsewhere to certain abnormal conditions of the hands (Contractures, p. 104; Clubbed Fingers, p. 145; Spade-hand, p. 373; Heberden's Nodes, p. 163; Morvan's Disease, p. 201). It is unnecessary in this place to consider the local surgical affections of the hand. Attention may be drawn to the effects on the hands of gout and osteo-arthritis.

**Osteo-arthritis.**—The metacarpo-phalangeal and interphalangeal joints are affected; they are painful, swollen, often red, stiff, and filled with effusion. As the disease progresses the joints grate, and the fingers are twisted into deformed shapes. The fingers are often deflected towards the ulnar side, flexed at the metacarpo-



phalangeal joints and hyperextended or flexed at the remaining joints. In some cases the chief damage is the formation of Heberden's nodes. In other and less common instances the affection is acute in its course.



FIG. 26.—OSTEO-ARTHRITIS.

Gout also causes disturbances in the hands. The nails may be ribbed and irregular; the fingers may become enlarged, stiff, and deformed; chalky concretions (tophi) may form under the skin.

**HARRISON'S SULCUS** (Transversely Grooved Chest). See Thorax, Shape, etc., p. 457.

#### HEAD, Size and Shape of.

The head is enlarged in hydrocephalus, rickets, cretinism, osteitis deformans, acromegaly.

A square head (see Caput Quadratum, p. 98) is to be seen in rickets, a rather rounder form in hydrocephalus; in acromegaly the enlargement affects the face more than the cranial bones, giving to the head and face an oval form, with the larger end downward; the contrary is the shape of osteitis deformans—viz., a large head and small face.

**HEADACHE (Cephalalgia).**

Varieties — Migraine — Neuralgia — Dyspepsia and Constipation — Neurasthenia — Anæmia — Affections of the eye, nose, and nasopharynx — Intracranial disease — Syphilis — Fevers — Uræmia — Poisoning by alcohol, nicotine, lead, mercury, impure air — Rheumatism — Gout — Diabetes — Disease of the bones of the head.

Site of the pain — Unilateral headache — Frontal — Vertical — Occipital — Temporal — Generally distributed.

Character of the pain: stabbing, sharp, dull, throbbing, periodical.

Pain in the head may be due to a great variety of disturbing circumstances. The following types of headache and their distinguishing features may be enumerated:

1. **Migraine** (sick headache, bilious headache, hemicrania). The pain is usually situated in one side of the head, involving the distribution of the branches of the fifth nerve; it may be severe, but has not the darting, lancinating character of neuralgia. It is usually accompanied by sickness and vomiting, and is often ushered in by visual phenomena, such as flashes of light, trembling movement of bright specks or zigzag lines, or restricted field of vision.
2. **Neuralgia** of the head is felt as a sharp, darting pain along the course of one of the nerves of the face or head; it is liable to periodical exacerbations, and may often be traced to peripheral irritation of a branch of the affected nerve—*e.g.*, carious teeth. It may also be due to general morbid states, such as gout, debility, malaria. The pain is almost always one-sided, and the nerve may be tender to the touch.
3. **Dyspepsia** and **constipation** are very frequent causes of a dull, throbbing headache, chiefly affecting the frontal and supra-orbital regions; it is usually bilateral, and is aggravated by movement or stooping. It is usually accompanied by signs of indigestion.
4. In **neurasthenia** a vertical headache is common; it is of a tense, pressing or bursting character, and may be diffused over the whole head, or not infrequently is felt as a tight band round the head; it is usually worse in the morning than the evening. A variety of this form is the **clavus hystericus**, experienced by hysterical subjects, who complain of a pain as of a nail being driven into the crown of the head.
5. **Anæmia** is often accompanied by a headache resembling in many respects that of neurasthenia.

6. Disease of the **abdominal organs** generally, and of the **female organs of generation** in particular, frequently gives rise to headache, which is often sharp and radiating, with its chief seat in the occiput; it may, however, affect any region of the head.

7. **Affections of the eyes**, and especially errors of refraction, often give rise to obstinate headache, which usually affects the supra-orbital and frontal regions, but may be seated in the temporal or occipital regions. Disease of the **nose** and **naso-pharynx** causes a very similar type of headache.

8. **Intracranial disease** is very often accompanied by a headache, which is perhaps more common in the occipital than in the other regions of the head. Meningitis, tumours, abscess, etc., are frequent causes. Occasionally, when the disease is strictly localized (*e.g.*, thrombosis), the seat of the pain may give an indication as to the locality of the disease; but, as a rule, this is not a trustworthy guide.

9. **Syphilis**, independent of intracranial syphilitic growths, causes a persistent, severe headache; it is often occipital, but may be general, and is almost invariably worse at night.

10. **Fever** of any description may cause headache; an intense frontal, vertical, or perhaps general headache, is characteristic of the first week of typhoid fever.

11. In **nephritis** headache is a common symptom; it is often frontal, but may change its position; it is usually to be regarded as a sign of uræmia. A similar headache is experienced in other toxic states—*e.g.*, chronic poisoning by **alcohol**, **nicotine**, **lead**, **mercury**, **impure air**.

12. In **rheumatism**, **gout**, and **diabetes**, headache of a general distribution is common.

13. Disease of the **bones of the head** gives rise to severe pain. In the neighbourhood of the ear (middle-ear disease, mastoid abscess) this is often tubercular; in other regions, especially the frontal, it is often syphilitic.

Some guidance may be obtained by comparing the chief seats of the pain in the head and its character in the different conditions causing it.

**Site of the Pain.**—*Unilateral*: Migraine; neuralgia.

*Frontal*: Dyspepsia; constipation; affections of the eye (especially errors of refraction), of the nose, and of the naso-pharynx; fevers (especially typhoid); nephritis; neurasthenia and anæmia frequently.

*Vertical*: Fevers; neurasthenia; anæmia; hysteria (clavus hystericus); intracranial tumours and inflammation.

*Occipital*: Disease of the abdominal organs and of the female genital organs; syphilis; intracranial disease; eye affections.

*Temporal*: Affections of the eye, ear, and nose; neuralgia; migraine.

*Generally or indiscriminately distributed*: This is often the case in neurasthenia, anæmia, disease of the abdominal and female genital organs, dyspepsia, intracranial disease, syphilis, fevers, various toxic conditions of the blood—*e.g.*, nephritis, rheumatism, gout, poisoning by alcohol, lead, nicotine, mercury, impure air.

**Character of the Pain.**—*Stabbing or lancinating*: Neuralgia; hysteria; migraine may cause this type of pain; inflammatory disease of ear.

*Sharp, severe*: Migraine; neuralgia; hysteria.

*Dull, often severe*: Dyspepsia; constipation; intracranial disease; syphilis (worse at night); neurasthenia (worse in the morning); disease of the female genital organs; disease of the eyes and nose; fevers (especially typhoid); nephritis; various toxic states (gout, rheumatism, plumbism, etc.).

*Throbbing*: Dyspepsia; constipation; inflammatory affections of the ear, etc.

*Periodical*: Neuralgia.

### HEART-BLOCK.

The conduction of the peristaltic wave of contraction passing from the entrance of the great veins into the auricles, across the auricles, and on to the ventricles, is conducted by means of special muscle fibres which connect the auricles with the ventricles. In some diseased conditions, of which Stokes-Adams disease is an extreme example, the conducting fibres are inefficient, and heart-block results. (See Arrhythmia, p. 40.)

### HEARTBURN (Cardialgia).

As a result of imperfect digestion, a pain is often felt in the epigastrium. When it is localized in the præcordial region, as is not infrequently the case, it is termed heartburn, and may be felt in the back, below the left shoulder-blade. It is probably caused by the irritating effect of the gastric acids upon the cardiac end of the stomach and on the lower end of the œsophagus.

**HEART-SOUNDS.**

The normal heart-sounds should be carefully studied; any departure from the usual rhythm, the relative loudness of the individual sounds, or from their normal quality and tone, must be noted. It should be borne in mind that much information concerning the state of the heart and other organs can be obtained from this source; and if murmurs are present it is even more necessary to study the condition of the heart-sounds. The subject is considered at p. 424.

**HEBERDEN'S NODES.**

Bony prominences found chiefly at the distal interphalangeal joints; they occur most commonly in elderly subjects, and most frequently in females. They are signs of osteo-arthritis, but are favourable phenomena, as this form of the affection is not likely to take on the serious and progressive character so often observed in the disease.

**HEBETUDE** (L. *hebetō*, to make blunt). See **Unconsciousness**, p. 498.

**HELLER'S TEST** (for Albumin). See **Urine Examination**, p. 531.

**HELLER'S TEST** (for Blood in the Urine). See **Urine Examination**, p. 533.

**HEMIANOPSIA** (*ἡμιος*, half; *ἄν*, privative; *ὤψ*, the eye).

A loss of sight in one half of the field of vision. It may occur in one or both eyes, and is observed in several forms, according to the situation of the lesion in the optic path (see Fig. 72, p. 546). The subject is considered more fully in the article on Disturbances of Vision (p. 543).

**HEMIPLEGIA** (Gr. *ἡμιος*, half; *πληγῆ*, a stroke).

The term is used to denote loss of voluntary muscular power of the whole or a part of one lateral half of the body, the result of a cerebral lesion. One-sided paralysis often follows a lesion of the peripheral motor nerves, and occasionally may result from spinal

lesions; in these two latter conditions the word 'hemiplegia' is not, strictly speaking, applicable.

The subject is considered in some detail in the article on Decrease of Movement (p. 232 *et seq.*).

### HEMISYSTOLE.

In consequence of imperfect conduction of the muscular contraction from the auricles to the ventricles (heart-block), it may happen that one ventricle may beat less frequently than the other. By a comparison of the apex-beat, carotid and jugular pulsation, together with alterations in the second sound of the heart, it is possible to diagnose the condition. (See Arrhythmia, p. 38.)

**HERPES FACIALIS.** See **Skin Eruptions**, p. 367.

### HICCUGH.

A sudden repeated contraction of the diaphragm. It may be the result of slight irritation of the stomach or may be due to more serious disease. Peritonitis, abdominal tumours, ulcer or catarrh of the stomach, disease of the liver, and intestinal obstruction, are among the more important abdominal affections by which a recurring and often obstinate hiccough is produced. Functional and organic nervous diseases may be the cause—*e.g.*, hysteria, emotional disturbances, tumour, or inflammation of the brain and meninges. It may result from lung diseases, toxic states of the blood—*e.g.*, Bright's disease, gout, diabetes, and conditions of extreme prostration, such as the typhoid state.

As a rule, the symptom is of little importance as a guide either in diagnosis or in prognosis, but in the graver conditions its occurrence is often a sign of ill omen.

### HIPPUS.

The alternate contraction and dilatation of a pupil suddenly exposed to light after previous rest in darkness. The oscillation of the pupil may be exaggerated in multiple sclerosis, and in conditions in which the reflexes are increased (see pp. 319, 325 and 342).

**HOARSENESS.** See **Voice, Abnormalities of**, p. 555.

**HYPÆSTHESIA** (ὑπό, diminished; αἰσθησις, a sensation).

Diminution in the perception of sensory stimuli, the result of interruption to the passage of afferent nerve impulses. The condition is considered in the article on Disturbances of Sensation (p. 356).

**HYPALGESIA** (Gr. ὑπό; ἄλγησις, sense of pain).

A diminished acuteness of perception of painful stimuli. The subject is considered in the article on Disorders of Sensation (p. 356).

**HYPERÆSTHESIA** (Gr. ὑπέρ, excess; αἰσθησις, a sensation).

An exaggeration of the perception of common tactile sensations, resulting commonly from functional nervous disturbances, or from direct irritation of the sensory path. This usually takes place at the posterior spinal roots. The subject is further considered in the article on Disorders of Sensation (p. 356).

**HYPERALGESIA** (Gr. ὑπέρ; ἄλγησις, sense of pain).

An exaggerated sensibility to painful stimuli. (See the articles on Disorders of Sensation, p. 356, and Pain, p. 268.)

**HYPERCHLORHYDRIA.**

An excessive amount of hydrochloric acid in the gastric juice. (See Stomach, Examination of, p. 392.)

**HYPEROREXIA** (Gr. ὑπέρ; ὄρεξις, an appetite).

An abnormal increase in the desire for food. It may be seen in diabetes, in hysteria, in idiocy, and in dementia. (See Appetite, p. 35.)

**HYPERPYREXIA** (Gr. ὑπέρ; πῦρ, fever heat; ἔχω, to hold).

A body temperature exceeding 106° F. It may occur in the course of some of the fevers, notably in acute rheumatism, typhoid fever, malaria, and sunstroke. It is more commonly seen as the termination in fatal cases of cerebral hæmorrhage, injuries of the brain and cervical portion of the spinal cord, typhoid fever, scarlet fever, yellow fever, tetanus, etc. (See Temperature, p. 495.)

**HYPERTROPHY** (Gr. *ὑπέρ*; *τροφή*, nourishment).

An increase in the bulk of an organ or tissue is the result of (a) excessive use of the part; (b) an excessive amount of nourishment; (c) various morbid processes. The last group consists of (i.) degenerative changes—*e.g.*, muscles, kidneys, arteries; (ii.) inflammatory changes—*e.g.*, subcutaneous inflammatory swellings, enlargements of the tonsils, salivary glands, etc.; (iii.) gaseous distension of hollow organs or tissues—*e.g.*, meteorism, pseudo-cyesis, surgical emphysema; (iv.) dropsical or other effusions into serous cavities and subcutaneous tissues; (v.) new growths.

The subject is further considered in the article on Trophic Disturbances (p. 487).

**HYPOCHLORHYDRIA.**

A defective amount of hydrochloric acid in the gastric secretion. (See Stomach, Examination of, p. 397.)

**HYPOCHONDRIAC REGIONS.**

On each side of the body the hypochondriac region is bounded above by a line drawn horizontally across the chest at the level of the sixth rib in the nipple line; internally by the costal margin of its own side as far as the point where the costal margin is joined by a vertical line passing through the mid-point of Poupart's ligament, thence by the latter line as far as the point where it is crossed by a horizontal line joining the lowest points of the two tenth ribs; inferiorly, the last-named line is the boundary. The region extends back to the vertebral column.

The various abnormalities to be found in this region are enumerated in the article on Abdominal Shape, etc., at p. 6; on Abdominal Percussion, at p. 16; and in that on Pain, at p. 272.

**HYPOGASTRIC REGION.**

That portion of the abdomen lying immediately above the symphysis pubis. It is bounded above by a horizontal line joining the two anterior superior spines of the ilia; at the sides by two vertical lines passing through the mid-points of Poupart's ligament; below by the pubic bones. Abnormalities in this region are described in the articles on the Shape, etc., of the Abdomen, at p. 6; Percussion, p. 15; and in that on Pain, at p. 274.



**ILIAC REGIONS.**

On each side of the body the iliac region is bounded above by a horizontal line joining the two anterior spines of the ilia; internally by a vertical line passing through the mid-point of Poupart's ligament; below and externally by that ligament,

The abnormalities to be found in this region are referred to in the articles on Abdominal Shape and Movements, Pain, etc., at pp. 6 and 273.

**INCONTINENCE OF FÆCES.**

Inability to maintain control over the evacuation of the rectum, owing to disease of the nervous system. The conditions under which this symptom is produced resemble in many respects those causing incontinence of urine. The rectal function is, however, less sensitive than the vesical, and is less useful as an aid to diagnosis. (See Defæcation, p. 116.)

**INCONTINENCE OF URINE (Enuresis).**

Inability on the part of the patient to prevent the escape of urine from the bladder. This may occur either as a constant dribbling or as an intermittent evacuation. It may be observed in three forms: (*a*) active incontinence, due to an excessive excitability of the reflex mechanism; (*b*) passive incontinence, the result of a defective reflex mechanism, or weakness of the bladder muscles; (*c*) false or paradoxical incontinence, the constant dribbling of urine from an over-full bladder. The subject is considered in more detail in the article on Disorders of Micturition (p. 195).

**INFRA-AXILLARY REGIONS.**

That portion of the side of the thorax on each side, bounded before and behind by the axillary folds and above by a line drawn horizontally at the level of the third costal cartilages. The abnormalities to be found in this region are discussed in the articles on the Shape, etc., of the Thorax (p. 465), Percussion (p. 450), Auscultation (p. 407), and Pain (p. 271).

**INFRACLAVICULAR REGIONS.**

That portion of the thorax lying below the clavicle down to the level of the third costal cartilage in front and on each side.

The abnormalities to be found in this region are referred to in the articles on the Shape, etc., of the Thorax (p. 465), Percussion (p. 450), Auscultation (p. 407), and on Pain (p. 270).

### **INFRASCAPULAR REGIONS.**

That portion of the back of the thorax lying below the level of the inferior angle of the scapula, on each side, and extending forward as far as the posterior axillary line. The abnormalities to be observed in this region are referred to in the article on the Shape, etc., of the Thorax (p. 465), Percussion (p. 450), Auscultation (p. 407), and Pain (p. 271).

### **INGUINAL REFLEX.**

Irritation of the skin of the inner side of the thigh by stroking or pinching causes a contraction of some of the fibres of the internal oblique muscle above and along Poupart's ligament. This reflex takes the place in females of the cremaster reflex. It is, however, also seen in males (see p. 343).

### **INOCULATION, Diagnosis by.**

Negative or inconclusive results of the examination of fluids or tissues for micro-organisms may be supplemented by the inoculation of animals. The effects of this procedure afford invaluable aid in diagnosis, and especially in tuberculous affections. The subject is one for the laboratory, and the reader is referred to the more special treatises on the subject.

### **INTERSCAPULAR REFLEX.**

On stroking or otherwise irritating gently the skin over the spinal edge of the scapula, it is drawn towards the spine. (See Reflexes, p. 343.)

### **INTERSCAPULAR REGIONS.**

That portion of the back lying on each side between the vertebral border of the scapula and the median line. The abnormalities to be found in this region are referred to in the articles on the Shape, etc., of the Thorax (p. 465), Percussion (p. 450), Auscultation (p. 407), and Pain (p. 271).

**INTESTINAL OBSTRUCTION.** See Defæcation, p. 118.

**INTESTINAL SAND.**

The presence of a sand-like material in the stools has been frequently observed. It may be due to multitudes of minute gall-stones; or it may be the remains of certain articles of food which have resisted digestion, and which have become coated with lime salts; or it may be a mass of fine, gritty particles, formed in the bowel under pathological circumstances (usually colitis), and consisting mainly of lime salts.

**IRRITABLE AORTA.**

An exaggerated pulsation of the aorta, seen and felt in the epigastrium. It is frequently observed in dyspeptic and neurotic persons, and is without serious prognostic significance. (See Abdominal Shape, etc., p. 12.)

**ITCHING (Pruritus).**

A variety of paræsthesia found commonly in various skin affections, of which urticaria is a characteristic example; less frequently it is a symptom of general diseases. In diabetes a severe itching of the external genitals and their immediate neighbourhood is frequently found; at times the irritation may extend to more distant regions of the surface. Jaundice from any cause is often attended by general itchiness of the skin. Neurotic subjects may suffer from itch, which is most frequently situated at or near the genitalia. Organic affections of the nervous system may also be accompanied by itching, but without sufficient constancy to be a diagnostic guide. It is occasionally the form assumed by the aura of epilepsy, when it is often situated in the hands and arms. It may also accompany gout and lead-poisoning. Itching at or near the anus may be the result of piles or thread-worms. A senile form of itching without obvious cause is not uncommon.

**JAFFE'S TEST (for Indican).** See Urine Examination, p. 538.

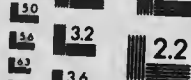
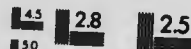
**JAUNDICE (Icterus).**

The presence of bile pigment in the blood. It is easily recognized by the yellow discoloration of the conjunctivæ, of the skin, of the mucous membranes, of the urine, and often of the perspiration. In severe cases the quantity of bile in the blood is so great that the surfaces become olive green (black jaundice). The tests



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by which bile may be detected in the urine are described in the article on the Examination of the Urine (p. 538).

The yellow colour is generally first seen in the conjunctivæ; it is also plainest where the skin is thinnest—*e.g.*, the skin of the trunk and other regions which are habitually covered. It is useless to look for the yellow colour by artificial light, unless the discoloration be intense. In examining the conjunctivæ one must not be misled by the presence of a thickened yellowish mass of conjunctival tissue close to the edge of the cornea, which, in spite of its name (*pinguecula*; L. *pinguis*, fat), does not contain fat. The presence of bile in the urine and in the sweat is often first revealed to the patient by yellow discoloration of his underclothing. Other symptoms referable to the presence of bile in the blood, regardless of its cause, may be briefly enumerated: Alteration in the character of the stools: constipation is usual, though diarrhœa may occur; the motions are colourless, have an offensive odour, and may contain most of the fat which has been ingested. Itching of the skin (*pruritus*) commonly occurs. The pulse is slowed, especially in the catarrhal form. *Dyspepsia* commonly accompanies jaundice, with nausea, flatulence, anorexia, irritability of temper, etc.

Two forms of jaundice are usually described—(a) obstructive or hepatogenous, and (b) toxæmic or hæmatogenous jaundice. The former includes affections of the bile-ducts preventing the outflow of bile, whereby the tension in the biliary channels is raised above that in the capillary bloodvessels. The toxæmic form is not so obvious in its nature. It has hitherto been assumed that the jaundice observed in some cases of pneumonia, in yellow fever, in pyæmia, etc., is the result of hæmolysis, by which colouring matter of the blood is liberated from the red cells, and so tinges the skin. There are, however, reasons for believing that the pigment found in the skin in such toxic cases, though it originates in the blood, is actually formed in the liver. The condition might therefore be termed hæmo-hepatogenous jaundice.

(a) **Obstructive jaundice** occurs in the following affections, which include most of the conditions of diagnostic importance: Cholangitis, catarrhal and infective; obstruction of the common bile-duct (by gall-stones, cicatrices, tumours, pancreatitis); malignant disease of the liver, in which jaundice appears in about half of the cases (Monro); cirrhosis of the liver (here the jaundice is often slight or absent); Weil's disease. (See the Table of Enlargements of the Liver, p. 190.)

(b) **Toxæmic** or **hæmo-hepatogenous jaundice** may be in part a result of catarrh of the smaller biliary canals brought about by the toxic condition of the blood, and in part a result of the excessive quantity of blood pigment presented to the liver owing to the hæmolytic action of the poisons in the blood. This form is exemplified in those cases of jaundice occurring in pneumonia, pyæmia, small-pox, and yellow fever. It may also be the consequence of more destructive disease of the liver—*e.g.*, phosphorus-poisoning and acute yellow atrophy of the liver. The jaundice of new-born infants may possibly be a catarrhal form, though in some instances it is toxic.

### **JAW-JERK.**

In disease of the nerve centres causing increased reflexes one may find this symptom. It is produced by percussing the finger laid across the lower teeth or just above the chin, with the mouth open. An involuntary contraction of the masseters jerks the lower jaw upwards, and, on pressure being kept up, clonic movements of the jaw may in extreme cases occur. (See Reflexes, p. 340.)

### **JENDRASSIK'S REINFORCEMENT OF THE REFLEXES.**

In order to obviate the inhibitory action of the mind on reflex acts—*e.g.*, during the investigation of the patellar reflex—it is advisable to distract the attention of the patient from the proceedings. As suggested by Jendrassik, he is directed to clasp his hands in front of his chest, and to endeavour to pull them asunder, the eyes being meanwhile fixed on the ceiling.

Other means of diverting his attention may be adopted, such as by asking him questions as to his health, etc.

### **JOFFROY'S SIGN.**

A patient suffering from Graves' disease is directed to look upward, his head being kept steady, his forehead remains smooth, instead of being thrown into transverse wrinkles, as occurs in normal individuals.

### **JUGULAR PULSATION.**

Pulsation of the veins occurs under certain abnormal conditions of the right heart—*viz.*, dilatation of the auricle, dilatation of

the ventricle, with incompetence of the tricuspid valve, hypertrophy of the walls of either chamber.

The internal jugular vein is in some respects the most convenient vessel to observe. The subject is further considered in the article on the Venous Pulse (p. 312).

### KERNIG'S SIGN.

A spasmodic contraction of the hamstring muscles, observed chiefly in meningitis. On flexing the legs at the hips it is found that the legs cannot be extended at the knees (see p. 251).

### KIDNEYS, Examination of.

Situation of kidney—Method of examination—Movable kidney—Floating kidney—Percussion of the kidney—Enlargement of the kidney—Sarcoma—Carcinoma—Cystic disease—Hydro-nephrosis—Pyonephrosis—Perinephric abscess.

The normal kidneys may in many cases be recognized by palpation, but not by inspection nor by percussion. On the right side the kidney extends down in the abdomen to a level of about 1 inch above the umbilicus, and about 3 inches out from the middle line; the left kidney is about  $\frac{1}{2}$  inch higher. In order to palpate the organ the patient is laid flat on his back, with his knees drawn up, and his head slightly raised on a pillow. One hand of the examiner is passed behind the patient's back, so that the lower two ribs and the region immediately below them rest on the hand. By this hand the kidney is pressed forward, while the other hand presses firmly and steadily on the abdomen opposite the kidney, exploring the region under the costal margin. By directing the patient to breathe deeply, and by sinking the hand deeper into the abdomen at the end of each expiration, the lower end of the kidney, especially the right, may be distinctly felt in those cases in which the abdominal walls are easily relaxed. This procedure is by no means always successful, even with an experienced examiner. Instead of using two hands, some prefer to use one only, the fingers being placed dorsally in the position just described, and the thumb pressing in front over the lower end of the kidney. If the greater part of the organ can be thus felt, the kidney is said to be **movable**; if it can be bodily displaced to the lower part of the abdomen, or across the middle line to the other side of the abdomen, it is known as a **floating kidney**.

By this method of examination one may also detect enlargement of either kidney, or alteration in its consistency or shape.



Instead of the dorsal position, some observers prefer to have the patient sitting or standing, as in this posture it may be easier to palpate the enlarged or movable kidney.

While percussion is useless as a means of examining the normal kidney, it is of much assistance when the organ is enlarged or displaced. In the former case the swelling of the kidney tends to come forward toward the umbilical region, and may be dull on percussion; the colon (ascending if the right kidney is under consideration, descending if the left) lies in front of the tumour, and may generally be recognized as a band of resonance lying more or less vertically over the enlarged kidney. If the kidney is floating, the percussion-note may be somewhat dulled over it, but one may generally recognize a certain tympanitic quality of note immediately below the border of the liver. By this sign one may distinguish the floating kidney from an enlarged gall-bladder, the dulness of which is continuous with that of the liver.

Enlargements of the kidney may be due to:

(a) **Sarcoma**.—If primary, it usually occurs in infancy or childhood; it is, however, very frequently secondary, and may occur at a later age.

(b) **Carcinoma** may be primary, and is found as a rule in middle-aged or elderly subjects. It is believed that many of the tumours of this description owe their origin to the presence of remnants of adrenal tissue in the kidney.

In a large number of the cases of malignant kidney tumours there is hæmaturia, accompanied by renal colic, due to the passage of blood-clots through the ureter; there is, however, in most instances, pain in the tumour independent of the passage of blood. The kidney may retain its shape fairly well, and is, as a rule, firm in consistence. Cachexia is usually well marked when the tumour is palpable.

(c) **Cystic Disease of the Kidneys**, a congenital affection involving both organs as a rule; it may be observed in infancy, or may not be detected till adult life. The kidneys may grow to an enormous size, retaining their shape, but in many cases the nodular cystic surface can be recognized by palpation. The urine is copious, of low specific gravity, and may contain little or no albumin.

(d) **Hydronephrosis**, a dilatation of the pelvis and calices of the kidney, forms a cystic, fluctuating tumour, which may be either bilateral or unilateral, in accordance with the cause. It is due to a block in the ureter (calculus, hypertrophied bladder, kinking of ureter, pressure of tumours). The cyst contains a clear fluid, the constituents of which are those of dilute urine.

(e) **Pyonephrosis**, a condition resembling the last-named, but with purulent contents, and, unlike hydronephrosis, this affection gives rise to severe constitutional symptoms (fever, rigors, sweating).

(f) **Perinephric Abscess** gives rise to a tumour, very similar in situation (though tending to advance rather to the lumbar region than to the umbilical). It is in its earlier stages less firm or fluctuating and more 'boggy' or œdematous than the renal tumours. The constitutional disturbance may be severe.

The enlargement of the kidneys due to inflammatory or waxy changes, though considerable in some cases, does not come under our present consideration. The changes in these affections are not profitably studied by physical examination of the kidneys themselves, but of the urine. See the articles on the Examination (p. 526) and on the Abnormalities of the Urine (p. 502).

#### **KNEE-JERK (Patellar Reflex).**

On flexing the leg at the knee a condition of reflex readiness of the extensor muscles ('myotatic irritability'—Gowers) is produced. A slight tap on the patellar tendon is then sufficient to evoke a contraction of the muscles in question. An exaggeration or a diminution in the activity of this phenomenon may be noted in one or both limbs in various diseased conditions (see p. 338).

#### **KORSAKOFF'S SYNDROME.**

In cases of chronic alcoholism a complex of symptoms described by Wilks and Jackson is to be recognized. Its constituents are polyneuritis, loss of memory, and hallucinations as to experiences which the patient imagines he has undergone. The condition was termed a polyneuritic psychosis by Korsakoff, after whom the symptom-complex is generally named.

#### **KUSSMAUL'S SIGN.**

A swelling of the jugular veins occurring during the act of inspiration, observed in cases of adherent pericardium.

#### **KYPHOSIS (Gr. κῦφός, a hump).**

A posterior convexity of the spinal column, which may be merely an exaggeration of the normal curve of the dorsal vertebræ, or may include the lower part of the spine which is normally convex toward the front. It may be the result of simple weak-

ness of the muscles supporting the column. The spinal form of osteo-arthritis may show kyphosis, as in the affection known as spondylose rhizomélique (Strumpell-Marie), which is by some observers regarded as a separate disease. In rickets the deformity is frequently observed. The barrel-shaped chest of emphysema owes its form in some measure to kyphosis.

Pott's curvature of the spine is due to caries of the bodies of the vertebræ, which fall together, causing one or more spinous processes to project. Unusual prominence of the seventh cervical spine, or of the eighth, ninth, or tenth dorsal spines, is not uncommon, without obvious cause.

**LAGOPHTHALMOS (Hare's Eye)** (Gr. λαγῶς, a hare; ὀφθαλμός, the eye).

Inability to close the eye, the result, as a rule, of facial paralysis. The term is applied to this condition in accordance with the popular belief that the hare sleeps with its eyes open (see p. 222).

**LATERITIOUS URINE** (*L. later*, a brick).

The presence of urates imparts to urine a reddish colour (often pinkish white in children). The sediment formed by the insoluble urates resembles wet brick-dust, and is sometimes spoken of as the lateritious deposit. It is dissolved on heating (see p. 528).

**LEGAL'S TEST (for Acetone).** See **Urine, Examination of**, p. 537.

**LETHARGY.** See **Unconsciousness**, p. 498.

**LEUCOCYTOSIS** (Gr. λευκός, white; κύτος, a hollow vessel).

Number and varieties of white corpuscles in normal blood.

Physiological leucocytosis: of digestion, of pregnancy; of newborn infants.

Pathological leucocytosis: Inflammatory; chemotaxis; phagocytosis; immunity — Post-hæmorrhagic — Cachectic — Antemortem—Experimental or toxic.

Leucocytes in various diseased conditions: Specific infectious diseases; local infectious inflammations; serous inflammations; catarrh; malignant disease; diseases of the blood and blood-forming organs; diseases of the liver, kidneys, and sundry other organs and tissues.

Normal blood contains from 5,000 to 12,000 white corpuscles in each cubic millimetre of the fluid, an average count being about 8,000. This wide variation in numbers is produced by passing physiological processes, the chief of which is the state of digestion and absorption. It also depends largely on the peculiarity of the individual.

When the number of white cells found in the blood can be shown to be decidedly greater than is usually present in the blood of that individual, or of one resembling him in age and other circumstances, the condition is termed **leucocytosis**.

The white blood-corpuscles (leucocytes) may be differentiated into several distinct forms, a description of which, and of the methods to be adopted in making an examination of the blood, will be found in the article on Blood Examination, by Dr. Houston (p. 67 *et seq.*). The more important varieties are: small and large lymphocytes, polymorphonuclear leucocytes, eosinophile leucocytes, mast cells, myelocytes.

When the term 'leucocytosis' is used without qualification, it indicates increase in the polymorphonuclear leucocytes; if the eosinophiles are in excessive numbers, the condition is termed **eosinophile leucocytosis**, or **eosinophilia**; abnormal increase in the number of lymphocytes is termed **lymphocytosis**.

1. **Physiological Leucocytosis**.—Leucocytosis may occur to a moderate extent apart from disease. The following examples may be cited:

(a) **Digestion Leucocytosis**.—One hour after a meal an increase may be found in all the white cells, which attains its maximum three or four hours later. The process of digestion and absorption causes an activity in the lymphoid tissue of the alimentary tract, with a proliferation of the mononuclear cells of those tissues; at the same time the thoracic duct conveys lymph cells in abundance to the circulation, the result being an increase of the numbers of lymphocytes in the blood. The absorption of albumins from the digestive tract into the circulating blood calls forth the activity of the bone-marrow, which, in obedience to the stimulus of the chemotactic properties (see p. 176) of the absorbed albumins, discharges into the blood a number of the polynuclear white cells, causing them to be found in excess when the blood is examined. We have thus a mixed leucocytosis during digestion, which is usually moderate in extent, showing a count of perhaps 3,000 or 4,000 leucocytes in excess of the usual number.

Digestion leucocytosis may fail to appear in conditions where the act of digestion is prolonged and imperfect, and this is especially the case in cancer of the stomach, while in chronic gastric catarrh and ulcer of the stomach digestion leucocytosis is said to occur as a rule. This phenomenon may therefore in obscure cases be possibly of some diagnostic value.

(b) During the later months of **pregnancy** a mixed leucocytosis is usually present, which disappears soon after parturition. This is most constantly observed in primiparæ, and is probably the result of cellular activity in the uterus, breasts, and vascular system.

(c) **New-born infants** show a considerable lymphocytosis, which falls nearly to normal in the first week, but rises again in a few days. For some months the lymphocyte count is much above normal.

2. **Pathological Leucocytosis** is of more interest to the diagnostician. The following varieties may be recognized: (a) inflammatory; (b) post-hæmorrhagic; (c) cachectic; (d) ante-mortem; and (e) experimental leucocytosis. It is almost exclusively the first of these divisions that is of practical importance to the clinician.

(a) **Leucocytosis of Inflammation.**—In many inflammatory affections leucocytosis is a constant feature; a knowledge of the conditions in which it may be expected is therefore likely to be of assistance in diagnosis. In order to render the subject intelligible, a brief reference is desirable to recent views on inflammation, and on the defensive processes at work in the organism.

*Chemotaxis.*—It has been shown that certain living cells—*e.g.*, the white cells of the blood—are attracted to or repelled from various chemical or bacterial irritants. This sensitiveness of the cells was termed **chemotaxis** by Pfeiffer, **positive chemotaxis** when the cell approaches the irritant, **negative chemotaxis** in the rare instances (denied by many observers) that the cell is repelled by the irritant. It is found that this chemotactic property is possessed by many, but not by all, pathogenic organisms, as well as by certain chemical substances. The introduction of such a substance into the circulation results in the appearance in the blood of a vast army of newly-arrived white cells, of which the polymorphonuclears form the principal part. It is probable that the increased number of white cells is effected by a new production of leucocytes in the viscera, and by an increased supply of them

from all available sources. This increase in the white cells is often preceded by a period immediately following the infection, in which the number of leucocytes is diminished (leucocytosis). In cases of great severity hypoleucocytosis may persist, and death results without the appearance of leucocytosis. The more rapidly leucocytosis follows the initial decrease the better is the defensive reaction of the system to the infection, and the greater is the hope of recovery. As a result of this change in the blood the bacterial or chemical poison is, in favourable cases, rendered innocuous.

*Phagocytosis.*—It was shown by Metchnikoff that certain cells, mainly the leucocytes and also endothelial and other tissue cells, have the power of embodying micro-organisms in their substance and destroying them. This process he termed **phagocytosis**, and the cells **phagocytes**. By this process, according to Metchnikoff, the animal is rendered immune to the disease produced by that micro-organism, and he attributed to the cell alone this vastly important function. It has been demonstrated by Ehrlich and many other observers that the process whereby immunity is established is by no means so simple as the above theory would indicate. The blood-plasma contains the active principles which deal with the bacteria in the blood. By accepting this opinion Metchnikoff has been able so to modify his theory of phagocytosis as to amplify it and bring it into accordance with the later views. He now regards the destruction of bacteria as due to the combined action of two principles contained in the plasma—the **immune body**, or **fixator**, and the **complement**, or **alexine** both derived mainly, if not entirely, from the leucocytes. The agglobing of the micro-organism by the cell is not, then, the essential act of phagocytosis, but may be taken as the measure of the immunizing process. The recent work of A. E. Wright indicates the presence of a body in the plasma termed by him **opsonin**, the amount or activity of which in the blood of an individual determines the degree of his resistance to the toxic substance of which it is the opponent. A distinct opsonin is apparently necessary for each micro-organism, and it has been demonstrated that opsonin is a separate body from the complement.

It may, therefore, be stated, in Ewing's words, that 'leucocytosis represents Nature's attempt to rid the blood and the system, by means of leucocytes and their products, of the bacterial and toxic causes of disease.'

We can now understand that the degree of leucocytosis present is to be taken as a measure, not of the virulence of the infection, but of the activity of the defensive reaction.

It is in this group, the inflammatory leucocytoses, that the chief diagnostic interest lies; but before enumerating the conditions characterized by increase in one or other form of white cell, it will be more convenient to mention the remaining groups of pathological leucocytosis.

(b) **Post-hæmorrhagic Leucocytosis.**—The polymorphonuclear cells are chiefly affected, but the eosinophiles and lymphocytes may be increased. The more extensive and rapid the hæmorrhage, the more pronounced is the leucocytosis. Transfusion still further increases the leucocytes, which again diminish in number when the blood is regenerated.

(c) **Cachectic Leucocytosis.**—Chronic anæmia in cachectic conditions may cause leucocytosis, owing to the increased activity of the bone-marrow, the hydræmia, and the lowered blood-pressure, which are features of chronic anæmia.

A more important factor is local inflammation, occurring in or around malignant growths, syphilitic or tubercular formations. The toxins which are elaborated by new growths do not seem to have any effect on leucocytes. In the cachexia arising from these conditions leucocytosis should, therefore, suggest inflammatory or necrotic complications.

(d) **Ante-mortem Leucocytosis.**—Shortly before death, even from diseases which usually do not produce leucocytosis, a definite increase in the white cells is observed. It may be merely the accumulation of white cells in the capillaries, owing to failure of the circulation.

(e) **Experimental Leucocytosis.**—It has been found that the administration of various substances—drugs, bacteria, organic extracts, etc.—is followed in many instances by leucocytosis: among the drugs, potassium chlorate, phenacetin, arsenic, chloroform, camphor, oil of cinnamon, etc. The subcutaneous or intravenous injection of almost any pathogenic bacterium will cause positive leucocytic chemotaxis. Various bacterial and other extracts have been shown to produce leucocytosis; the results of putrefaction, extracts made from certain animal tissues, peptone, nuclein, bacterial proteins, have given definite leucocytosis. Koch's tuberculin gives a moderate and transitory polynuclear leucocytosis at the height of the reaction.

It is possible that the artificial production of leucocytosis, whereby the bactericidal power of the blood is increased, may be of therapeutic service. Hitherto the results have not been very encouraging.

The following is an epitome of the results obtained by examining the leucocytes in various pathological conditions :

1. **Specific Infectious Diseases.**—**Variola** shows a moderate degree of leucocytosis, mainly lymphocytic. When pustulation is established, there is no great increase in the polymorphonuclear cells, which only occurs if boils, abscesses, or other inflammatory complications arise.

**Varicella** and **vaccinia** present a slight or moderate mixed leucocytosis, in which the lymphocytes preponderate.

**Scarlet Fever.**—A considerable polynuclear leucocytosis is observed, reaching a maximum on the second day of the rash, then diminishing. The degree of leucocytosis corresponds with the intensity of the disease, and especially of the angina, rather than with the range of temperature. Lymphocytes at first may be diminished. Eosinophile cells, which in most acute polynuclear leucocytoses are decreased or absent, are in scarlatina often increased especially in the later stages of the febrile period.

**Diphtheria.**—Leucocytosis to a considerable extent, and mainly polynuclear, is the rule. In many cases, however, especially in children, lymphocytosis occurs. Probably the leucocytosis is less than that found with a non-diphtheritic sore throat of equal severity. After injection of antitoxin there is usually a prompt reduction of the leucocytosis, and in favourable cases show a steady, gradual diminution. Myelocytes are not infrequently observed.

**Whooping-cough** gives rise to a marked leucocytosis, the lymphocytes numbering over 50 per cent. It begins in the catarrhal stage, and disappears slowly during convalescence.

**Croupous Pneumonia.**—An intense polynuclear leucocytosis almost invariably occurs. It fails to appear in very mild and in very grave cases, in both instances owing to defective reaction. In the mild cases an active resistance is unnecessary; in the grave cases the intensity of the infection may overwhelm the subject, or his powers of resistance may be abolished by previous ill-health, by alcoholic excess, or by hereditary debility. A decreased leucocyte count (**leucopenia**) accompanying local evidence of a severe attack enhances the gravity of the prognosis in pneumonia. On the other hand, a marked leucocytosis under the same circum-



stances indicates a sthenic type of affection, with active resistance. With the crisis, or perhaps a day before it, a sudden drop in the leucocyte count usually occurs. The eosinophiles disappear during the course of the disease, and their reappearance coincides with other evidence of a favourable outlook.

**Acute Rheumatism.**—In slight cases, without exudation, there may be no increase in the white cells; in more severe cases a moderate leucocytosis is to be observed. When the leucocytes number 20,000 or more, suspect pneumonia, pericarditis, or other complication.

**Septicæmia, Pyæmia, Infective Endocarditis, Osteomyelitis.**—Polymorphonuclear leucocytosis is the rule, and varies in degree directly with the severity of the attack. Those cases of severe type without leucocytosis are probably analogous to the similar condition in pneumonia.

**Erysipelas.**—A moderate or considerable leucocytosis, chiefly polynuclear, is the rule. The eosinophiles decrease at the height of the affection, to return as the condition improves. A high leucocyte count frequently indicates suppuration in the course of the disease.

**Syphilis.**—Leucocytosis to a moderate extent (under 20,000) is usual in the secondary stage, and is mainly lymphocytic, coincident with the enlargement of the lymphatic glands. The eosinophiles may also be increased, especially when the papular eruption is well marked. In the tertiary stage, with gummatous lesions, there is also leucocytosis, not, however, so markedly lymphatic in character. The primary stage does not usually show leucocytosis.

**Gonorrhœa.**—An acute attack is accompanied by a moderate polynuclear leucocytosis, which becomes more intense with the supervention of epididymitis, cystitis, or other complications. Eosinophiles may be abundant in the discharge, but are not usually in great excess in the blood.

**Epidemic cerebro-spinal meningitis** is always accompanied by a marked polynuclear leucocytosis. This may serve in the diagnosis of this affection from tuberculous meningitis, and from typhoid fever (see below).

A considerable leucocytosis is usually to be found in **cholera**, **dysentery** (ulcerative), **glanders**, **actinomycosis**, **bubonic plague**.

The affections enumerated above are all characterized by a greater or less degree of leucocytosis, unlike the remaining

members of this group, which, as a rule, only present an increase of white cells when complications arise.

**Typhoid Fever.**—The white cells during the first week of the disease are usually normal or somewhat increased in number in uncomplicated cases, though this period is unreliable in this respect. From the second week onward there is a progressive diminution in their number, and especially in the polynuclear cells. During convalescence the leucocytes gradually increase, and there may even be leucocytosis in the late periods without obvious complications to account for it. The lymphocytes do not diminish in the same ratio, but soon show a relative increase, which may last for a couple of months, and in children even longer. Eosinophile cells are diminished while the fever lasts, but reappear in normal or increased numbers during convalescence.

The decrease in the leucocytes is due to the influence exerted by the typhoid toxin upon the blood-forming tissues: the greater that influence—that is to say, the more intense the intoxication—the lower is the leucocyte count.

It is frequently found that leucocytosis occurs during the course of typhoid fever. This is usually due to the advent of some complication. Bronchitis—especially if it involves the smaller bronchi—pneumonia, abscesses and boils, hæmorrhage, severe diarrhœa, perforation of the bowel, and other affections, may be the cause. In some cases of perforation, or other severe complication, the leucocyte count may not rise, for the same reason as given above concerning pneumonia (p. 180).

For diagnostic purposes the absence of leucocytosis in uncomplicated typhoid fever may at times be of service. Pneumonia, septicæmia, suppurative appendicitis, and other forms of active purulent inflammation, can usually be at once distinguished from typhoid by the white blood count. It is a different matter with tuberculous affections, for which typhoid fever is frequently mistaken: the behaviour of the leucocytes is somewhat similar in both conditions, and is of no assistance in diagnosis.

The blood state in a relapse is similar in all respects to that of the primary affection.

The following points in prognosis are stated by Nægeli: The persistence of the eosinophiles during the height of the fever, or their reappearance during the stages of the fastigium or of remission, and the early and distinct increase in lymphocytes, indicate

a favourable course. A considerable diminution of all the leucocytes, or an absence of leucocytosis when complications have arisen, points to an unfavourable termination.

**Tuberculosis**, when uncomplicated, does not give rise to any increase in the number of leucocytes which may, on the contrary, be diminished. In pulmonary tuberculosis leucocytosis is usually caused by recently-formed suppurating cavities, by pneumonia, by cachexia, and by hæmorrhage. The consolidation of the lung found in acute phthisis does not raise the leucocyte count, and may thus be distinguished from true lobar pneumonia. In acute generalized miliary tuberculosis leucocytosis is equally wanting. The serous membranes (meninges, pleuræ, peritoneum, joints, etc.) may be the seat of tuberculous inflammation without increase being observed in the leucocytes. In a certain proportion of cases leucocytosis occurs, the result, in a majority of the instances, of a mixed infection whereby other pyogenic organisms have been introduced. With regard to tuberculous disease in bones, glands, skin, and other tissues and organs, the same deficiency of the leucocytes is observed where the infection is purely tubercular. In some chronic cases, especially those with enlarged lymphatic glands, there is a definite lymphocytosis. As a rule, in tuberculous diseases the lymphocytes are found in a larger proportion than in normal blood, but the leucocytosis due to complications is in this class of affection, as in simple inflammatory conditions, of the polynuclear type. As stated above in referring to cerebro-spinal meningitis, the absence of leucocytosis in a case of meningitis is a strong point in favour of the tuberculous form of the disease.

In **measles** and in **malaria** the white cells are diminished in number (leucopenia).

There is no material change in the leucocytes in **typhus fever**, **influenza**, **German measles**, **mumps** (thus distinguishing the orchitis or mumps from that of gonorrhœa), **yellow fever**, **Malta fever**, and **leprosy**.

**2. Local Infectious Inflammation ; Suppuration — Appendicitis.**—In the catarrhal or simple exudative form there is usually no leucocytosis, or only a very slight increase in the white cells ; if there is local peritonitis, a moderate leucocytosis ensues ; if abscess, gangrene, or general peritonitis occurs, the leucocytosis is considerable, except in those overwhelming and asthenic forms (as referred to above under Pneumonia), where the

reaction is wanting. By this observation we may exclude, as a rule, typhoid fever, fæcal impaction, and intestinal colic.

**Abscess.**—Leucocytosis almost always occurs in acute circumscribed suppuration. Old-standing collections of pus may not cause any increase in the leucocytes.

**3. Inflammations of Serous Membranes (Non-tuberculous).**—During the exudative stage of serous and of purulent inflammations of the large serous surfaces (pleura, peritoneum, pericardium, meninges), the leucocyte count is usually increased. The purulent process is marked by a more intense leucocytosis than the serous, but in both forms the leucocyte count falls when the active exudation is at an end.

The blood examination may therefore be of service in distinguishing between tuberculous and non-tuberculous pleurisy, meningitis, or peritonitis, if the examination be made while the exudation is actively proceeding.

**4. Catarrhal Inflammations of Mucous Membranes** are not, as a rule, characterized by much increase in the white cells. Catarrh of the gastro-intestinal tract may not cause leucocytosis, but it is found to a moderate extent in catarrh of the bronchi (especially of the smaller tubes) and of the genito-urinary tract.

**5. Malignant Disease.**—Cancerous tumours produce but little effect upon the leucocytes; any increase in their number is probably due to accompanying inflammatory changes in or around the tumour. Sarcoma is more likely than cancer to be accompanied by leucocytosis in the absence of ulceration or other inflammatory complication.

**6. Diseases of the Blood and Blood-forming Organs.**—In **chlorosis** the leucocytes are not increased, and the same may be said of **pernicious anæmia**; a relative increase of the lymphocytes is commonly observed. Leucocytosis in these affections indicates the presence of complications.

It is in **leukæmia** that the greatest increase in white cells is observed. The condition is not, however, one of leucocytosis in the sense of a chemotactic effort on the part of the blood and tissues, whereby the polynuclear neutrophile cells in particular would be the chief elements present. While these cells are much more numerous than in health, they form a smaller proportion of the total mass of white cells than is found normally. The condition is further considered in the article on Blood Examination (p. 67). It will suffice to state here that the diagnosis will

mainly rest, in one form of the disease (myelæmia), on the presence of an abundance of myelocytes, and in the other form (lymphæmia) on a great increase of the lymphocytes.

In **Hodgkin's disease** and in **splenic anæmia** the leucocytes are in normal or diminished proportions, with a tendency toward lymphocytosis.

**Anæmia infantum pseudo-leukæmia** may be mentioned, as a considerable mixed leucocytosis with a large percentage of lymphocytes is always present.

**Purpura** and **scurvy** are usually accompanied by a polynuclear leucocytosis.

7. A great variety of observations upon the characters of the white cells have been made in many diseased conditions. In most instances not already referred to the diagnostic value is not great, and only a few general results will now be mentioned.

In **liver** affections the condition varies. **Catarrhal jaundice** usually shows a slight increase of the leucocytes. **Gall-stones** may be present, and may give rise to severe attacks of biliary colic without producing leucocytosis. When jaundice supervenes or signs of inflammatory changes appear, there may be a moderate leucocytosis. **Alcoholic cirrhosis** does not increase the white cells unless hæmorrhage or jaundice occur, but the **hypertrophic biliary cirrhosis** of Hanot shows a moderate leucocytosis. Abscess of the liver usually causes leucocytosis, especially when it takes the form of multiple small abscesses due to cholangitis.

**Diseases of the kidneys** do not cause any material departure from the normal condition of the leucocytes. In some cases, especially when œdema is present, an increased proportion of lymphocytes may be observed.

**Gout** commonly gives rise to a mixed leucocytosis.

In **rickets** there is usually no increase in the white cells unless complications are present.

**Tonsillitis**, if phlegmonous, may cause a considerable polynuclear leucocytosis; the follicular form produces a similar but less extensive increase in the leucocytes.

**Intestinal parasites** may cause a slight polynuclear leucocytosis. The eosinophile cells are usually in considerable excess.

Many **skin diseases** present a well-marked eosinophilia, and at times a mixed leucocytosis.

No material change in the leucocytes is found in **nervous diseases**.

**Summary.**—Increase of polynuclear leucocytes is the result of chemotaxis.

Increase of lymphocytes is due to the mechanical washing out of the cells from the lymphatic glands and other lymphoid tissues.

Increase of eosinophiles is probably the result of eosinophile chemotaxis.

Increase of polynuclear leucocytes is found in the following conditions: Pneumonia, diphtheria, septic infection, erysipelas, scarlet fever, acute rheumatism, cerebro-spinal meningitis, non-tuberculous pleurisy and peritonitis, abscess, tonsillitis, leukæmia, von Jaksch's anæmia, scurvy, purpura, catarrhal jaundice, hypertrophic biliary cirrhosis of the liver, gout.

Increase of more than one form of white cell may be found in many of the above-named conditions, also in the following: during digestion and absorption, during pregnancy, in cachectic states, after hæmorrhage, as a result of certain drugs, and in the ante-mortem state.

Increase of lymphocytes almost exclusively is seen in syphilis, variola, varicella, vaccinia, whooping-cough, sometimes in diphtheria, in hydræmic conditions, and in new-born infants.

The leucocytes are either in normal numbers or decreased in the following affections: tuberculosis, typhoid fever (here the polynuclears are decreased, while the lymphocytes may be increased), typhus fever, influenza, measles, rubella, mumps, pernicious anæmia, chlorosis, splenic anæmia, Hodgkin's disease, alcoholic cirrhosis of the liver, renal affections, rickets. In catarrh of the mucous membranes there may be no leucocytosis, but it is often seen in bronchitis, especially in the capillary form. In malignant disease the leucocytes are normal unless inflammation or cachexia be present.

Increase of eosinophile leucocytes (eosinophilia) occurs in skin affections, syphilitic and otherwise, asthma, leukæmia, intestinal parasites (*ankylostoma*, *Tania mediocanellata*, *Tania echinococcus*, *oxyuris*, *ascaris*, *trichina spiralis*); post-febrile eosinophilia, observed during convalescence from most fevers; in scarlet fever, and sometimes in acute rheumatism, contrary to the rule that eosinophiles decrease during fevers; chronic tumours of the spleen, and after removal of the organ. Eosinophiles are more numerous in children's than in adults' blood.

Increase of mast cells is found in myelogenous leukæmia, in

tuberculous, syphilitic, and other skin affections. As a rule, they are increased when the eosinophiles are more numerous than normal.

Myelocytes (mononuclear neutrophile leucocytes and mononuclear eosinophile leucocytes), occur in spleno-medullary leucæmia; conditions causing polynuclear leucocytosis, diphtheria in particular; in severe primary or secondary anæmias (due to hyperplasia and hyperæmia of the bone-marrow).

### LEUCOPENIA (Gr. λευκός, white; πένης, poor).

The number of white corpuscles in a given volume of blood varies in different conditions of health as well as in disease, the average count being about 8,000 per cubic millimetre. One finds in certain conditions a considerable excess over this number (see Leucocytosis, p. 176), while in others the numbers may be much lower. The latter condition is known as leucopenia.

The number of leucocytes is increased in many varieties of anæmia, but is diminished in splenic anæmia. In typhoid fever the leucocytes are normal or decreased in number, unless inflammatory complications ensue. Leucopenia is the rule in measles, especially during the period of eruption. Generally speaking, a diminution in the number of white corpuscles is of less assistance in diagnosis than the contrary condition of leucocytosis.

### LIENTERY (Gr. λείος, smooth; έντερον, the intestine).

The presence of undigested food in the fæces. The name of the condition is derived from the idea that the food has passed unaltered through an apparently smooth and functionless tube. (See Fæces, p. 143.)

### LIGHTNING PAINS.

Among the early symptoms of locomotor ataxia are pains in the legs, which are often moderate in severity, but usually sudden, sharp, and stabbing in character. They are known as lightning pains, and may be experienced in the lower part or upper regions of the limb, or in all its length, or may involve the trunk.

**LINEÆ ALBICANTES (Striæ Gravidarum).**

Whitish streaks may be seen on the surface in situations where the skin has been unduly stretched. When recent they have a reddish colour. They are best developed in the abdominal wall in cases of pregnancy and of tumours, but are also seen in the thighs, breasts, armpits, etc., when a considerable deposition of fat has formed, and especially if the accumulation has been fairly rapid.

**LITTEN'S SIGN (the Diaphragm Phenomenon).**

A movement of the intercostal spaces in the infra-axillary regions, caused by the downward movement of the diaphragm during inspiration. The interspaces are to a certain extent drawn inwards, and by placing the patient in a suitable position in relation to the light the moving spaces can be seen as a shadowy groove across the infra-axillary space passing downward with inspiration, and to a less extent upward with expiration (see p. 471).

**LIVER DULNESS, Loss of.**

The normal area of liver dulness, extending in the right nipple line from the sixth rib to the costal margin, and behind from the tenth to the twelfth rib, may, under certain circumstances, be abolished. This occurs most characteristically when free gas escapes into the peritoneal cavity, when the liver dulness is only found in the most dependent parts. It may also be found in tympanites, in emphysema, and in pneumothorax (see pp. 16, 55).

**LIVER, Enlargement of.**

It may be taken as a rule, to which the exceptions are few, that the liver is not enlarged in an upward direction, but, on the contrary, enlargement of the organ is shown by the appearance of its lower border at a lower level than usual. The normal liver reaches the costal margin in the right nipple line, and about a hand's breadth below the ensiform cartilage in the middle line. Its upper level of absolute dulness is a line joining the following points: the intersections of the right scapular line with the tenth rib, of the right mid-axillary line with the eighth rib, of the right



nipple line with the sixth rib, of the right side sternal line with the sixth costal cartilage. To the left of the sternum the hepatic dulness is indistinguishable from that of the heart. These limits vary somewhat with respiration, and are further discussed in the article on the Percussion of the Thorax, at p. 451 *et seq.*

The chief conditions in which enlargement of the liver occurs are enumerated and compared in the table on p. 190.

### LIVER, Pulsating.

The liver may be seen at times to pulsate, and under conditions very similar to those which give rise to venous pulsation (see p. 312). In order to demonstrate the pulsation careful palpation will suffice in the better-marked instances, but some of the less obvious cases may best be detected by means of recording tambours actuated by a cup-shaped receiver placed over the liver. Care must be taken not to mistake the pulsation of the heart transmitted through the liver for those of the liver itself. By sinking the edge of the hand well under the edge of the liver it is possible in some cases to feel the expansion of the organ; or with one hand pressing the liver forward from behind and the other laid over the hypochondrium and epigastrium, one may be able to make certain of the pulsation.

The pulse felt is a wave transmitted from the right heart backward along the inferior vena cava to its branches. It is rare that such a wave can be perceptible by the agency of the right auricle alone (as may occur in the jugular pulse). When the graphic record shows that a venous pulse is synchronous with the auricular systole, it is stated by Mackenzie to be strong evidence of the presence of tricuspid stenosis. A ventricular systolic liver pulse indicates dilatation of the right heart with tricuspid incompetence. The wave is initiated by the contraction of the right ventricle, passes through the patent tricuspid orifice, and without being materially impeded by the auricle, which has presumably lost most of its tone and contractility, it traverses the venæ cavæ and their branches. Venous pulsation in the liver is, then, an indication that the right heart is in an advanced stage of dilatation and engorgement, which is consequent in most cases on disease of the mitral orifice. On rare occasions it may be the result of tricuspid stenosis, with hypertrophied and dilated right auricle.

A ventricular systolic pulse may in rare instances be produced

## ENLARGEMENTS OF THE LIVER

Disease.	Jaundice.	Pain.	Causation.	Other Distinguishing Symptoms.
<b>Hyperæmia (Passive Congestion)</b>	Usually slight or absent	Painful to pressure	Heart affections	Edema, venous pulsation, dyspnoea
<b>Cholangitis, Catarrhal and Infective</b>	Almost always present	Often pain and tenderness	Digestive disorders; gall-stones	Sometimes pyrexia, rigors, sweating
<b>Obstruction of Common Bile-duct</b>	Always present	Often absent	Gall-stones, cicatrices, enlarged glands, tumours, pancreatitis	Often pyrexia and rigors. Other signs of the primary affection
<b>New Growths</b>	Present in 50 per cent. of cases. Less often in the rare primary cancer	Painful as a rule	Secondary to disease elsewhere in most cases	Signs of the primary disease
<b>Cirrhosis of Liver</b>	Usually slight; often absent	Liver often tender	Alcoholism in most cases	Ascites, enlarged spleen, hæmatemesis, piles
<b>Amyloid Disease of Liver</b>	Absent	Absent	Prolonged suppuration, syphilis	Amyloid disease elsewhere
<b>Fatty Liver ..</b>	Absent	Absent	Alcoholism, phthisis, diabetes, phosphorus poisoning, obesity	Signs of the primary disease
<b>Rickets</b>	Usually absent	Absent	Unsuitable diet	Signs of rickets
<b>Syphilis (Acquired)</b>	Usually absent	Often present	Contagion	Irregular and nodular liver. Signs of syphilis elsewhere
<b>Syphilis (Hereditary)</b>	Usually absent	Often present	Hereditry	Enlarged spleen, occurs in childhood
<b>Abscess of Liver</b>	Slight or absent	Usually present	Dysentery, pyæmia, sepsis	Liver may be enlarged upwards, pyrexia, sweating
<b>Hydatids</b>	Absent	Absent	<i>Tania echinococcus</i>	Often in left lobe of liver. Thrill
<b>Lacing Liver</b>	Absent	Sometimes present	Tight-lacing	Dyspepsia, Irregular enlargement of liver
<b>Weil's Disease</b>	Present	Present	Bacterial infection	Head symptoms; acute nephritis. Disease very rare

by active arterial pulsation in cases of aortic incompetence and in inflammation of the liver. This pulsation is of extremely infrequent occurrence, and forms an exception to the statement made above, that the pulsating liver is an evidence of a distended right heart and incompetent tricuspid valve.

**LORDOSIS** (Gr. *λορδός*, bent back).

An exaggeration of the natural curving forward of the lumbar spine. It is a result of the need to throw the centre of gravity backward in cases of large abdominal tumour or of pregnancy, the shoulders falling back behind the level of the buttocks.

The curve is well marked in cases of pseudo-hypertrophic muscular paralysis, and may be seen in progressive muscular atrophy.

Flexion of the hip-joint, which is persistent (congenital or unreduced dislocation, *morbis coxæ*, etc.), is a common cause in surgical conditions.

**LUMBAR PUNCTURE.**

The withdrawal and examination of fluid from the spinal canal has been shown by Quincke to be a useful and instructive diagnostic method.

The patient is placed on his side, the body bent forward, with his back to the operator. The needle of an exploring syringe is inserted, with aseptic precautions, into the spinal canal, immediately below the spine of the third, fourth, or fifth lumbar vertebra, and about  $\frac{1}{4}$  inch to one side of the median line, so as to avoid the dense interspinous ligament. In children the canal is reached at a depth of about 1 inch, and in adults at 2 to 3 inches from the surface. A specimen of the fluid is then withdrawn.

If the fluid is at normal pressure in the canal, it will exude from the cannula drop by drop. If the pressure is raised it will escape more rapidly, and when pressure is extreme it will emerge in a stream.

The operation is harmless as a rule, but care should be taken not to withdraw a very large quantity of fluid, as danger has been known to result from lowering too much the intraspinal pressure.

Normal spinal fluid is a clear, watery liquid with a density of 1003 to 1006, and very little albumin. An increase of the albumin and a raised specific gravity are found in meningitis. A cloudy

fluid, and the presence of leucocytes also indicate inflammation. In tuberculous meningitis, however, and occasionally in other forms of inflammation, the fluid may be quite clear.

Stained films of the centrifugalized sediment may detect micro-organisms. The *Diplococcus intracellularis* of Weichselbaum is seen in cases of epidemic cerebro-spinal meningitis; staphylococci, pneumococci, tubercle bacilli, and other micro-organisms may be found.

### LUMBAR REGIONS.

On each side of the body the lumbar region is bounded internally by a vertical line passing through the mid-point of Poupart's ligament; above by a horizontal line joining the lowest points of the two tenth ribs; below by the crest of the ilium and by a horizontal line joining the two anterior superior iliac spines. The region extends back to the vertebral column.

The various abnormalities to be found in this region are enumerated in the articles on Abdominal Shape, Percussion, Pain, etc., at pp. 6, 16, and 273.

### LYMPHATIC GLANDS, Enlargement of.

It may be assumed that lymphatic glands which can be felt are abnormal. Their enlargement is caused by the presence in the lymph which passes through them of some toxic substance. A variety of diseased conditions have for their chief symptom enlargement of these glands.

Painful, inflamed glands, which tend to suppurate and are somewhat fused together in advanced cases by surrounding inflammation, are usually tubercular in origin. Their commonest seat is the neck, and the patients are children or youths.

A more acute suppurating type of gland, occurring in the groin, in a transverse chain close to Poupart's ligament, is due to infection from the soft or multiple chancre.

Painful, inflamed glands, which tend to resolve without suppuration, but may in some instances break down and suppurate, are the result of septic infection in the region drained by the lymphatics which pass through the affected glands.

Inflammation and frequently suppuration of groups of glands, most commonly the inguinal, constitutes one of the chief symptoms of bubonic plague.

Enlarged glands, which are very slightly or not at all painful, do not suppurate, remain fairly distinct and hard ('bullet bubo'), are chiefly observed in the transverse inguinal group, and are commonly found in young adults, are probably syphilitic.

Chronic enlargement of one or more groups of glands, which do not tend to suppurate, but in time grow enormously and may ulcerate or necrose, while the patient becomes progressively weaker, and the blood is found to be of the secondary anæmia type (see Anæmia p. 30), is probably due to a cancerous growth in the region involved.

A general enlargement of the groups of lymphatic glands throughout the body, without evidence of inflammatory changes, the patient's blood showing a marked lymphocytosis, indicates lymphatic leukæmia.

The glands of the body generally are enlarged, but those of the upper parts of the body most markedly, and the disease often first shows itself in the neck. They have little tendency to suppurate, and for a considerable time the glands remain separate and distinct, though considerably enlarged. Eventually they may be more or less coalesced, and may become necrotic or may suppurate. The blood is anæmic, of the secondary type, with in many cases an increased number of white cells, which, however, are chiefly polymorphonuclears. The condition is Hodgkin's disease, or pseudo-leukæmia.

An enlargement of groups of glands (often the mediastinal or bronchial glands to begin with), progressing rapidly to the formation of masses consisting of the glands and surrounding tissues infiltrated with an active new growth. The condition is probably lympho-sarcoma.

### LYSIS.

The gradual defervescence which so commonly forms the termination of fevers. It is observed in rheumatism, typhoid fever, lobular pneumonia, scarlet fever, pleurisy, septic infections, and many other affections characterized by raised temperature. The occurrence of lysis where usually crisis is the form of termination often indicates the supervention of some complication. (See Temperature, p. 405.)

**MACULAR ERUPTIONS.** See *Skin Eruptions*, p. 365.

**MAIN-EN-GRIFFE.** See *Contractures*, p. 105.

**MAMMILLARY LINE (Nipple Line).**

A vertical line drawn on each side of the front of the chest through the nipple, or through the middle of the clavicle. It is used in referring to the topography of the chest. (See *Thorax*, *Shape*, etc., p. 464.)

**MAMMARY REGIONS.**

That portion of the front of the chest lying between the levels of the third costal cartilage and of the sixth rib in the nipple line on each side. The abnormalities to be found are referred to in the articles on the *Shape*, etc., of the *Thorax*, p. 464; *Percussion*, p. 450; *Auscultation*, p. 407; and *Pain*, p. 270.

**MEAT-JUICE DIARRHŒA.**

The fecal discharge in cases of dysentery may be sero-purulent, or may be serum mixed with blood and scraps of fibrinous or mucoid material. This condition of feces, when the motions are frequently repeated, as is generally the case, is sometimes known as 'meat-juice diarrhœa.'

**MELÆNA** (Gr. μέλαινα, the black disease).

The appearance of black blood in the stools is known as 'melæna.' Its causes and nature are considered in the article on the *Fæces* (p. 140).

**METALLIC TINKLING.**

A faint, clear, musical note may be heard at intervals on listening over a hydropneumothorax, resembling the sound of a drop of water falling into a reverberating vessel. A somewhat similar sound is to be heard in pulmonary cavities, by the addition of the amphoric quality to the coarse râles.

**METAMORPHOSED BREATH-SOUNDS.**

During the act of respiration changes in the character of the sound may be heard. Its pitch may alter, or bronchial breathing may acquire a cavernous quality, or sudden alterations in the intensity of the sound may be noted (see p. 413).

**METEORISM** (Gr. *μετέωρος*, raised up).

A distension of the abdomen by gas (see p. 7).

**MICTURITION, Disorders of.**

Nature of the bladder reflex—Physiology—Causes of disturbance of the bladder's functions—Incontinence of urine—active, passive, and false or paradoxical incontinence—Retention of urine—Suppression of urine.

The act of emptying the urinary bladder, while to some extent a voluntary one, depends for its efficient performance upon the integrity of certain reflex mechanisms. Disturbances of the function furnish us with valuable information as to the condition of the nervous system. The physiology and pathology of reflex action generally are briefly considered at p. 334, and the present article deals chiefly with the disturbances of the bladder reflex which are of service in medical diagnosis.

Recent investigations have furnished evidence that subsidiary centres for the control of the bladder and other pelvic viscera are to be sought for in the sympathetic nerves and ganglia. The views on this subject hitherto held by physiologists, and not yet disproved, are as follows: The **bladder reflex** is really a combination of several reflexes. The peripheral portions of their reflex arcs are formed by the first three lumbar, and second, third, and fourth sacral nerves, together with the sympathetic nerves and plexuses in connection with the viscus. Their central portions—that is, the 'nuclear reflex arcs' (see Reflexes, p. 335)—are situated in that region of the cord from which the above-named nerves spring—viz., the lumbar enlargement. It is assumed that this portion of the cord contains a 'centre' which regulates and co-ordinates the mutually dependent reflexes which effectuate urination. In addition to the lumbar portion of the reflex arcs, which corresponds to the shortest pathway between afferent and efferent nerves, other less direct pathways traversing the higher regions of the cord and the brain are presumed to exist, whereby the cerebral influences mentioned below reach the lumbar centre.

Accepting these anatomical arrangements, the reflexes governing the bladder's function may now be described, and the diagram on p. 196 may be consulted. As soon as sufficient urine has collected in the bladder to give rise to a certain distension, or when

a few drops of urine have escaped from the bladder into the urethra, a stimulus is given to sensory end-organs in the mucous membrane of the bladder and urethra.

The impression thus received is carried by the afferent limb of the reflex arc to the central portion of the reflex arc, in the lowest part of the cord. The impulse is there transformed into a motor

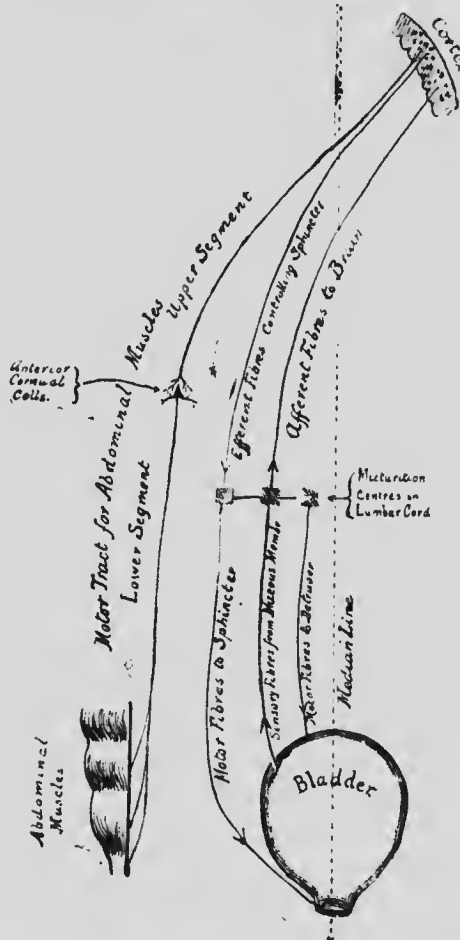


FIG. 27.—DIAGRAMMATIC REPRESENTATION OF THE BLADDER REFLEXES.

impulse directed to the sphincter vesicæ, which is thereby maintained in a state of tonic contraction. When a certain degree of distension has been reached, the urgent impressions received by the sensory nerves are transferred in the cord to a different efferent path. Instead of being directed to the sphincter they



pass to the detrusor, and at the same time an inhibitory stimulus from the brain is directed to the sphincter, which relaxes in consequence, while the detrusor, assisted by the abdominal muscles, expels the urine, of which the last portion to escape is aided in its exit by the perineal muscles. In some cases where the cerebral influence is abolished by lesion above the lumbar enlargement, the direction and co-ordination of the distinct and separate reflexes involved is accomplished by means of the controlling 'centre' in the lumbar cord. This automatic micturition in cases of transverse lesion of the cord is rare in man, but occurs more frequently in the lower animals.

The reflexes, then, normally engaged in the bladder's function are—(a) continuous or tonic contraction of the sphincter, preventing the inconvenient escape of urine; (b) intermittent contraction of the longitudinal muscular fibres of the bladder, the detrusor vesicæ, whereby the contents are expelled; (c) contraction of the ejaculatoriæ and other perineal muscles (to a great extent an involuntary action), completing the evacuation of the bladder; (d) in the case of undue distension of the bladder, with sensation of urgent need for urination, muscles which, under ordinary circumstances, are voluntarily brought into action may now uncontrollably take part in the act of emptying the bladder—viz., abdominal muscles, diaphragm, and adductors of the vocal cords. In addition, another factor is necessary—(e) inhibition of the contractions of the sphincter. This is commonly a voluntary act, but may be a reflex one. Normally inhibition of the sphincter is the chief share taken by volition in micturition, the detrusor being beyond our control, and the voluntary muscles mentioned above not being absolutely necessary to the act. Inhibition, moreover, in man is rarely performed independently of will, with the result that in lesions interrupting the course of impulses between the brain and lumbar enlargement of the cord the sphincter usually remains tightly closed until excessive fulness of the bladder overcomes the muscle's resistance. The same result is produced by affections of the brain which interfere with cerebration, such as extensive cerebral hæmorrhage, intracranial pressure, toxic conditions, such as typhoid fever, typhus, etc. As the bladder possesses a bilateral innervation, retention of urine of cerebral origin only occurs when both hemispheres are inactive. The consequence of the inability to inhibit the sphincter's action is a constant overflow or dribbling from the distended

bladder, the so-called **paradoxical** or **false incontinence**, which has to be distinguished from **true incontinence** due to imperfect closure of the sphincter, as seen in interruption of the reflex arc.

The contrary condition to that last mentioned is an excessive readiness of the bladder reflex to act, and is often caused by an innate and inherited sensitiveness of the nervous system generally, and of this reflex in particular, or by peripheral irritation, weakness of the sphincter, etc.

Disorders of the bladder reflex are revealed by disturbances of the bladder's functions—that is, by abnormalities of micturition—of which the following clinical varieties may be recognized:

1. **Incontinence of Urine, or Enuresis**, is an involuntary escape of the bladder contents, either as a constant dribbling or as an intermittent evacuation. Three forms of incontinence may be distinguished:

(a) **Active Incontinence** is the condition mentioned in the previous paragraph, in which an undue excitability of the nervous 'installation' of the bladder mechanism is present. The reflex is not only intact, but actually overdoes its part, completing the act of micturition in obedience to peripheral stimulation; not waiting for the cerebral order to inhibit the sphincter, but transferring that inhibiting impulse directly from the periphery, through the 'spinal reflex arc,' to the efferent path to the sphincter. In addition, it may be that the sphincter in these cases is less strong and resistant than usual, and is relaxed more easily than normal. We find this condition in children, and especially in boys. The peripheral irritation is supplied by abnormal qualities of the urine, such as excessive acidity, high specific gravity, the presence of uric acid or other crystals; phimosis; rectal irritation from worms, polypus, or prolapse of the anus. In both children and adults a similar involuntary micturition occurs in irritation of the bladder from actual disease of that organ, as seen in calculus, ulcer of the bladder, and other surgical affections of the organ. This form of incontinence may be seen in those rare cases of transverse lesion of the cord in which the intermittent discharge of urine occurs in response to peripheral impulses set up by the accumulating urine in the absence of cerebral direction towards inhibition of the sphincter. (The usual consequence of this lesion is retention of urine; see below.) A variety of active incontinence may be seen in emotional disturbance, as in hysteria and terror.

(b) **Passive Incontinence** denotes the involuntary passing of urine owing to a defect in the reflexes (a) and (b) (p. 196)—that is, defective contraction of the sphincter and of the detrusor of the bladder. The consequence of an interruption at any part of the sphincter reflex is paralysis of the sphincter, and is revealed by the constant dribbling of urine from an **almost empty** bladder. Lesions of the lumbar enlargement of the cord will interrupt this reflex arc (as well as that concerned in the action of the detrusor), and injury to the sphincter muscle itself, from previous overdistension, may also interfere with the proper performance of the reflex. The detrusor muscle, the contracting factor in the reflex (b), may also be injured by distension of the bladder, so causing failure of this reflex, and both muscles may suffer from simple weakness, or **atony**, as a result of exhausting disease affecting any part of the body. This in many cases, no doubt, accounts for the retention of urine in typhoid fever, etc., though imperfect cerebral inhibition of the sphincter can also in these cases be at fault (p. 197). **Atony of the bladder**, as a result of interruption of either central or peripheral factors in (b) the detrusor reflex is shown by constant dribbling from a **partially full** bladder. Overdistension, causing atony of the detrusor and, to a less degree, of the sphincter, may be the result of failure to inhibit the sphincter's action, as seen in transverse lesions of the cord above the lumbar enlargement, or in bilateral cerebral disease. It is, however, more commonly the result of obstruction to the outflow of urine caused by some surgical affection of the prostate or urethra. It may be due to spasm of the sphincter, following injury to the urinary or generative organs; also from prolonged voluntary retention of urine.

(c) **False, or Paradoxical Incontinence** is the constant dribbling of urine from an **overflow** bladder. This is seen in the condition referred to at p. 197, under (e), and subsequently, and is produced by transverse lesions above the lumbar cord, and by bilateral cerebral affections. It is frequently found in surgical obstructions to the complete emptying of the bladder, and should be always borne in mind when dealing with incontinence of urine.

2. **Retention of Urine** is the inability from any cause to expel the contents of the bladder. It is the earlier stage of the condition mentioned in the last paragraph, and is produced by the same causes. The symptoms are absence of micturition, with an increasingly distended bladder, and it must be carefully dis-

tinguished from **suppression of urine**, in which the bladder remains empty. The latter condition is an affection of the kidneys, and is discussed at p. 400.

**Summary.**—The complex bladder reflex consists of the following acts and conditions: (a) Tonic contraction of the sphincter vesicæ; (b) intermittent contraction of the detrusor vesicæ. These phenomena are entirely automatic. The first may be voluntarily abolished by (c) inhibition of the sphincter, which in disease may be relaxed involuntarily or by pressure; (d) other muscles, which are often used voluntarily, may in micturition take part in an involuntary reflex act—viz., the perineal muscles, the abdominal muscles, the diaphragm.

Disorders of bladder reflex give rise to:

1. **Incontinence of Urine.**—(a) **Active Incontinence**, seen in cases of peripheral irritation, such as altered and irritable condition of the urine; irritation of the rectum (worms, polypus, etc.); phimosis, usually found in children, especially males. Emotional disturbances sometimes are a cause (terror, hysteria), and rarely transverse lesions of the cord above the level of the lumbar enlargement. (b) **Passive Incontinence**, from paralysis of the sphincter, is recognized by constant dribbling from an almost empty bladder. Due to lesions of the lumbar cord, injuries to the sphincter or its innervation from previous overdistension, atony from exhausting diseases, in which the detrusor shares, as shown by the dribbling from a partially filled bladder. (c) **False or Paradoxical Incontinence**. The constant dribbling from an overfull bladder, seen in coma; intracranial hæmorrhage; transverse lesions of the cord above the lumbar enlargement; toxæmic conditions, such as typhoid and typhus fevers; mechanical obstruction to the outflow from the bladder, etc.

2. **Retention of Urine** is produced by the affections just named as causing false incontinence, of which it is the earlier stage.

Incontinence of urine occurs either as a constant dribbling or as a single or repeated involuntary escape of water.

Dribbling from an **empty** bladder indicates paralysis of the sphincter, with intact detrusor. Seen chiefly in lesions of the lumbar portion of the cord.

Dribbling from a **partially full** bladder denotes paralysis of both sphincter and detrusor vesicæ. Seen in injury to lumbar cord and atony of bladder from previous distension.

Dribbling from a **full** bladder points to obstruction to the out-

flow from the bladder, either from spasm of the sphincter or from mechanical impediments to the act of micturition. It therefore indicates a transverse lesion of the cord above the lumbar enlargement, intracranial lesions which abolish the power of inhibiting the sphincter's action, toxic conditions, and surgical affections of the bladder or urethra.

Micturition **during** a convulsion suggests epilepsy; but evacuation of the bladder **after** a convulsion is more characteristic of hysteria.

Involuntary intermittent urination may on rare occasions be the result of a transverse lesion above the lumbar enlargement, but is more likely to be due to a hypersensitive condition of the reflexes combined with peripheral or central irritation.

#### **MID-AXILLARY LINE.**

A vertical line drawn on the side of the chest, midway between the anterior and the posterior axillary lines. It is used in the topography of the thorax (see p. 464).

#### **MID-STERNAL LINE.**

A line drawn vertically down the middle of the sternum; one of the artificial landmarks in the topography of the chest. (See Thorax, Shape, etc, p. 464.)

#### **MITRAL AREA.**

That portion of the chest-wall in the immediate neighbourhood of the apex-beat is known as the mitral area, for it is at this region that sounds generated at the mitral orifice are, as a rule, best heard (see p. 407).

#### **MOEBIUS'S SIGN.**

An inability to produce convergence, or even the presence of divergence of the visual axes in the attempt to accommodate vision to near objects. This sign is sometimes seen in exophthalmic goitre. (See Exophthalmos, §. 135.)

#### **MONOPLÉGIA** (Gr. *μονός*, one; *πληγή*, a stroke).

A one-sided paralysis, confined to one circumscribed region, such as one side of the face, one limb, one group of muscles, or even one muscle (see p. 232).

**MORO'S TEST.**

An ointment is prepared of one part of Koch's old tuberculin rubbed up with one part anhydrous lanolin. A portion about the bulk of a pea is to be rubbed into the skin (say of the thorax), while a control inunction of simple lanolin should be used on the opposite side. In tuberculous cases the portion anointed with Moro's reagent shows in a day or so a papular or pustular eruption, which disappears in a week, while healthy subjects are stated by some observers to have no reaction. This may be the case in children, and in those under three years of age the test may be regarded as reliable; in adults, however, a positive reaction is stated to occur in 55 per cent. of apparently healthy individuals.

**MORVAN'S DISEASE.**

Among the outstanding symptoms of syringomyelia are certain trophic affections of the fingers, which have been described as a separate affection under the name Morvan's disease. The term is a convenient means to indicate in a word the group of anatomical changes implied by the term, just as one speaks of 'Charcot's joint' or 'the tabetic foot,' but it seems unnecessary to raise this symptom-group into a separate disease.

The conditions found are painless whitlows on the fingers, causing deep ulceration and even necrosis of the terminal phalanges, together with œdema, bullæ, and injury to the nails.

**MOVEMENT, Abnormalities of.**

The movements of the body which are normally under the control of the individual may be disturbed by disease in three different directions—viz., (A) Weakened or abolished movement in the affected part; (B) increased or exaggerated muscular action; and (C) perverted or disorderly movements of the part.

Of the group of affections presenting (A) evidence of diminished muscular action, we may recognize in very many cases—(i.) the peripheral or central nervous system as the seat of the affection; in others (ii.) the muscles themselves are at fault; or (iii.) the bones and the various structures entering into the formation of the joints may be affected.

The various affections characterized by (B) increased, or (C) perverted muscular action are, almost without exception,

instances of disease situated in the central or peripheral nervous system.

The disorders of motility are considered in separate articles under the headings Movement Decreased (p. 207), Movement Increased (p. 249), and Movement Disorderly (p. 245).

A brief reference to the anatomy and physiology involved may be here permitted. Contractions of the voluntary muscles are excited by impulses travelling from the cord or brain via the efferent nerves. These consist of bundles of nerve fibres, each of which is an axis-cylinder process (or **axon**) extending peripherally from its parent ganglion cell. The latter, in the case of spinal nerves, are situated in the anterior horns of the grey matter of the cord, and in the case of cranial nerves, lie in the nerve centres of the medulla, pons, and mesencephalon. These ganglion cells, with their axons, are known as the **peripheral neurons**, and form the **lower segment** of the **motor tract**. The ganglion cells are in functional connection, by means of mutually interlacing branched processes, directly or indirectly, with (*a*) fibres coming from that portion of the cerebral cortex known as the Rolandic or sensori-motor area, via the internal capsule, medullary decussation, and pyramidal tracts. This series of fibres, together with the cortical ganglion cells of which they are the axons, are the **central neurons**, and form the **upper segment** of the motor tract (see Fig. 28, p. 204). Further connections are effected in a similar manner between the ganglion cells of the peripheral neurons and (*b*) fibres proceeding to the cerebellum, (*c*) afferent fibres arriving from the periphery, and (*d*) afferent fibres proceeding via the optic thalamus, etc., to the sensori-motor and other portions of the cortex.

The efferent nerve impulses mentioned above as causing muscular contractions, and proceeding from the anterior cornual ganglion cells, are the result of impressions reaching the cells from the periphery, from the Rolandic area of the cortex, and probably from the cerebellum. The combined influences, central and peripheral, acting upon the peripheral neurons, produce in a healthy and educated individual the marvellously co-ordinated action of muscles and muscle groups. In order that this result may be secured, all the nerve paths and structures must be intact. Any defect in their course will produce a disorder of movement. We can now readily understand that an interruption in any portion of the motor tract will abolish voluntary contractions in the corresponding muscle fibres; any irritation affecting the same structures will cause excessive muscular action, and any lesion

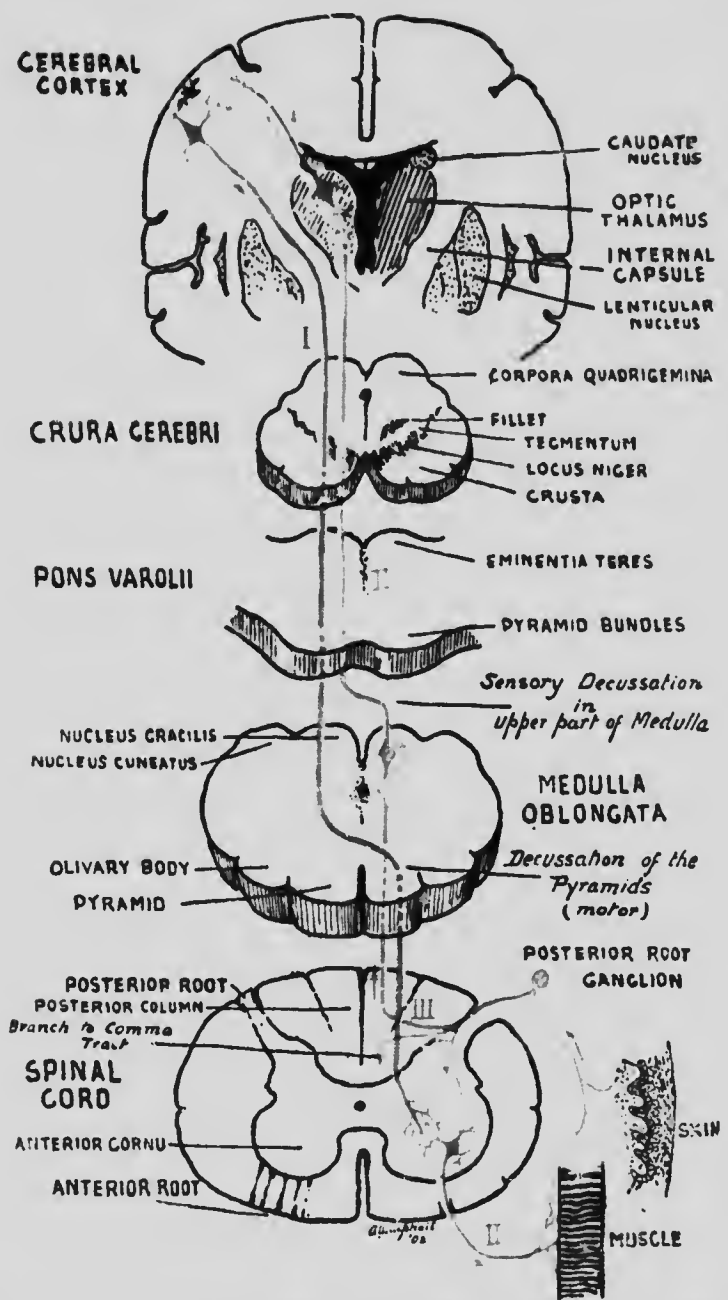


FIG. 28.—DIAGRAM REPRESENTING MOTOR AND SENSORY NEURONS.

The motor path (coloured red) consists of two series of neurons: the upper (I.) extending from the cerebral cortex to the anterior cornu, or to the homologous nuclei of the motor cranial nerves; the lower (II.) extending thence to the muscles. The particular sensory path here figured (in blue) consists of three series of neurons: the lowest (III.) extending from the periphery, by way of the posterior roots and posterior column of the same side, to the nucleus gracilis; the middle (II.) extending from that nucleus across the middle line, and on to the optic thalamus; and the highest (I.) passing thence to the cortex, where its arborizations are seen close to the dendrons of the upper motor neurons. It will be seen that both motor and sensory paths cross the middle line. The lowest part of the illustration shows a reflex arc such as is concerned in the knee-jerk (Monro.)



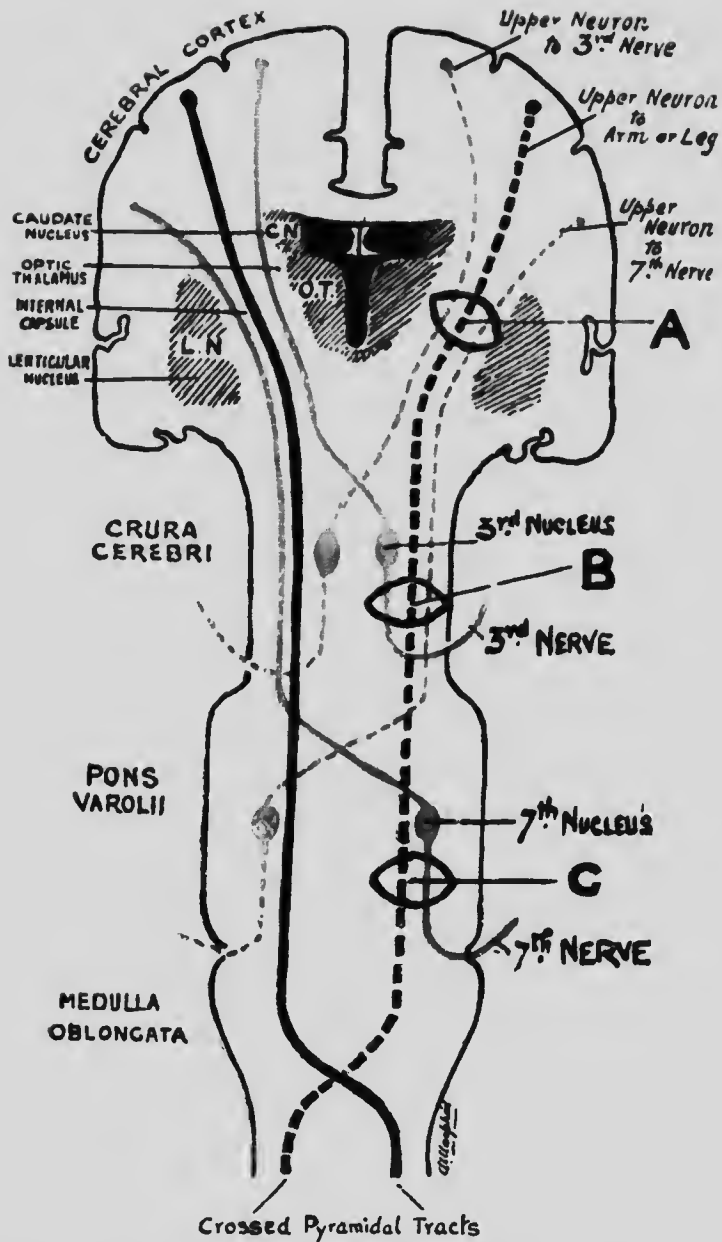


FIG. 29.—DIAGRAM OF THE MOTOR PATH FROM THE CEREBRAL CORTEX TO THE MEDULLA.

The path to the ocular muscles is indicated by blue, to the facial muscles by red, and to the limbs by black. The paths originating in the one hemisphere are indicated by continuous lines, while those starting from the opposite hemisphere are represented by interrupted lines.

It will be seen that a lesion at A (*internal capsule*) involves upper neurons only, so that the paralysis which it causes is on the opposite side (limbs and lower face).

A lesion at B (*crura*) involves the upper neurons for the limbs and face, which are thus paralysed on the opposite side; but it also involves the lower neurons for the ocular muscles, and in this way causes oculo-motor paralysis on the side of lesion.

A lesion at C (*pons*) involves the upper neurons for the limbs, which are thus paralysed on the opposite side; but it also damages the lower neurons for the face, which is therefore paralysed on the side of lesion. (Monro.)

interfering with the transmission of sensory impressions or cerebellar influences may give rise to inco-ordination, which may or may not be accompanied by diminished or by increased muscular action.

In order to determine whether the movements of the body are in any way abnormal, the patient is required to perform voluntary movements of various kinds to which resistance may be offered. The strength, efficiency, and neatness with which the action is performed are to be observed, and the opinion formed whether or not there is any departure from health. The position

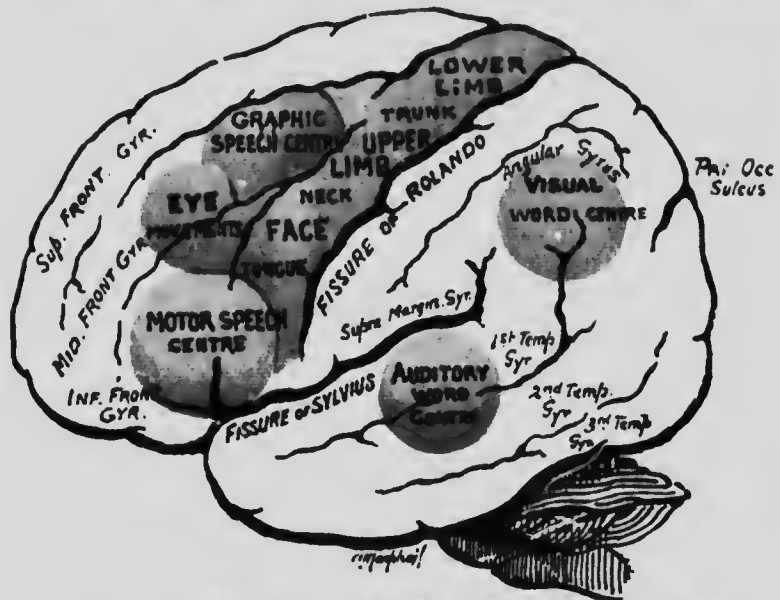


FIG. 30.—LATERAL ASPECT OF LEFT CEREBRAL HEMISPHERE.  
The so-called motor area is coloured red. The four speech centres are in blue.  
(Monro.)

of the body, or the situation of the part affected, may be distinctive—*e.g.*, paralysis of the eye muscles, the face, arm, or leg. Slight degrees of paresis may escape attention if a cursory examination is made, but it is generally possible, especially in one-sided paralysis, where comparison with the healthy side is of much assistance, to detect slight defects in groups of muscles. Instruments to demonstrate the muscular weakness are rarely required, but the dynamometer, as a test for the hand-grip, is not to be despised.

A careful examination of the affected part will in some cases show that the lesion is a local affection of the muscles or joints,

in which the nervous system may have no share. In all cases, of course, it is necessary to inquire fully into the various aspects of the case, in accordance with the methods usually adopted in clinical investigations. (See Case-taking, p. 99.)

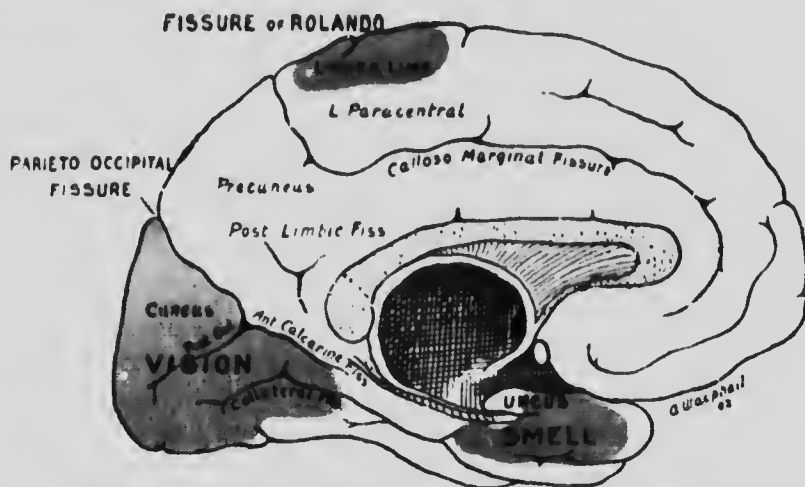


FIG. 31.—MESIAL ASPECT OF LEFT CEREBRAL HEMISPHERE.  
Some of the cortical centres are coloured red. (Monro.)

The further consideration of the disorders of motility is to be found in the articles on Movement Decreased (p. 207); Movement Increased (p. 249); Movement Disorderly (p. 245); Gait (p. 148); Thorax Movements, etc. (p. 469); Abdomen Movements, etc. (p. 10).

### MOVEMENT DECREASED.

Loss of motor power due to nervous affections—Paralysis: spastic and flaccid.

Conditions characterized by spastic paralysis:

- A. Spinal: Transverse interruption of the cord the result of compression, myelitis, or hæmorrhage—Sclerotic changes in the upper segment of the motor tract, occurring in primary lateral sclerosis, hereditary spastic paraplegia, amyotrophic lateral sclerosis, ataxic paraplegia, primary combined sclerosis, multiple sclerosis, syringomyelia, pellagra.
- B. Cerebral: from hæmorrhage, tumours, injuries, or degenerations.
- C. Peripheral nerve lesions.
- D. Functional disorders.

Conditions characterized by flaccid paralysis: Chronic degenerations of the ganglion cells in the motor nerve nuclei, comprising

progressive spinal muscular atrophy, bulbar paralysis, and ophthalmoplegia; acute inflammatory changes in the same situations, including infantile paralysis, acute bulbar paralysis, acute ophthalmoplegia, Landry's paralysis, lesions of the peripheral nerves.

Strabismus—Squint—Secondary deviation—Double vision—Erroneous projection—Facial paralysis—Phrenic paralysis—Erb's paralysis—Klumpke's paralysis—Dropped wrist—Dropped foot.

Hemiplegia—Lesions of the Rolandic region: of the centrum ovale; internal capsule; crura; pons—Crossed paralysis—Conjugate deviation—Lesions of medulla—Brown-Séquard's syndrome.

Paraplegia—Paraplegia due to lesions other than those of the spinal cord.

Loss of power due to disease of the muscles.

Loss of power due to disease of the bones, ligaments, and other tissues in the neighbourhood of the joints—Summary.

The voluntary muscular contractions in some portion of the patient's body are found to be deficient in strength, or to be abolished completely.

When a careful examination fails to locate the cause of the defective movements in the muscles, joints, or bones of the affected region, we may assume that the **Nervous System**, central or peripheral, is at fault, and the condition is one of **Paralysis**. This term should be reserved for those cases where the loss of power due to nervous disease in the part is complete, the word **Paresis** being employed to signify a partial loss of power. This distinction of terms is, however, not always observed.

Having decided that one is dealing with a case of paresis or paralysis, two points must next be determined—viz.:

(I.) What is the **condition** or **tone** of the affected muscles?

(II.) What is the **distribution** of the symptom—*i.e.*, the situation of the paralysed muscles?

### I. **Tone of the Affected Muscles.**

By the **tone** of a muscle is meant the amount of involuntary contraction of which it is capable. When this is considerable, the muscle is maintained in a fair state of nutrition and volume. Those forms of paralysis in which the loss of voluntary contractility is accompanied by involuntary tonic contraction are termed **spastic** or **tonic paralysis**. On the other hand, certain paralytic conditions are observed in which both voluntary and involuntary contractility are lost, the muscles becoming flaccid and wasted. The condition is then termed **flaccid** or **atrophic paralysis**.

The distinction between the spastic and flaccid forms of paralysis is, as a rule, made without difficulty. In both forms voluntary

movements are impossible. In the former the muscles can be felt between the fingers to have a certain degree of tone or rigidity, the reflexes are retained or may be increased, and the electrical reactions are unchanged from the normal. In the latter form the muscle is flabby and greatly wasted, the reflexes are diminished or lost, and the reaction of degeneration (see p. 329) may be observed. As described elsewhere (see Reflexes, p. 334), muscular tone is maintained by an automatic reflex process, the efficiency of which depends on the integrity of a 'reflex arc.' Lesions of the lower segment of the motor tract interrupt this reflex arc; hence an absence of muscular tone results from lesions in this region. On the other hand, lesions of the upper segment do not interfere with the reflex condition; hence the loss of volitional movement is accompanied by involuntary tonic muscular contraction, which may not only equal healthy muscular tone, but may exceed it, in consequence of excessive reflex action. The presence or absence of spasm in the affected region will then materially assist in locating the lesion causing paralysis. Thus it is obvious that cerebral lesions can but rarely cause flaccid paralysis. This can only occur in muscles supplied by cranial nerves whose nuclei or intracranial portions are destroyed by the lesion. In peripheral nerve lesions, on the contrary, flaccid paralysis is practically invariably found, and in several forms of spinal disease the 'nuclear reflex arc' is destroyed, causing flaccid paralysis, atrophy, and loss of reflexes.

i. **Spastic or Tonic Paralysis**, then, occurs in the following conditions:

A. **Disease of the Spinal Cord**: either a transverse interruption from any cause or sclerotic changes in the upper segment of the motor tract.

**Transverse Interruption of the Cord** usually causes spastic paralysis, but Bastian has shown that a **complete** interruption of the cord is generally followed by loss of reflexes and flaccid muscles in the regions below the level of the lesion. Transverse interruption may be due to the following conditions:

1. *Compression of the Cord* from (a) Caries of the vertebræ (Pott's disease), occurring most frequently in childhood, and producing angular curvature of the spine. In the early stages slight loss of power may be noticed, with increased reflexes. The disease in the spinal column at this period may be overlooked, unless a careful examination be made. Sudden pressure on the vertex, a smart shock on the heels with the legs extended, percussion

down the length of the spine, or the application of a warm sponge to the vertebral column, will elicit pain long before a deformity is found. (b) Tumours of the meninges and of the vertebræ: carcinomatous, sarcomatous, and syphilitic. (c) Pachymenin-



FIG. 32.—SPASTIC CEREBRAL PARAPLEGIA.

gitis, especially the hypertrophic form. (d) Aneurism of the aorta or of its branches. (e) Traumatic lesion of the vertebral column, and intrameningeal hæmorrhage.

2. *Myelitis*.—Acute and chronic inflammation of the cord, which may be either transverse or diffuse.

3. *Hæmorrhage into the Cord* produces a sudden interruption in the motor tract. The paralysis is usually better marked on one side than on the other, the hæmorrhage having been in the first instance, of course, unilateral.

The symptoms produced by transverse interruption of the cord must vary with the situation of the lesion. If in the neck, paralysis of all four limbs results from a complete interruption; if the lesion is as high as the fourth cervical segment, paralysis of the diaphragm may ensue from implication of the phrenic nerve. Caries of the atlas or axis is not uncommon. At that level complete interruption would soon result in death, but in many cases the pressure from the inflammatory process is exerted in other directions, producing dysphagia (from retropharyngeal abscess), pupillary and vaso-motor disturbances (pressure on the cervical sympathetic), etc. In the dorsal region the arms escape, and the girdle sensation is often experienced. When the lesion is in the lumbar region, the lumbar enlargement and the cauda equina may be injured, with the result that the functions of the bladder (see Micturition, Disorders of, p. 195) and rectum are disturbed, and the paralysis is of the flaccid type, with diminished reflexes and impaired muscular nutrition.

Spastic paralysis due to **Sclerotic Changes in the Upper Segment of the Motor Tract** are seen in—

1. *Primary Lateral Sclerosis, or Simple Primary Spastic Paralysis.*—In this condition, as described by Grainger Stewart, Dreschfeld, and others, sclerosis of the lateral columns is found without any evident causative lesion higher in the motor tract. The muscles grow gradually weaker, but more spastic, the joints becoming less supple and the reflexes increased. In walking the patient's muscles are unnecessarily tense, and he proceeds with the stiff gait of one who walks on a slippery surface. As the disease progresses the rigidity increases, the toes catch the ground in walking, and tonic or clonic spasms give annoyance. An instance of the latter is the sudden spasmodic extension of the leg which sometimes results from a movement of extension, and has been appropriately termed the 'clasp-knife reaction.'

2. *Hereditary Spastic Paraplegia*, a disease of infancy and childhood, occurring in families. It chiefly affects the legs, and is due to sclerosis of the lateral columns.

3. *Lateral Sclerosis*, complicated with other lesions of the cord. Several combinations of this nature may be mentioned:

(1) *Amyotrophic Lateral Sclerosis, or Charcot's Disease.*—A com-

bined degeneration of the lateral columns, of the anterior grey matter of the cord, and of the medullary and pontine nerve centres, exhibits the rigidity of spastic paraplegia, with the wasting of progressive muscular atrophy and bulbar paralysis. The spasm and increased reflexes persist as long as any muscle fibres remain contractile, and the contractures consequent upon muscular atrophy are exaggerated by the spasm. (See Contractures. p. 104.)

(2) *Ataxic Paraplegia* is a disease of the cord exhibiting the symptoms of spastic paraplegia combined with ataxia. The lesion is a sclerosis of the dorsal and lateral columns, and, unlike *tabes dorsalis*, it does not seem to be a result of syphilis.

(3) *Primary Combined Sclerosis of Putnam and Dana* is a spastic paraplegia of adults affecting the lower limbs, with both motor and sensory symptoms. The disease is a degeneration of the cord, affecting chiefly the dorsal and lateral columns, and is closely associated with various forms of anæmia.

(4) *Multiple Sclerosis (Disseminate or Insular Sclerosis)*.—Patches of sclerosis occur in the pons, cerebellum, basal ganglia, centrum ovale, and in the spinal cord, indiscriminately distributed in both white and grey matter. Muscular weakness, rigidity, and tremor are among the most important of the motor symptoms.

(5) *Syringomyelia* is a cystic condition of the spinal cord. The symptoms vary greatly, according to the exact situation and extent of the cavity. The resulting paralysis is usually of the spastic type, and the case may eventually present the motor symptoms of amyotrophic lateral sclerosis, with the characteristic sensory and trophic disturbances of syringomyelia.

(6) *Pellagra*, a disease seen chiefly in Southern Europe, exhibits symptoms resembling amyotrophic lateral sclerosis, and is caused by the ingestion of a poison occurring in diseased maize. Degeneration of the lateral and dorsal columns and atrophy of the ganglion cells in the anterior cornua have been found. A spastic paralysis is also produced by the use of a vetch—the *lathyrus*. The disease, which is termed *lathyrism*, occurs chiefly in India.

B. Less frequently spastic paralysis occurs as a result of **Cerebral Disease**. Here again the lesion must be above the nuclei of the motor nerves supplying the affected muscles. It is, therefore, found in disease or injury of the Rolandic area, or of the nerve fibres proceeding thence to the periphery. Hæmorrhage into the internal capsule, crura, or pons, tumours or



degenerative changes in the same regions, may produce a spastic type of paralysis (see below, p. 232).

C. Spastic paralysis from **Disease of the Nerves** is obviously an improbable event. It is stated, however, that one sometimes observes in the early stages of peripheral neuritis a condition of irritation in which there is loss of voluntary movement, with retention or exaggeration of muscular tone and reflexes.

D. A form of spastic paralysis may be seen which is independent of any obvious lesion of the nervous system. It occurs in **Hysteria** and allied conditions. The rigidity is usually less and the reflexes more marked than in the spinal affections. It is said that hysterical paralysis may develop into an organic form of spastic paraplegia, but such cases are probably instances of the organic disease occurring in a hysterical subject. It may form one of the features of traumatic neurasthenia, as in 'railway spine,' the result of injury and shock produced by a railway accident.

ii. **Flaccid Paralysis** is the result of (A) disease of those portions of the peripheral motor neurons contained in the brain and cord—*i.e.*, the motor nuclei situated in the anterior spinal cornua, in the medulla, pons, and crura; (B) disease of the remainder of the peripheral motor neurons—*i.e.*, of the peripheral motor nerves.

A Flaccid paralysis, due to disease of the central nervous system, occurs as (a) chronic degenerative changes in the ganglion cells of the motor nuclei, and (b) acute inflammatory affections of the same structures.

Division (a) comprises **Progressive Spinal Muscular Atrophy**, **Bulbar Paralysis**, and **Ophthalmoplegia**.

**Progressive Muscular Atrophy** is a disease of adult life, usually occurring in males under middle age. The cervical portion of the cord is the region commonly affected, giving rise to a wasting palsy of the upper extremities. It commences most frequently in the small muscles of the hand, or in the deltoid and other shoulder muscles, runs a slow, progressive course, and does not as a rule cause sensory disturbances. It is frequently associated with sclerosis of the lateral columns, and then constitutes the affection known as amyotrophic lateral sclerosis (Charcot). There may also be associated with the spinal affection that of the medullary nerve nuclei—*viz.* :

**Bulbar Paralysis.**—This may occur as an independent disease, or, as just stated, it may form part of a widespread degeneration, involving the nuclei of many of the spinal and cranial motor

nerves, together with in many cases sclerosis of the pyramidal tracts, and at times of the posterior columns of the cord. In the latter conditions the bulbar affection is often the latest development. Paralysis of the tongue, lips, palate, etc., is the result.

The oculo-motor nuclei in the crura may occasionally share in this extensive degenerative process, or less rarely they may undergo an independent sclerosis of syphilitic origin, giving rise to the affection known as **Ophthalmoplegia**. The external muscles of the eyeball may alone be paralysed (ophthalmoplegia externa), or the loss of power may be in the internal ocular muscles (ophthalmoplegia interna), as shown by inaction of the pupils and loss of accommodation.

(b) Flaccid paralysis is also produced by **Acute Inflammatory Changes** in the same situations as those just referred to—namely, in the anterior horns of the cord, and in the medullary, pontine, and crural motor nerve nuclei. The symptoms vary in accordance with the seat of the lesion. The following affections belong to this category:

1. **Infantile Paralysis** (acute anterior poliomyelitis), occurring most frequently in children under five years. An acute inflammation, probably of an infective and of a circulatory origin, destroying the ganglion cells in the anterior horns of the cord. An atrophic paralysis is the result. A similar condition is seen less often among adults. Excessive exercise in hot weather or exposure have been held responsible, but the origin of the affection is quite uncertain.

2. An acute form of **Bulbar Paralysis** is also on rare occasions observed.

3. A similar acute inflammation may attack the nuclei of the oculo-motor nerves, giving rise to an acute **Ophthalmoplegia**.

4. **Landry's Paralysis**.—An acute inflammation of the lower motor neurons first described by Landry. It is probably a peripheral nerve lesion, but evidence of the involvement of the spinal grey matter has been furnished.

B. Flaccid or atrophic paralysis may also be due to lesions of the peripheral efferent nerves. An interruption in their course separates the distal portion of the nerve and the attached muscle fibres from their trophic centres, the anterior cornual ganglion cells (or the medullary, pontine, or crural nuclear cells in the case of cranial nerves). The result is the same as that following damage to the cells themselves—viz., loss of power, diminished

reflexes, muscular wasting, etc. (see above). These symptoms are usually restricted to one side of the body.

The paralysis produced by these lesions gives rise to a great variety of symptoms, in accordance with the region affected. These are enumerated seriatim in the following description of the cranial and spinal nerve lesions. The table on p. 227, copied from Monro, may be referred to.

We may here, for convenience' sake, include the conditions due to injury or disease of both sensory and motor nerves, and, in order to avoid repetition, lesions of their respective connections in the medulla, pons, mesencephalon and anterior cornua have to be included in the descriptions.

*Cranial Nerve Lesions—Causation.*—Pressure of intracranial tumours, hæmorrhages, or inflammations; disease of the nerve nuclei, followed by descending degeneration of the nerve; fractures and disease of the bones of the skull; injury to the nerve trunks in their extracranial course from blows or wounds, pressure of inflamed glands or tumours; inflammation or degeneration of the nerves as a result of some toxic state of the blood—*e.g.*, rheumatism, diphtheria, lead-poisoning, alcoholism, etc.

The **Olfactory Nerve** may be damaged by intracranial disease or injury; excessive stimulation of the nerve, and hysteria may also cause disturbance of the nerve's function.

The *Symptoms* are loss of the sense of smell (anosmia), excessive acuteness of the sense (hyperosmia), or perverted and morbid sense of smell—*e.g.*, the aura of epilepsy, and in insanity.

**Optic Nerve.**—The exposed position of the optic nerves, chiasma, and tracts, lying between the base of the brain and the floor of the skull, gives rise to many lesions of this nerve. Inflammation of the nerve (optic neuritis) is the result of a variety of morbid conditions—*viz.*, (1) intracranial disease—*e.g.*, tumours in the brain and in the cerebellum, tumours and inflammation in the meninges, thrombosis of the cerebral sinuses, abscess, chronic hydrocephalus; (2) orbital tumours, inflammation, or aneurism; (3) general diseases, such as chlorosis, Bright's disease, enteric fever, lead-poisoning, septicæmia. At a later stage of the affection the optic nerve may become atrophied. It is, however, not unusual to find optic atrophy a primary affection, as in locomotor ataxia. The effect of a lesion of this nerve is to cause a disturbance of vision. The various visual disorders are treated in the article on Disturbances of Vision (p. 543).

**Oculo-Motor Nerves** (Third, Fourth, and Sixth Cranial

Nerves).—Paralysis of these nerves is usually a nuclear or peripheral defect (rarely due to supranuclear lesions). It results in limitation of movement of the eye and upper lid, strabismus, secondary deviation, erroneous projection, double vision.

The **third** is the motor nerve for all the external muscles of the eyeball except the superior oblique and the external rectus. It also innervates the sphincter of the iris, the ciliary muscle, and the levator palpebræ. Paralysis of this nerve causes ptosis; outward squint; inability to direct the eye upwards, downwards, or inwards; rotation of the eyeball on an antero-posterior axis, in the direction of the hands of a clock (as seen by the observer), on attempting to look downwards and outwards, from action of the intact superior oblique muscle; dilatation and immobility of the pupil; loss of accommodation. A slight protrusion of the eye may be observed when most of the eye muscles are paralysed. As a rule, the paralysis is incomplete, and the defects of movement may be only imperfectly developed.

The **fourth** nerve, supplying the superior oblique muscle, is rarely paralysed alone. Imperfect downward and inward movement is the result.

The **sixth** nerve, owing to its situation below the pons, is not infrequently damaged by diseases of the cerebellum, pons, basal meninges, and middle fossa of the skull. The result is inability to direct the eye outward, and convergent squint on looking toward the affected side.

**Limitation of Movement** is shown by more or less complete inability of the affected eye to follow the movements of the sound eye, by drooping of the upper lid (**ptosis**), and by one or more of the disturbances just mentioned.

**Ptosis** is usually unilateral. When congenital, it depends, as a rule, on a lesion of the nerve, and not of its nucleus. A spasmodic contraction of the orbicularis palpebrarum may simulate ptosis. In the former, however, the eyebrow is lower, and the surrounding wrinkles are better marked than on the sound side, while the contrary is the case in paralytic ptosis. In the latter also the inability to raise the upper lid is more evident. **Sympathetic ptosis** may be mentioned as occurring sometimes from lesions of the sympathetic, causing paralysis of Müller's muscle; the result is a narrowing of the palpebral fissure, with other evidence of disturbed sympathetic nerve influence—*e.g.*, contracted pupil, vaso-motor and secretory changes. Careful examination shows that the upper eyelid is not really paralysed.

Lastly, **reflex ptosis** may be seen as a result of peripheral irritation, a common cause being carious teeth.

**Squint (Strabismus).**—With paralysis of one or more of the oculo-motor nerves the normal position of the visual axes becomes disturbed. Paralysis of one external rectus causes the axes to converge in front of the 'fixation-point' (**convergent strabismus**), while paralysis of one internal rectus results in divergence anteriorly of the visual axes (**divergent strabismus**). This departure from the normal position of the axes, which should cross at the fixation-point, is termed **primary deviation**, and squinting as a result of paralysis of oculo-motor muscles is known as **paralytic strabismus**, as distinguished from **concomitant strabismus**, which is usually produced by hypermetropia. In paralytic strabismus an exaggeration of the squint is noticed when an effort is being made to bring the paralysed muscle into action. In a case of, say, convergent squint due to paralysis of the right external rectus muscle the sound (left) eye is covered, and the patient is directed to fix an object with his right eye. The increased effort made to produce a contraction of the paralysed right external rectus calls forth an excessive contraction of the left internal rectus, the muscle which habitually works in 'double harness' with the right external rectus. The result of this experiment is to increase the squint, and the phenomenon is termed the **secondary deviation**. A similar change occurs in the divergent squint due to paralysis of the internal rectus—the divergence is increased on attempting to call the paralysed muscle into play.

It is important to distinguish the concomitant squint from the paralytic. In the former the secondary deviation does not occur, and the angle enclosed by the visual axes remains nearly fixed in spite of the varied movements of both eyes; the paralytic squint, on the contrary, changes in degree with each movement of the eyeballs, and is more marked when the eyes are turned toward the paralysed side.

Paralysis of the superior and inferior recti and of the oblique muscles causes not only squinting, but also produces disturbance of the wheel movement, or rotation of the eyeball on the antero-posterior axis. The slighter forms of paralytic squint may be difficult to detect by inspection, but may often be recognized by the presence of **double vision**. This is commonly a symptom of paralytic, but not of concomitant, squint. In the latter condition the 'false image' is ignored, or, as suggested by Swanzy, a new physiological macula lutea may be formed.

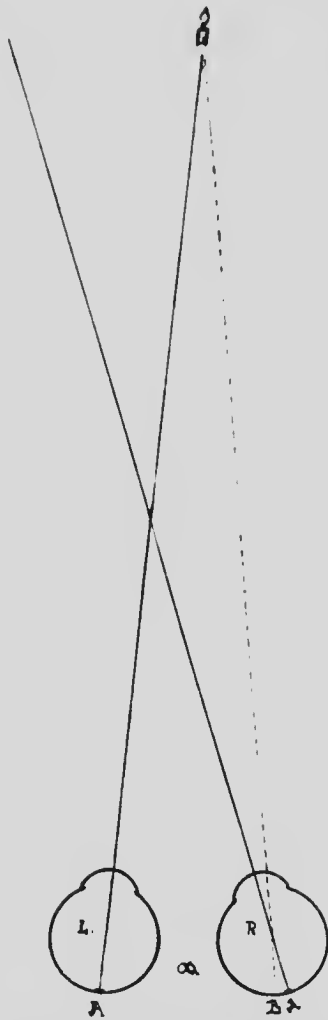
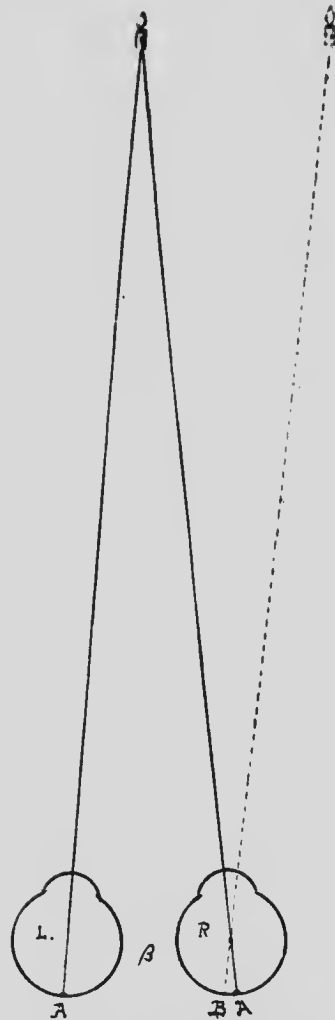


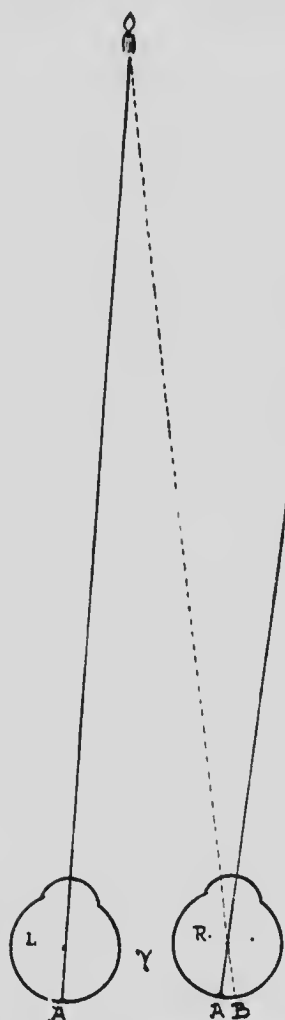
FIG. 33.—CONVERGENT SQUINT.

The right external rectus is paralyzed; the visual axes cross in front of the fixation-point (the candle). Rays from the object fall on the point B of the retina of the affected eye. A is the macula lutea of either eye; L, left eye; R, right eye. The unbroken straight lines represent the visual axes; the broken line the path of light rays from the object to the retina of the affected eye.

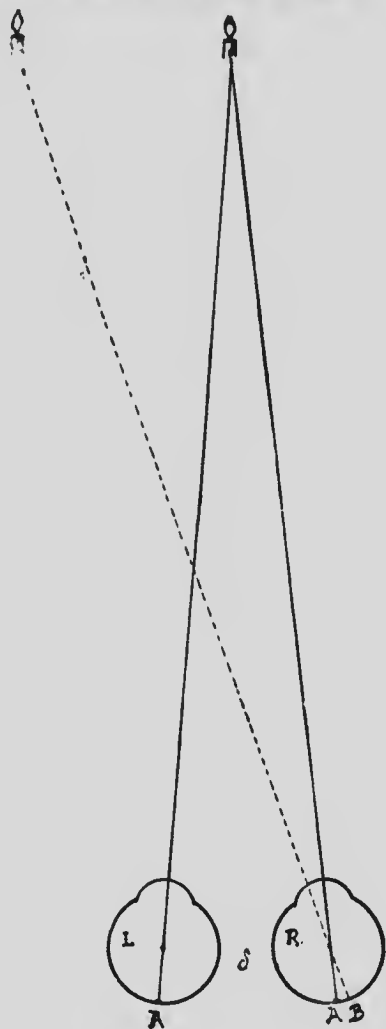
FIG. 34.—CONVERGENT SQUINT—  
HOMONYMOUS DIPLOPIA.

The lesion is the same as Fig. 33. The image formed at point B in Fig. 33 causes the impression that it is in the position shown to the right of the fixation-point. The image thus displaced is fainter than that received by the sound eye, as it does not fall on the macula lutea, and is known as the *false image*.

**Double Vision.**—As a result of the faulty direction of the visual axes in strabismus from any cause, a separate retinal im-



**FIG. 35.—DIVERGENT SQUINT.**  
Paralysis of the right internal rectus. The rays from the object fall on the point B, situated to the outer side of the macula lutea, A.



**FIG. 36.—DIVERGENT SQUINT—CROSSED DIPLOPIA.**  
The same lesion as Fig. 35. The patient imagines that the visual axes are in the normal position shown by the unbroken lines. The image is, however, received on the point B of the retina (see preceding figure), and is in consequence projected to the position shown by the dim candle—*i.e.*, to the sound side of the true image.

pression of an object is transmitted from each eye to the visual centres. That from the sound eye is the more distinct, coming from the macula lutea, and is termed the **true image**, whilst that from the paralysed eye, proceeding from a less sensitive part of the retina (see Fig. 33), is indistinct, and is termed the **false image**. When the false image is on the same side of the true image as the paralysis (which occurs in convergent strabismus), it is termed **simple** or **homonymous diplopia**; when the false image appears to the unparalysed side of the true image, it is termed **crossed diplopia**—this is found in divergent strabismus. The accompanying figures 33 to 36 explain the origin and position of the false image, in cases of paralysis of the internal or external rectus.

Double vision, due to want of parallelism of the visual axes, is called **binocular diplopia**, as both eyes are involved, and closure of one eye removes the second image. A form of diplopia due to affections of the lens, cornea, or iris, and to hysteria, and observed by the injured eye alone, is termed **monocular diplopia**.

When the eyes look directly forward, paralysis of the internal or external rectus causes simply defective lateral or horizontal rotation of the eyeball, without involving the wheel movements or turning on the antero-posterior axis. The two images are, therefore, parallel. Paralysis of any of the remaining four muscles of the eye produces disturbance of the wheel movement, which is best recognized by its effect on the false image. The wheel movement also occurs normally when the lateral muscles act in conjunction with the superior or inferior recti. When, therefore, the eyes are directed upwards or downwards, and at the same time inwards or outwards, paralysis of the internal or external recti is shown by the inclination of the images towards or from each other, according to which muscle or muscles happen to be affected. To remember the positions assumed by the images in paralysis of the different orbital muscles is no easy matter, and the accompanying Fig. 37, the ingeniously-constructed diagram of Werner, will be of assistance.

**Erroneous Projection.**—In order to form a correct estimate of the distance through which a limb or any other portion of the body has moved, or of the position of a limb, one judges chiefly by the amount of nerve energy made use of in the muscular act. This is made known to our consciousness by the **strength sense**, or **sense of innervation** (see p. 216). When the eye muscles are



weakened, a greater nerve effort than usual must be made in directing the eye towards the object, and the distance traversed by the eyeball is, in consequence, misjudged, especially if the object move. If now the patient be required to touch the object looked at, he may be unable to do so accurately, but will touch a spot in advance, in the direction in which the eye was moving. In addition, the equilibrium of the body is to some extent dependent upon the visual judgment of the relation of surrounding objects to our bodies; therefore this deficient judgment may cause a giddiness, termed **ocular vertigo**, which persists even when the eyes are closed. (See Vertigo, p: 539.)

The **Trifacial** or **Fifth Nerve** may be injured at its origin in the pons by hæmorrhage, tumours, degeneration, etc. Tumours

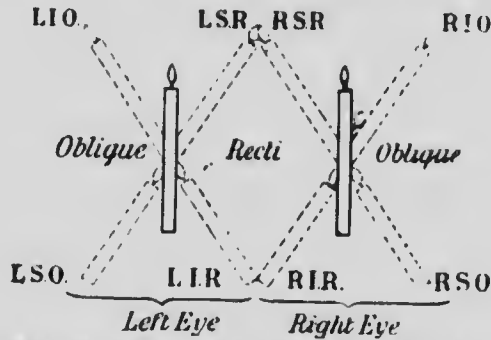


FIG. 37.—DIAGRAM OF THE DIPLOPIA DUE TO PARALYSIS OF ANY ONE OF THE ELEVATOR OR DEPRESSOR MUSCLES.

The form of diplopia which characterizes paralysis of each muscle is expressed by the position of the dotted candle bearing the name of the muscle. The dotted lines represent the false images belonging to the affected eye, the continuous lines the true images. (Werner.)

or basal meningitis may damage its trunk, while its divisions in front of the Gasserian ganglion may receive injury from violence, pressure, or inflammation. The symptoms arising in consequence of such injury may be—(a) Sensory: anæsthesia of that portion of the face and mouth which is supplied by the affected nerve or division. Pain or tingling sensations may precede the anæsthesia; loss of taste may be complete in lesions of the root of the nerve. (b) Trophic: inflammation of the cornea; swelling and ulceration of the gums, with loosening of the teeth; herpes zoster, etc. (c) Motor: loss of tone in temporal and masseter muscles on clenching the jaws. The jaw cannot be deviated to the sound side, and movement of the jaw, especially depression, causes it to

deviate to the paralysed side, owing to paralysis of the external pterygoid muscle.

**Facial Paralysis (Bell's Paralysis)** is the result of destructive lesions of the seventh nerve or of its nucleus. The nucleus may be damaged by injury to the pons from hæmorrhage, softening as a consequence of thrombosis, tumours, etc. (see below, p. 240). The nerve trunk may receive injury from intracranial pressure (tumours, etc.); from inflammations—*e.g.*, rheumatism, exposure to cold, ear disease, especially in its course through the temporal bone; from violence—*e.g.*, fracture of the base of the skull, or blows on the extracranial portion of the nerve.

The affected side appears expressionless, owing to the loss of mobility and to the disappearance of wrinkles. If the lesion is supranuclear, the loss of movement will be less noticeable in the muscles of the forehead and eye than in the lower part of the face, more especially when the movements are emotional rather than voluntary. This is owing to the fact that these muscles usually contract symmetrically when actuated by the emotions, and are believed to be innervated by both crossed and uncrossed fibres proceeding from both cortical centres to each nucleus in the pons. On the other hand, if the lesion is nuclear or peripheral, the forehead cannot be wrinkled so thoroughly on the paralysed as on the sound side, and the eye cannot be closed. Inability to close the eye is termed **lagophthalmus**, or **hare's eye**, after the popular belief that the hare sleeps with open eyes. The attempt to close the eyes under these circumstances results in the eye being rotated upward and outward under the unlowered lid—the so-called **Bell's phenomenon**. In addition, the corneal reflex (closure of the lids when the cornea is touched) will be very imperfectly performed. The lower lid droops and the eye waters (epiphora). The muscles of the cheek and mouth on the affected side are flaccid. Saliva may overflow, and particles of food accumulate between the cheek and the gums; the patient cannot whistle, and breathing is somewhat noisy, especially during sleep. The normal folds and wrinkles of the skin disappear, and the muscles are soft and atrophied. On attempting to smile or show the teeth the angle of the mouth on the sound side is retracted, while the affected side either remains motionless or is even drawn slightly across the middle line. The patient may not be able to speak distinctly, the labials being pronounced with difficulty. The nostril on the paralysed side may appear smaller than the

other from paralysis of the levator *ala nasi*. The tongue in facial paralysis of peripheral origin, is unaffected; it may be deviated to the **sound** side, but this will be seen on careful examination to be the result of the patient's attempt to keep the tongue in the centre of the displaced mouth, and not to any paralysis of the tongue muscles. When the lesion is in the pons, however, the nucleus of the twelfth nerve may also have suffered damage, producing a deviation of the tongue towards the **paralysed** side (genioglossal paralysis). It is said that paralysis of the soft palate and uvula occurs in facial paralysis, but authorities differ as to this symptom. Paralysis of the external ear muscles occurs, but it is usually difficult to demonstrate. The stapedius muscle is innervated by the facial, and its paralysis results in **hyperacusis**, an increased sharpness of hearing, in which the low-pitched notes are in particular loudly heard. This symptom is probably due to excessive action of the tensor tympani when released from the opposition of the stapedius. The nerve-supply for this little muscle, like that for the forehead and eye-closing muscles, is probably controlled by fibres from both hemispheres, so that this symptom is not found in central or supranuclear facial paralysis.

Lesions in the pons frequently result in damage to nerve nuclei on both sides, and to pyramidal fibres from both sides of the Rolandic area, giving rise to diplegia—*e.g.*, diplegia facialis and cerebral paraplegia or paralysis of the limbs of both sides.

In addition to hyperacusis from paralysis of the stapedius, the sense of hearing may be disturbed by lesions of the base of the brain, injuring both the facial and the acoustic nerves, which lie close together at their emergence from the lower border of the pons. Further, lesions in the aqueduct of Fallopius, near the geniculate ganglion, may readily damage the adjoining cochlea and semicircular canals.

The chorda tympani leaves the facial nerve near the lower end of the aqueduct of Fallopius. Lesions in the bony canal, therefore, may give rise to taste defects in the anterior half of the tongue, and suppression or diminution of the salivary secretion.

It must not be forgotten that 'late rigidity' may, by excessive muscular contraction, cause the paralysed side of the face to appear the more contractile of the two. (See Contractures, p. 104.)

The **Auditory or Eighth Nerve** may be injured at its nucleus in the pons by hæmorrhage or other brain affections. Tumours,

inflammation, violence, syphilis, etc., may be the cause of lesions to the nerve or its peripheral terminations. The results are deafness, various subjective sounds, such as ringing, rustling, rushing noises (tinnitus), vertigo.

The **Glosso-pharyngeal** or **Ninth Nerve** is rarely affected alone, and its lesions are hardly to be distinguished.

### FACIAL PARALYSIS

	LESION.				
	Supranuclear.	Nuclear.	Peripheral.		
<b>History</b> ..	A cerebral lesion	A cerebral lesion	Exposure, rheumatism, injury		
<b>Situation of paralysis</b>	Lower part of opposite side of face	Lower part, or perhaps whole, of same side of face	Whole of same side of face		
<b>Nutrition</b> ..	Muscles not atrophied, except from disuse	Muscles markedly atrophied	Muscles markedly atrophied		
<b>Electrical changes</b>	Absent	Partial or complete reaction of degeneration	Partial or complete reaction of degeneration		
<b>Corneal reflex</b>	Present	Lost or diminished	Lost or diminished		
<b>'Conjugate deviation' of the eyes, if present</b>	The eyes look to the lesion	The eyes look from the lesion	Absent		
			<b>Intra-cranial.</b>	<b>In Aqueduct of Fallopius.</b>	<b>Below Stylomastoid Foramen.</b>
<b>Secretory function (of salivary glands)</b>	Undisturbed	Undisturbed	Undisturbed	Secretion often diminished	Undisturbed
<b>Sense of taste</b>	Undisturbed	Undisturbed	Undisturbed	Often diminished	Undisturbed
<b>Sense of hearing</b>	Undisturbed	Undisturbed	Often disturbed	Often disturbed	Undisturbed

The **Vagus** or **Tenth Nerve** may be injured in its nucleus by degenerative and other changes; in its roots by meningitis, tumours, etc.; in its peripheral course by wounds, toxic blood states (*e.g.*, diphtheritic and other forms of neuritis), tumours, enlarged glands, aneurisms, etc.

The *symptoms* are difficulty in swallowing, paralysis of the vocal cords, Cheyne-Stokes respiration, tachycardia, gastric pain, and vomiting.

The **Spinal Accessory** or **Eleventh Nerve** supplies the sterno-mastoid, and in part the trapezius muscles. Paralysis of the former causes imperfect rotation of the head toward the sound side, and a flattening of the affected side of the neck. Paralysis of the upper part of the trapezius deepens the angle between the neck and the shoulder, lowers the point of the shoulder, displaces the inferior angle of the scapula inwards towards the spine, and renders forced respiration difficult. The arm can hardly be raised above the level of the head, and shrugging the shoulders is imperfectly performed.

The **Hypoglossal** or **Twelfth Nerve** suffers injury from causes similar to those which damage the eleventh. The movements of the tongue are defective in speaking, chewing, swallowing, and in the coarser movements of protrusion, etc. If one side only is paralysed, the various acts may be performed fairly well, but protrusion of the tongue causes it to be deviated toward the affected side by the unopposed action of the healthy genioglossus muscle. The nutrition of the tongue is markedly lowered in most cases, the whole or a part of the organ lying flaccid and wrinkled in the mouth. (See Fig. 69, p. 482.)

It will be noticed that the best-marked examples of paralysis of the cranial nerves are found as a result of lesions of the nerve nuclei, or of the nerve fibres peripheral to the nuclei, with the exception, perhaps, of the lower branch of the facial. Central or supranuclear lesions—*i.e.*, damage to the fibres connecting the cortex with the nerve nuclei—even when all the central fibres to any given nucleus are obviously functionless, do not necessarily completely paralyse the muscle supplied by the nerve in question. Further, the muscles supplied by the cranial nerves are, for the most part, habitually used in conjunction with their fellows on the opposite side of the body—*e.g.*, the eyebrows, the oculo-motor apparatus, the muscles of mastication, and the vocal cords. This pathological observation and this physiological fact are both accounted for by the free intercommunication which exists between *both* cerebral hemispheres and the nuclei of those nerves which habitually provoke symmetrical muscular contractions. This observation can be extended to the rest of the body. Lesions of the supranuclear fibres—*i.e.*, of the upper segment of the

motor tract—have less paralysing effect upon those muscles which habitually produce bilateral movements than upon those which more frequently act independently of the opposite side (Broadbent). For example, in hemiplegia the arms are more powerless than the legs, while the trunk muscles commonly escape. The more frequent involvement of the muscles of the lower half of the face than those of the upper is another example. It is much easier for most persons to raise or depress one angle of the mouth than to do the same with one eyebrow. (See Broadbent's Law, p. 95.)

Lesions of the **spinal nerves** will now be considered, those only being referred to which are of diagnostic interest.

**Phrenic Paralysis.**—A respiration entirely thoracic, with projection of the abdomen during expiration, and retraction during inspiration, indicates **inaction of the diaphragm**. This may be due to disease in the thorax or abdomen, such as pleuritis, pleural effusions, peritonitis, abdominal tumours, tight-lacing, and, more rarely, degenerative changes in the muscle of the diaphragm. Moreover, the normal breathing in women is mainly thoracic during quiet respiration. When the want of power is of nervous origin, it is usually the result of paralysis of the phrenic nerve. This may be due to lesions in the spinal canal involving the third or fourth cervical nerve roots, or the segments of the cord from which they spring (disease or injury of the spinal column, hæmorrhage, pachymeningitis, or tumours), to neuritis (diphtheria, lead, alcohol, beri-beri, etc.), or to lesion of the nerve trunk in the neck or thorax. This formidable condition conduces to congestion of the lungs and bronchial catarrh, and diminishes the respiratory activity of the bases of the lungs, as shown by the weakened breath-sounds to be heard over the lower thoracic regions. As a rule, the lesion is bilateral, but if unilateral it may easily escape recognition.

**Brachial Plexus Paralysis.**—The nerves of the brachial plexus may be damaged in whole or in part by lesions in the spinal canal similar to those mentioned as causing phrenic paralysis, by dislocations or fractures in relation to the shoulder-joint, by wounds, by injuries during parturition (to the infant), and by neuritis.

The following are the principal varieties of paralysis observed in complete or partial lesions of the plexus :

**Complete Brachial Paralysis**, usually traumatic, affects the whole

TABLE SHOWING APPROXIMATE RELATION TO THE SPINAL NERVE ROOTS OF CERTAIN MOTOR, SENSORY, AND REFLEX FUNCTIONS OF THE CORD (MONRO).

Spinal Complexus		<i>Motor.</i>		<i>Cutaneous.</i>		<i>Reflex.</i>	
		C. 1. } Small rotators of head. 2. } Depressors of hyoid bone 3. } Serno-mastoid. 4. } Scaleni. 5. } Levator anguli scapulae. 6. } Trapezius. 7. } Diaphragm. 8. } Shoulder abductors. 9. } Shoulder adductors. 10. } Extensors of wrist and digits. 11. } Elbow extensors, Latissimus. 12. } Small muscles of hand. D. 1. } Flexors of wrist and digits. 2. } Pectorales. 3. } Intercostals. 4. } Rectus et obliqui abdominis. 5. } Hip flexors. 6. } Adductors and knee extensors. 7. } Abductors. 8. } Hip extensors and knee flexors. 9. } Perineal muscles. 10. } Foot muscles.	C. 1. } Occiput and neck. 2. } Top of shoulder 3. } Outer aspect of upper limb. 4. } Digits. 5. } Inner aspect of upper limb. 6. } Skin of chest and abdomen. 7. } Ensiform region (6-7). 8. } Umbilicus (9-10). 9. } Hip. 10. } Anterior surface of thigh. 11. } Knee, except at back. 12. } Inner aspect of leg. L. 1. } Root of penis dorsally. 2. } Back of limb and outer border of foot. 3. } Perineum, lower part of scrotum, and 4. } most of penis. 5. }	C. 1. } Sudden inspiration on sudden pressure 2. } beneath lower costal margin, 3. } 4. } Dilatation of pupil. 5. } 6. } Scapular. 7. } 8. } D. 1. } 2. } 3. } 4. } Epigastric. 5. } 6. } 7. } 8. } 9. } Abdominal. 10. } 11. } 12. } L. 1. } Cremasteric. 2. } 3. } Knee-jerk. 4. } Gluteal. 5. } Plantar. S. 1. } Ankle-convulsion. 2. } 3. } 4. } 5. }			

of the upper limb. Atrophy of the muscles, with the usual electric and reflex changes, may occur, and trophic changes in the skin or joints may be observed.

**Erb's Paralysis**, or the upper-arm type, causes inability to abduct and raise the arm, owing to paralysis of the deltoid, and to flex and supinate the forearm, from paralysis of the biceps, brachialis anticus, and supinator longus, sometimes also the supraspinatus and infraspinatus and the supinator brevis. The fifth and sixth cervical are the nerves affected, and the lesion is often caused by pressure on the shoulder close in to the neck, as may occur with porters or labourers carrying weights.

**Klumpke's Paralysis**, or the lower-arm type, gives rise to paralysis of the small muscles of the hands and of the flexors of the fingers, from involvement of the eighth cervical and first dorsal nerves. There may be, in addition, contraction of the pupil and diminution of the palpebral fissure on the same side, from implication of the cervical sympathetic.

Paralysis of the **serratus magnus muscle** is shown by rotation backward of the lower angle of the scapula on attempting to raise the arm, especially in a forward direction. The bone projects backwards like a wing, and lies nearer the spinal column than normal, owing to the unopposed action of the rhomboids. The arm cannot be well raised above the horizontal; forcible pushing is impossible, and forced inspiration with the arms raised is difficult. The affection is due to paralysis of the **long thoracic nerve**, formed by branches of the fifth and sixth cervicals, and may be produced by pressure or wounds in the neck, by toxic neuritis, or by inflammations adjacent to the nerve. Paralysis of this muscle is more commonly found as part of wider-spread atrophic paralyzes, as in progressive muscular atrophy or the myopathies. (See Trophic Disturbances, p. 487.) Sensory symptoms are often absent.

Paralysis of the **deltoid** interferes with elevation of the arm, and is due to lesions of the **circumflex nerve**, which is one of the terminal branches of the posterior cord of the plexus. The lesions may be caused by dislocations, blows, or crutch pressure, or they may form part of toxic or spinal diseases. Trophic changes may be found, not only in the muscle (atrophy), but also in the joint (adhesions).

**Dropped Wrist** (or dropped hand) is the result of paralysis of the extensors of the wrist and fingers, in consequence of lesions



of the musculo-spiral nerve. The exposed position of the nerve as it winds round the humerus renders it particularly liable to damage from pressure. A common cause is prolonged pressure of the arm on the edge of the bed or the back of a chair during sleep ('Saturday-night palsy'), or a badly-fitting crutch. When the injury is in this, the usual, situation, the triceps, and often the supinator longus, escape. Lying on the arm on a hard bed, fractures, or dislocations may also be causes of the paralysis. Lead-poisoning causes the characteristic lesion of certain branches of the nerve. The result of extensor paralysis is to cause flexion of the wrist and metacarpo-phalangeal joints and opposition of the thumb—*i.e.*, the typical **dropped hand**. In lead-poisoning the palsy is usually bilateral, and the supinator longus escapes. There may be some slight sensory disturbance (tingling, anæsthesia), but these are often absent.

**Median Nerve** (derived from the Sixth, Seventh, and Eighth Cervical and First Dorsal Nerves).—Often injured by fractures or wounds of the forearm, or by neuritis.

*Symptoms*.—Inability to completely pronate the forearm, to flex the wrist toward the radial side, or to oppose the thumb to the tips of the fingers. The first row of interphalangeal joints cannot be flexed, nor can the distal phalanges of the first and second fingers. Wasting of the affected muscles, especially those of the ball of the thumb. Sensation may be lost, or pain and tingling be felt over the radial half of the palm, the palmar surface of the thumb, and adjacent one and a half fingers.

**Ulnar Nerve** (derived from the Eighth Cervical and First Dorsal Nerves).—*Symptoms*.—Inability to flex the wrist toward the ulnar side; lateral movements of the fingers with the hand flat are deficient, the interossei being paralysed; the thumb is abducted; the metacarpo-phalangeal joints are extended; while extension of the two distal sets of phalanges, especially those of the ring and little fingers, is imperfectly performed. In cases of long duration the ulnar type of **claw-hand** results. (See Contractures, p. 105.) Sensory disturbances (pain, burning, anæsthesia) may be experienced over the ulnar region of the hand—*viz.*, anteriorly one and a half fingers, posteriorly two and a half fingers.

**Lumbar Plexus**.—The lesion may be one of those mentioned as causing spinal nerve lesions generally, or it may arise from pelvic or abdominal disease or injury—*e.g.*, psoas abscess, fractures,

difficult labour, etc. The effect is paralysis of the flexors of the hip, of the abductors of the thigh, of the extensors of the knee, and of the cremaster muscle. Lesions of some of the separate nerves of this plexus may be mentioned :

**Obturator Nerve** (from the Second, Third, and Fourth Lumbar Nerves).—Especially liable to injury during parturition.

*Symptoms.*—Paralysis of the adductors of the thigh, with inability to cross the legs; imperfect inward and outward rotation of the thigh.

**Anterior Crural Nerve** (from the Second, Third, and Fourth Lumbar Nerves). The injury may occur in the groin or in the pelvis.

*Symptoms.*—Inability to extend the knee; loss of knee-jerks; difficulty in flexion of hip in pelvic lesions, when the branch to the iliacus may be implicated; anæsthesia or paræsthesia over the front and inner surface of the thigh, inner side of leg and foot, and great toe.

**Sacral Plexus.**—Inability to extend the hip, to rotate the limb, to flex the knee, and to move the foot. The separate nerves are more frequently affected than the whole plexus—*e.g.* :

**Superior Gluteal Nerve** supplies the gluteus medius and minimus. Lesion causes imperfect abduction of thigh.

**Inferior Gluteal Nerve** supplies the gluteus maximus. Lesion causes difficulty in rising from a sitting posture.

**Sciatic Nerve**, the continuation of the main part of the sacral plexus, supplies the muscles at the back of the thigh, and by the branches into which it divides innervates all the muscles below the knee and the greater part of the integument of the leg and foot. In the pelvis the nerve may be injured by tumours, by instrumental delivery in a narrow pelvis, or even by the pressure of the fœtal head. In the thigh the nerve may suffer in fractures, dislocations, wounds, by pressure, or by exposure to cold. Rheumatic and other morbid blood states may also cause inflammation of the nerve. Lesions of the nerve trunk cause inability to extend the thigh, to flex the knee, or to make use of the muscles below the knee.

**Sciatica** is the name given to a painful condition of the sciatic nerve and its branches. This may in some cases be a form of neuralgia in which there may be no demonstrable lesion of the nerve, but it is in all probability in most cases a neuritis due to rheumatism, exposure, or some other source of irritation. It occurs most commonly in adult males about middle age.

*Symptoms.*—Pain of a boring or burning character in the nerve trunk or its branches, intensified by movement, and felt chiefly in the back of the thigh, but also in the gluteal region, in the calf, in the outer and plantar surfaces of the foot. Stretching the nerve (as by flexion of the hip) gives pain, and there is tenderness on pressing over the nerve, especially at the sciatic notch, behind the great trochanter, over the middle of the femur behind, and behind the head of the fibula. Sometimes there is anæsthesia with tingling or other abnormal sensations, while muscular atrophy, weakness, and perhaps electrical changes, may be present.

**External Popliteal (Peroneal) Nerve**, a branch of the sciatic, supplies the peronei and extensor muscles of the foot, the skin in front of the leg, and that of the dorsum of the foot, also some articular branches to the knee, ankle, and foot. This nerve is commonly affected in intrapelvic injury. It is specially vulnerable in certain forms of neuritis, particularly in that of alcoholism, also in the neuritis of diphtheria, diabetes, beri-beri, etc. Its exposed course below the head of the fibula renders it especially liable to injury.

*Symptoms.*—Inability to flex the ankle, to extend the first phalanges on the dorsum of the foot, or to raise the outer border of the foot. There is the characteristic **steppage gait** on walking. On raising the foot from the ground it hangs downward (**foot-drop**). In bed the foot lies extended. By the unopposed action of the healthy muscles talipes equinus results. The muscles atrophy. Sensation is lost on the outer half of the front of the leg, and on the dorsum of the foot.

**Internal Popliteal Nerve** supplies all the muscles of the back of the leg and sole of the foot, the skin of the sole and that of the lower half of the back of the leg. The lesion is commonly part of a toxic neuritis.

*Symptoms.*—Inability to extend the foot and flex the toes. Talipes calcaneus may eventually develop from imperfect muscular opposition, and this, with atrophy of the muscles and secondary contractions, may produce the claw-like foot resembling the similar condition in the hand. Anæsthesia in the skin areas supplied.

The tables on pp. 234-238 give a comparison of the flaccid forms of paralysis and their prominent symptoms.

## II. The Distribution of the Paralysis.

**A. Hemiplegia.**—The loss of power may be confined to one side of the body. This can rarely be due to disease of the spinal cord, where the nerve fibres destined for each side of the body cannot readily be discriminated by the disease. One-sided paralysis is therefore commonly of intracranial or of neural origin. When due to cerebral disease, it is termed **hemiplegia**; if confined to one limb, or to the side of the face alone, the term **monoplegia** is used.

Hemiplegia may be caused by disease in the following situations :

**Lesions of the Cerebral Cortex.**—The damage to produce this result must be of such a nature as will inhibit or destroy the psycho-motor function of those portions of the cortex which exercise controlling influence over movements. The majority of lesions here, however, have the contrary effect—that is, irritation, which produces excessive muscular action or **spasm** (see p. 249). Irritative lesions at times become destructive, in which case spasms or convulsions would be followed by paralysis. It is almost exclusively the Rolandic or sensori-motor area which presides over bodily movements. Destructive lesions in this region give rise to paralysis of those groups of muscles whose cortical centre is injured. The position of these cortical centres is shown on Figs. 30 and 31, pp. 206 and 207.

The lesion may be caused by external injury (pressure of fractured cranial bone or of effused blood), tumours of the brain or skull (syphilitic, tubercular, gliomatous, or malignant), hæmorrhage from diseased bloodvessels into the substance of the cortex, embolism, or thrombosis. The vascular causes just mentioned are less likely to occur here than in the deeper structures. Inflammatory lesions may rarely cause paralysis, as in the last stages of meningitis or abscess. The latter, however, is less likely to occur in the motor area than in the temporal lobe, where it only causes motor symptoms when it exerts sufficient pressure upon the adjacent motor area. Lastly, an infantile form of hemiplegia may occur due to atrophy of the cortex or to a cystic degeneration (porencephalus).

Lesions of the cortex, other than of the Rolandic area, rarely produce motor disturbances, unless some portion of the motor tract is likewise involved. The exceptions to this statement are few, and without diagnostic interest.

The diagnosis of destructive lesions of the Rolandic area is admirably summarized by Ferrier as follows :

1. Destructive lesions of the Rolandic area cause paralysis of voluntary motion of the opposite side, general or limited, according to the extent and locality of the lesion. The electrical reactions of the paralysed muscles are not affected.
2. When the whole motor cortex is involved, the hemiplegia in all respects resembles that caused by destruction of the motor division of the internal capsule.
3. Frequently paralysis of cortical origin is dissociated or consists in a succession of dissociated paralysees or monoplegias. Thus a monoplegia may become a hemiplegia by advance of the lesion from its primary seat.
4. Paralysis of voluntary motion of the leg alone, or of the arm and leg, or of certain movements of the arm, or of the arm and face, or of the face alone, may be looked on as dependent upon lesions of the cortex of the Rolandic zone or of the subjacent medullary fibres.
5. Hemiplegia from cortical lesion of the left hemisphere is almost invariably associated with aphasia.
6. Crural monoplegia, or this combined with a greater or less degree of paralysis also of the arm, indicates lesion at the upper extremity of the central convolutions.
7. Brachial monoplegia indicates lesion of the middle two-fourths of the Rolandic area.
8. Brachio-facial monoplegia indicates lesion of the lower third of the Rolandic area.
9. Facial monoplegia (or this combined with aphasia, if the lesion is in the left hemisphere) indicates lesion of the lower extremity of the Rolandic zone posterior to the third frontal.
10. While it is probable that the centres of movement of the vocal cords and of the muscles of mastication are also situated at the lower extremity of the Rolandic area, there is as yet no case on record in which these muscles have been paralysed by cortical lesions, unilateral or bilateral.
11. Paralysis of cortical origin may be, and frequently is, entirely independent of impairment of cutaneous or muscular sensibility. . . .
12. When the motor cortex is destroyed, secondary sclerosis ensues in the pyramidal tracts, and contracture in the paralysed limbs.

## ATROPHIC PARALYSES OF

Causes and Symptoms.	Infantile Paralysis.	Amyotrophic Lateral Sclerosis.
1. Age .. ..	Children, mostly under three years (occasionally adults)	Adults mostly between twenty-five and fifty
2. Sex .. ..	The majority boys	The majority females
3. Heredity .. ..	Nil	Unimportant
4. History and other causal factors	Occurs most frequently in warm weather. Probably infective in origin	Is sometimes preceded by injuries, exposure, etc., of doubtful causal influence
5. Mode of onset ..	Sudden; feverish attack	Gradual
6. Distribution of the atrophied muscles, and the order in which they are attacked	Often asymmetrical; physiological groups of muscles affected; upper or lower limbs alone, or only one, or all four limbs attacked. Trunk muscles are rarely involved	First hands and arms rigid, later atrophied, or <i>vice versa</i> ; legs afterwards spastic, but rarely atrophied; lastly, bulbar paralysis supervenes
7. Muscles which usually escape	Face muscles still more rarely involved; bladder and rectum unaffected	Lower limbs usually escape
8. Electrical reaction	Partial or complete reaction of degeneration	Excitability diminished
9. Resulting defects in movement of the affected part	Paralysis of the affected groups of muscles	Loss of power and stiffness. Fingers and wrists flexed, forearm pronated, elbow semi flexed; sometimes claw-hand
10. Fibrillary twitching	Usually absent	Present
11. Sensory and vasomotor disorders	Nil	Often pain and tingling in early stages; no anaesthesia
12. Disturbances of reflexes	Diminished or lost	Increased in arms and legs; jaw-jerk present
13. Contractures and deformities	Talipes equinus, claw-hand, partial dislocations, etc.	Flexed fingers, wrists, and elbows; claw-hand
14. Bladder and rectum	Unaffected	Unaffected
15. Course of the disease and prognosis	Partial recovery inside a few weeks in most cases, after which the paralysis is permanent	Progressive and of long duration; ultimate prognosis bad
16. Degree of atrophy	Extreme	Extreme
17. Trophic changes (in addition to atrophy)	Nil	Nil
18. Inco-ordination ..	Nil	Nil
19. Other symptoms ..	Unimportant	Mental symptoms are melancholy and emotional

## CEREBRO-SPINAL ORIGIN

Progressive Muscular Atrophy.	Bulbar Paralysis.	Syringomyelia.
Young adults	In middle and advanced life	Often congenital; may first appear in adult life
The majority males	The majority males	The majority males
Nil	Nil	Unimportant
Same as amyotrophic lateral sclerosis	Same as amyotrophic lateral sclerosis	Nil
Gradual	Gradual	Gradual
First the small hand muscles; then forearm flexors, extensors; deltoid and other scapular muscles; biceps; lower half trapezius; upper half pectoralis major. Sometimes commences in shoulder. Usually bilateral	First the tongue, then lips, soft palate, pharynx, larynx, muscles of mastication. Usually bilateral	Hand and arm muscles resemble those of progressive muscular atrophy; back muscles often affected; legs may be spastic
Remaining halves of pectoralis major and trapezius until late in disease; legs often escape	In uncomplicated bulbar paralysis the rest of the body escapes	Legs usually escape
Partial reaction of degeneration	Partial reaction of degeneration	Partial reaction of degeneration in advanced cases
Awkwardness in using the affected limb; cannot well oppose thumb or move fingers laterally when hand is laid flat	Articulation (dentals and linguals) indistinct; swallowing difficult; food regurgitates through nose; voice monotonous; coughing imperfect	Weakness and paralysis of the affected muscles; antero-posterior and lateral spinal curvatures
Present	Present	Present
Tactile sensibility diminished or lost	Tactile sensibility diminished or lost	Tactile sense normal; heat and pain senses lost
Diminished or lost	Diminished or lost	Diminished in arms; increased in legs
Claw-hand	Atrophied tongue	Claw-hand; spinal curvatures
Unaffected	Unaffected	Usually unaffected
Very slow; ultimately fatal	Slow; ultimately fatal	Very chronic; well-marked cases ultimately fatal
Extreme	Extreme	Extreme
Nil	Nil	Charcot's joint in 10 per cent. of cases; Morvan's disease
Nil	Nil	Sometimes present
Bulbar symptoms sometimes supervene	Rarely an acute form occurs with sudden onset (apoplectiform bulbar paralysis)	Bulbar symptoms sometimes supervene

## ATROPHIC PARALYSES

Causes and Symptoms.	Landry's Paralysis.	Progressive Neural Muscular Atrophy (Peroneal Type).
1. <b>Age</b> .. ..	Adults under middle age	Children or adolescents
2. <b>Sex</b> .. ..	The majority males	Either sex
3. <b>Heredity</b> .. ..	Nil	Well-marked
4. <b>History and other causal factors</b>	Has been attributed to exposure, alcoholism, syphilis, and the effects of acute diseases	Sometimes follows an acute specific disease, especially measles
5. <b>Mode of onset</b> ..	Gradual but rapid advance	Gradual progress
6. <b>Distribution of the atrophic muscles, in the order in which they are attacked</b>	Begins in legs; ascends rapidly to thighs, trunk, and arms. Symmetrical	Commences in feet and peroneal group of muscles; after a time (perhaps years) attacks hands and arms. Symmetrical. Rarely begins in arms
7. <b>Muscles which usually escape</b>	The muscles supplied by the cranial nerves as a rule	Nil
8. <b>Electrical reaction</b>	Partial reaction of degeneration if patient survive	Diminished excitability
9. <b>Resulting defects in movement of the affected part</b>	Weakness, and later paralysis of the affected regions	Difficulty in walking
10. <b>Fibrillary twitching</b>	Absent	Present
11. <b>Sensory and vasomotor disorders</b>	Anæsthesia; paræsthesia; or perhaps sensation normal	Pains in limbs; tactile sensibility impaired
12. <b>Disturbances of reflexes</b>	Diminished or lost	Diminished or lost
13. <b>Contractures and deformities</b>	Nil	Double talipes equinus or equino-varus
14. <b>Bladder and rectum</b>	Unaffected	Unaffected
15. <b>Course of the disease and prognosis</b>	Rapidly progressing; 60 per cent. are fatal within three weeks	Very slow; prognosis bad
16. <b>Degree of atrophy</b>	Slight; absent in very rapid cases	Extreme at periphery
17. <b>Trophic changes (in addition to atrophy)</b>	Nil	Nil
18. <b>Inco-ordination</b>	Nil	Sometimes
19. <b>Other symptoms</b>	Nil	Nil



## OF NEURAL ORIGIN

Peripheral Neuritis.	Erb's Paralysis (Upper-Arm Type).	Klumpke's Paralysis (Lower-Arm Type).
Varies with cause	Often in children (injury at birth); adults (pressure)	Any age
Either sex	Either sex	Either sex
Nil	Nil	Nil
Toxic blood states: alcoholism; arsenic, lead, mercury; infections, especially diphtheria, diabetes, etc.	Pressure of a load on shoulder injures fifth and sixth cervical nerves; injury during parturition	Pressure injures eighth cervical and first dorsal nerves; perhaps sixth and seventh cervicals (tumours, etc.).
Commonly gradual	Rapid	Rapid
Bilateral, Peroneal group in alcoholic and arsenical forms; palate, oculo-motors, ciliaries, peroneals, etc., in diphtheritic; extensors of hand in plumbism	Deltoid, biceps, brachialis anticus, supinator longus; sometimes also supra- and infraspinati and supinator brevis	Small muscles of hand; flexors of fingers; sometimes (sixth and seventh cervicals) extensors of fingers, triceps, pronators, and flexors of wrist
Lumbricales and interossei in the wrist-drop of lead palsy	All except above named	All except above named
Partial or complete reaction of degeneration	Partial or complete reaction of degeneration	Partial or complete reaction of degeneration
'Steppage gait'; squint; loss of accommodation; nasal regurgitation of solids and fluids; dyspnoea	Imperfect abduction of arm, flexion of elbow, supination, rotation outward of humerus	Weakness or paralysis of above muscles. Pupil may be contracted and palpebral fissure diminished in size (lesion of sympathetic)
Sometimes present, or tremor	Absent	Absent
Muscular tenderness; pain along nerves; anaesthesia, etc.	Pain; perhaps anaesthesia (circumflex, musculo-cutaneous)	Pain; anaesthesia; hyperaesthesia
Diminished or lost	Absent	Absent
Talipes equinus; wrist-drop	Unimportant	Claw-hand
Unaffected	Unaffected	Unaffected
Recovery usual on removal of cause	Slow. Prognosis uncertain; paralysis often permanent	Slow. Prognosis uncertain
Considerable	Often considerable	Often considerable
Herpes zoster; glossy skin	Usually none	Usually none
Rarely found	Nil	Nil
Tremor; cutaneous pigmentation	Nil	Nil

## ATROPHIC PARALYSES OF MUSCULAR ORIGIN

Causes and Symptoms.	Pseudo-Hypertrophic Paralysis.	Erb's Juvenile Form (of Muscular Dystrophy).	Facio-Scapulo-Humeral Form (of Muscular Dystrophy).
1. Age .. ..	Childhood	About puberty	Usually in infancy; sometimes in youth and adult ages
2. Sex .. ..	The majority boys	Either sex	Either sex
3. Heredity ..	Yes; transmitted via females	An important factor	An important factor
4. History and other causal factors	Nil	Nil	Nil
5. Mode of onset ..	Gradual	Gradual	Gradual
6. Distribution of the atrophied muscles, and the order in which they are attacked	First weakness and hypertrophy of calf muscles; then glutei, lumbar, and other trunk muscles; next muscles of shoulder and upper arm. Symmetrical	Arm muscles earlier atrophied than leg; trapezius, serratus magnus, pectoralis major; forearm only slightly; glutei, flexors of hip, extensors of knee, peronei. Symmetrical	Atrophy of orbicularis oris and palpebrarum, levator anguli oris; shoulder and arm muscles next; legs as in the juvenile form. Symmetrical
7. Muscles which usually escape	Those of hand, forearm, and neck	Hand muscles; deltoid, spinati, subscapularis; all below knee except peronei; face and neck muscles	Flexors of wrist and fingers; oculomotors; tongue; pharynx
8. Electrical reaction	Diminished excitability in advanced stages; no reaction of degeneration	Diminished excitability in advanced stages; no reaction of degeneration	Diminished excitability in advanced stages; no reaction of degeneration
9. Resulting defects in movement of the affected part	'Waddling gait'; rises from floor by 'climbing up his legs'	Weakness of the affected muscles; lordosis or other spinal curvature	Face mask-like, or with sad expression; spinal curvatures
10. Fibrillary twitching	Absent	Absent	Absent
11. Sensory and vasomotor disorders	Nil	Nil	Nil
12. Disturbances of reflexes	Diminished in advanced stages	Diminished in advanced stages	Diminished in advanced stages
13. Contractures and deformities	Lordosis	Lordosis on standing, kyphosis on sitting	Lordosis and kyphosis
14. Bladder and rectum	Nil	Nil	Nil
15. Course of the disease and prognosis	Slowly progressive to fatal termination, which usually occurs about puberty	Progressive; much slower than pseudo-hypertrophic paralysis	Slow; ultimately fatal
16. Degree of atrophy	Apparent hypertrophy (fat)	Considerable	Considerable
17. Trophic changes (in addition to atrophy)	Nil	Nil	Nil
18. Inco-ordination	Nil	Nil	Nil
19. Other symptoms	Nil	Nil	Nil

Paralytic symptoms produced by destructive lesions of the **Centrum Ovale** are practically indistinguishable from those due to cortical lesions. The nearer the lesion lies to the cortex the greater is the probability of a monoplegia resulting. On the other hand, even a small lesion situated near the entrance of the convergent Rolandic fibres into the internal capsule, and still more if it invade the internal capsule, will probably interfere with the impulses destined for more than one limb.

**Lesions of the Internal Capsule** are the commonest cause of hemiplegia, and are very frequently the result of hæmorrhage from ruptured lenticulo-striate branches of the middle cerebral artery. This accident will also probably injure the lenticular and caudate nuclei, and possibly also the optic thalamus. Paralytic symptoms referable to lesions of these ganglia cannot be distinguished.

Emboli are a not infrequent cause of lesion in this region, the hemiplegia of cardiac disease being commonly of this origin. Thrombosis also occurs, while abscess and tumours may furnish a small proportion of the cases. (See Fig. 29, A, p. 205.)

**Crura Cerebri.**—Hæmorrhage and thrombosis are here less frequently found. Tumours of the base of the skull or meningitis may give rise to paralytic symptoms from pressure on the crus. Abscess may be an extension from the pons or higher parts of the cerebrum.

Pressure on the crus usually damages the third nerve nucleus, or the third or fourth nerve trunks may be compressed, causing oculo-motor paralysis of the same side. At the same time the pyramidal fibres in the crus proceeding from the internal capsule to the periphery are injured, causing hemiplegia of the opposite side. Possibly also the fibres from the cortex to the opposite facial nucleus may share in this lesion, giving rise to a central or supra-nuclear facial paralysis, which will also, of course, be on the opposite side, and will affect chiefly the lower facial muscles. This form of **crossed paralysis**, then (limbs of one side with eye muscles of opposite), indicates lesion of one crus. (See Fig. 29, B, p. 205.) A tumour in this region is very likely to affect the other crus as well, producing double hemiplegia, which is sometimes termed **cerebral paraplegia**, or **diplegia**.

**Corpora Quadrigemina.**—Lesions of the corpora quadrigemina are rarely separate from those of the neighbouring structure. Damage to the subjacent oculo-motor nucleus may be

the source of paralytic symptoms. The diagnosis would depend on loss of power in the eye muscles, internal probably as well as external muscles (ophthalmoplegia interna and externa), together with a reeling gait and possibly deafness.

**Pons Varolii.**—Hæmorrhage is fairly common in this situation, and thrombosis from syphilitic or atheromatous degeneration of the arteries occurs here frequently, producing softening. Abscess of the pons is not often seen, and tumours situated in its substance are uncommon. The pressure of tumours from neighbouring regions (cerebellum, or base of the skull) is a more usual cause of paralysis.

Damage to the pontine tissue is a formidable lesion, and is chiefly shown by loss of power on one or both sides of the body. The motor tract rarely escapes when lesions occur here.

A one-sided lesion in the pons will injure, not only the fibres from the motor area of the same side, but especially if occurring in the lower half of the pons, also probably one or more of the motor nerve nuclei situated there—namely, those of the fifth, sixth, or seventh nerves on the side of the lesion. The result of a destructive lesion of this nature will obviously be paralysis of the limbs of the opposite side, together with paralysis of the muscles supplied by one or more of these cranial nerves on the side of the lesion—viz., the muscles of mastication, the external rectus of the eye—viz., the muscles of mastication, the external rectus of the eye, and the facial muscles. This form of **alternate or crossed paralysis** is characteristic of lesions of the pons. (See Fig. 29, C, p. 205.)

The paralysis must vary in its distribution according to the situation of the lesion in the pons. If it lie in the upper part of the pons, the fifth, sixth, and seventh nuclei may escape, and the symptoms will be those of a lesion of the internal capsule. Although the cranial nerve nuclei may have escaped, the supra-nuclear fibres, destined for the nerve nuclei in the opposite half of the pons, may be injured before decussation, along with those proceeding to the descending columns of the cord. In this latter case the hemiplegia will be restricted to the side opposite to the lesion, and may exhibit one or more of the following palsies: convergent squint (sixth nerve); displacement of lower jaw, when in action, toward the paralysed side, owing to the unopposed action of the external pterygoid muscle on the sound side (motor division of fifth nerve); paralysis of the muscles of the lower half of one side of the face, with slight diminution of power of the upper

facial muscles of the same side (supranuclear fibres of seventh nerve); and paralysis of the arm or leg (pyramidal fibres).

The following table compares the general symptoms of cerebral hæmorrhage, thrombosis, and embolism :

Symptoms.	Hæmorrhage.	Thrombosis.	Embolism.
<b>Onset</b> .. ..	Sudden	Perhaps some premonitory symptoms	Sudden
<b>Symptoms of pressure :</b>			
<b>Consciousness</b> .. ..	Usually lost	Often retained	Often retained
<b>Pulse</b> .. ..	At first slow and irregular	Unaffected	Unaffected
<b>Respiration</b> .. ..	Slow. Cheyne-Stokes in fatal cases	Unaffected	Unaffected
<b>Temperature</b> .. ..	At first falls; later it rises	Unaffected	Unaffected
<b>Heart</b> .. ..	May be hypertrophied	Unaffected	Recent endocarditis in 90 per cent. of the cases
<b>Age</b> .. ..	Elderly	Any age	Young
<b>Commonest site of lesion</b> .. ..	Right or left middle cerebral artery	Any intracranial artery	Left middle cerebral artery
<b>Commonest cause</b>	Arterio-sclerosis and high arterial tension	Syphilis	Endocarditis

**Conjugate Deviation.**—A deviation of both eyes to either side—the visual axes remaining parallel—may occur when the lesion is pontine, involving the nucleus of the sixth nerve. Association fibres from the sixth to the opposite third nucleus are, at the same time, damaged, with the result that there is paralysis of the external rectus on the side of the lesion, and of the internal rectus of the opposite side. The eyes look away from the lesion. Lesions above the pons, interrupting the fibres before decussation, which proceed from the cortical association centres to the nerve centres in the pons, may produce conjugate deviation in the opposite direction—the eyes look to the lesion. The post-frontal region of the cortex is most constantly associated with conjugate movements, but they are also observed in lesions of the parietal and occipital lobes. The supranuclear lesion is temporary, owing to bilateral innervation. It is not, however, only in paralytic conditions that conjugate deviation occurs. The opposite condition, or irritation, which is so common a result of cortical lesions (see

below, p. 252), may so stimulate the lower centre for conjugate movements (the opposite sixth nerve nucleus) as to deviate the eyes towards the sound side—although the lesion is above the pons, the eyes look away from the lesion. (See Fig. 38.)

Lesions of the **Medulla Oblongata** causing paralysis are usually associated with injury to the pons and cerebellum. Owing to the vital importance of the centres situated here, serious injury of which is incompatible with life, lesions of the medulla usually terminate fatally without delay.

There may, however, occur lesions of a less destructive nature which pick out the nerve nuclei situated in those regions, and

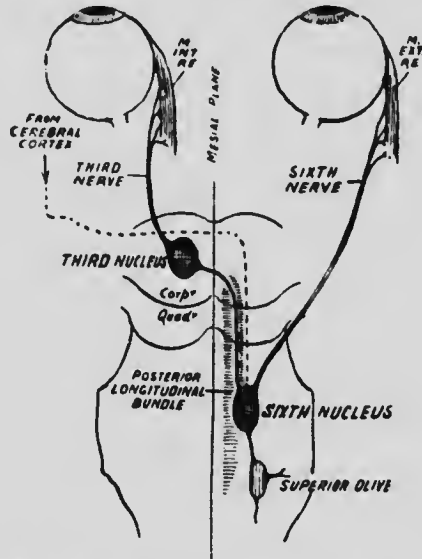


FIG. 38.—MECHANISM FOR THE CONJUGATE MOVEMENT OF BOTH EYEBALLS TOWARDS THE RIGHT. (MONRO.)

give rise to paralytic symptoms lasting for a considerable time before death ensues. These are chiefly the chronic inflammatory or degenerative changes which, occurring in the anterior cornua of the spinal cord, cause progressive muscular atrophy; in the medulla and pons, bulbar paralysis; in the pons and crura, ophthalmoplegia. In these affections the paralysis is more frequently bilateral than hemiplegic.

Finally, diffuse inflammatory or degenerative scleroses of the brain may give rise to paralysis, often bilateral, of which multiple sclerosis, general paralysis, and meningitis are examples.

It rarely happens that disease or injury of the **Spinal Cord** produces a one-sided paralysis. This may, however, be the result of a tumour, of syphilis, of tuberculosis, or of an injury, especially a stab. The combination of symptoms produced by such a lesion is termed the **syndrome of Brown-Séguard**, and consists, shortly, in paralysis of the same side of the body below the level of the lesion. The reflex action in the same area is at first lessened, and later increased. The sensation at the level of the lesion and on the same side is diminished or lost, while at a lower level it is increased. On the opposite side, below the level of the lesion, there is loss of sensibility.

B. **Paraplegia**.—More commonly paralysis resulting from lesions of the spinal cord affects both sides of the body, when it is termed paraplegia. If the disease or injury of the cord involves only a short length, the muscles supplied by the nerves arising from the damaged segments of the cord may soon become flaccid and wasted; but the muscles connected with the segments below the lesion, influenced by their uninjured nutritional and reflex centres in the cord, retain a certain automatic contractility, and do not rapidly waste, although the power of voluntary movement is lost in them. Such lesions, therefore, commonly produce a spastic paralysis.

When the paralysis affects the legs alone (*paraplegia cruralis*), it is usually due to transverse lesions of the dorsal or lumbar cord, while paralysis of all four limbs (*paraplegia totalis*, or *paraplegia brachialis*), more rarely met with, is the result of lesion high in the cord, including or above the cervical enlargement. The exceptions to the statement that paralysis of both sides of the body is due to the spinal lesions are:

(a) Cerebral hæmorrhages, embolic or thrombotic softening, and tumours, chiefly pontine, which interrupt both motor tracts. The condition is termed 'cerebral paraplegia,' or 'diplegia.'

(b) General inflammatory or degenerative cerebral changes, as found in the late stages of locomotor ataxia and general paresis, and of hydrocephalus and tuberculous meningitis.

(c) Acute and, less rarely, chronic inflammation and degeneration of the motor nuclei in the medulla (bulbar or glosso-labio-laryngeal paralysis), pons and crura (ophthalmoplegia), or a bilateral destructive lesion of the cortex or internal capsule, involving the fibres proceeding to those nuclei (pseudo-bulbar paralysis).

(d) Birth palsies, the spastic paralyses of infancy, or Little's disease—a spastic diplegia found at, or shortly after, birth, affecting chiefly the arms, but it may be the legs or one limb which is the seat of the paralysis. In addition to the loss of muscular power, there may be spasm, with irregular, incoordinated, and involuntary movements. Varieties of this condition are termed **spastic chorea** and **bilateral athetosis**. Children suffering from these affections are frequently idiotic, and convulsions may occur. The morbid anatomy is atrophy, cystic degeneration (porencephalus), or other destructive lesion of the brain, followed in many cases by descending sclerosis, and due in most instances to meningeal lacerations, hæmorrhages, or pressure, resulting from difficult labour.

(e) Neuritis and other affections of the peripheral nerves.

(f) Muscular dystrophies. (See Trophic Disturbances, p. 491.)

(g) Hysteria.

The diminution or loss of power may be the result of disease of the **Muscles** themselves. This, however, is of infrequent occurrence—*e.g.*, Myositis (inflammation of the muscles); myalgia (muscular rheumatism); and the muscular dystrophies, *viz.*, pseudo-hypertrophic paralysis, Erb's juvenile form, the facio-scapulo-humeral form (see p. 238).

The loss of power may be due to affections involving the **Bones, Ligaments, and Other Tissues in the Neighbourhood of Joints**—*viz.*: Rheumatism, acute and chronic; osteo-arthritis (arthritis deformans, rheumatoid arthritis); gout; trophic affections of the joints; tumours, inflammations, injuries, and other surgical affections.

#### Summary of the Conditions showing Decreased Movement.

Weakened or abolished movement of any part of the body is due to lesion of (I.) the central or peripheral nervous system; (II.) the muscles; and (III.) the bones and other structures entering into the formation of, and adjacent to, the joints.

I. In cases of nervous origin one investigates (i.) the condition or tone of the affected muscles, and (ii.) the distribution of the symptom—*i.e.*, the situation of the paralysed muscles.

(i.) **Condition:** (a) Spastic or tonic paralysis; (b) flaccid or atrophic paralysis.

(a) Spastic paralysis is found in (A) diseases of the spinal cord



(transverse interruption of the cord from compression, myelitis or hæmorrhage, sclerotic changes in the upper segment of the motor tract, viz., primary lateral sclerosis, amyotrophic lateral sclerosis, multiple sclerosis, hereditary spastic paraplegia, ataxic paraplegia, syringomyelia, combined sclerosis of Dana, pellagra, etc.). (B) Diseases of the brain (hæmorrhage, tumours, inflammations, degenerations). (C) Functional nervous affections (hysteria, neurasthenia, 'railway spine').

(b) Flaccid paralysis occurs in (A) disease of the brain and spinal cord involving the peripheral neuron (progressive muscular atrophy, bulbar paralysis, ophthalmoplegia, infantile paralysis, Landry's paralysis, cerebral hæmorrhages, tumours, etc., involving the motor nuclei). (B) Disease of the peripheral nerves, either singly or in groups.

(ii.) **Distribution:** (a) Hemiplegia; (b) paraplegia.

(a) Hemiplegia is usually the result of (1) cerebral lesions: Hæmorrhage, tumours, etc., implicating the cortex, internal capsule, crura, pons, or medulla. (2) Spinal cord (rarely). (3) Lesions of the peripheral nerves frequently cause one-sided paralysis, to which the term 'hemiplegia' is not usually applied.

(b) Bilateral paralysis: Paraplegia; diplegia.

II. Disease of the muscles may be the cause of decreased motility: Myositis, myalgia (muscular rheumatism), myositis ossificans, muscular dystrophies.

III. Disease of the bones and joints: Various surgical affections, acute and chronic rheumatism, gout, osteo-arthritis, trophic joint affections (Charcot's joint).

### MOVEMENT DISORDERLY (Inco-ordination or Ataxia).

Reflex character of co-ordination—Innervation sense—Examination of a patient suffering from ataxia—Romberg's sign, or static ataxia.

Affections characterized by inco-ordination: (1) Lesions of the posterior columns of the cord; (2) lesions of the peripheral nerves; (3) cerebral lesions; (4) cerebellar lesions; (5) lesions of the semicircular canals.

The free and accurately adjusted movements of the body depend, not only on the efficient contractility of the respective muscles, but also on the perfectly combined action of the different groups of muscles. An effective regulation of the mutually supporting and opposing muscular contractions results

in perfect **co-ordination** of the movements of the part. On the other hand, if the controlling influence is insufficient to ensure this mutual and combined action, disorderly movements ensue, the result being **inco-ordination** or **ataxia**.

The mechanism of co-ordination is probably of the nature of a **reflex action** (see Reflexes, p. 334), of which the central portion is situated in the motor cortex of the brain. Afferent impulses convey impressions from the periphery, the most important for this purpose being those which inform the higher centres of the condition and position of the various parts of the body in relation to surrounding objects. This is accomplished by impulses transmitted from sensory nerve terminations in muscles, tendons, and joints, and to a less extent in skin as well. As a result of these stimuli reaching the motor centres, the latter are impelled to emit the amount of energy necessary to maintain the limb in the desired position. The estimate formed by the 'sensorium' of the amount of energy expended in the act is the chief source of information as to the position of the various parts of the body. It is, indeed, this estimate of the amount of nerve energy expended in any muscular action which constitutes the **innervation sense** (or **strength sense**). The so-called **muscular sense** is merely a modification of the innervation sense. Additional factors in the maintenance of co-ordination are the visual impulses passing from the retina to the sensorium via the optic thalamus, and the auditory impressions from the labyrinth through the vestibular portion of the auditory nerve to the cerebellum. In addition, Sherrington has shown that the reciprocal action of antagonistic muscles, locally excited, is an important factor in the production of reflex and co-ordinated acts.

In order to test a patient's power of co-ordination, he is directed to perform some of the ordinary movements of the part. In the case of the lower limb, the act of walking readily betrays inco-ordination. In the typical form seen in locomotor ataxia the gait resembles somewhat that of a child learning to walk, or that of an inexperienced landsman in walking on the decks of a ship at sea: the foot is brought down in an uncertain manner, as if the patient were doubtful of the level of the floor; he raises his foot unnecessarily high, and brings it down in a forcible, stamping manner; he finds it impossible to walk in a perfectly straight line—for example, he cannot confine his footsteps to one board in a wooden floor; he can only with difficulty touch a desired spot on

the floor with his toe; the uncertain movements of his legs are best marked when he attempts to turn. If he is deprived of the assistance of his sight, as by walking in the dark or with his eyes closed, his difficulties are vastly increased. Not only his movements, but the power of maintaining his balance may be impaired (static ataxia). Even in the early and ill-marked conditions of inco-ordination he may be unable to stand upright with his heels together and his eyes closed (**Romberg's sign**). If he is in the recumbent position, he is directed to touch some spot with the limb under observation; thus, he should attempt to touch one patella with the opposite heel or toe, or he may place his foot on a marked spot on his bed. Movements of the hands and arms are to be similarly tested, by requesting him to touch some indicated spot on his body or elsewhere with his eyes open. And, again, when they are closed; or he may attempt some more delicate action, such as picking up a pin or threading a needle, which he may be unable to accomplish or may perform in a clumsy manner.

In some conditions the inco-ordination is shown by a reeling or staggering, rather than by an ataxic, gait, while in other cases it is difficult to decide whether we are dealing with ataxia or with loss of power of one or more muscles. (See Gait, p. 148, and Vertigo, p. 539.)

The conditions in which ataxia may be observed can be enumerated in the five following groups:

1. Disease or injury of the posterior columns of the spinal cord. This portion of the cord is practically a continuation centrewards of the posterior nerve-roots, through which the sensory impulses reach the cortical centres; interruption to the passage of the latter (with or without interruption to the passage of sensory impulse from the skin) prevents a correct estimate being formed as to the position and movements of the affected region in the periphery. Hence inco-ordination is a feature in locomotor ataxia, Friedreich's ataxia, and general paralysis of the insane.
2. Lesions of the peripheral nerves, especially polyneuritis. It is open to question if we are here dealing with a true inco-ordination, and, indeed, the condition is by some writers described as **pseudo-ataxia**, in the belief that the irregular muscular contractions are mainly due to a loss of muscular contractility of greater or less degree and extent. With the loss of power there is, however, a loss of sensory impressions which must of necessity interfere

with the innervation sense. In the case of the lower limb, the resulting defective movements bear a fairly close resemblance to those consequent upon disease of the posterior columns of the cord. The gait in cases of diphtheritic, alcoholic, diabetic, and other forms of neuritis has a certain stamping, ataxic character, but the loss of muscular power is seen in the dragging toes, necessitating an excessive elevation of the knees in order that the toes may clear the ground. This constitutes the so-called **steppage gait**.

3. Certain cerebral lesions, and especially those involving the parietal lobe, give rise to a form of inco-ordination. The finer and more delicate movements are clumsily executed, and the loss of muscular sense is well marked.

4. Tumour, atrophy, or other lesion of the cerebellum, gives rise to a reeling or staggering gait, resembling that of alcoholic intoxication, with inability to preserve the balance of the body when standing (**vertigo**). It is frequently found that the ataxia due to cerebellar disease disappears when the patient assumes the recumbent position.

5. Disease of the semicircular canals, resulting in increased intralabyrinthine pressure, or of other portions of the vestibular nerve, causes a similar form of ataxia to that consequent upon cerebellar disease. There is a paroxysmal vertigo, accompanied by noises, faintness, nausea, etc. (Ménière's disease). These symptoms are sometimes produced by inflammatory or other diseases of the middle ear, or by the pressure of wax in the external auditory canal. Long-continued or forcible syringing the ear may cause a somewhat similar series of symptoms. (See *Vertigo*, p. 539.)

**Summary.** — The co-ordination of muscular contractions depends upon the integrity of reflex arcs, whose afferent limbs convey impulses from muscles, tendons, joints, the retina, and the semicircular canals.

Inco-ordination is shown by a disorderly or clumsy action of the affected limb. Static ataxia is the inability to preserve the balance while standing.

Inco-ordination occurs in :

1. Lesions of the posterior columns of the cord : Locomotor ataxia, Friedreich's ataxia, general paresis.
2. Peripheral nerve lesions : Neuritis due to alcohol, lead arsenic, diphtheria, diabetes, etc.

3. Cerebral lesions, especially those involving the parietal lobe: Post-hemiplegic chorea, athetosis, Little's disease, etc.
4. Cerebellar lesions: Tumours, atrophy, abscess, etc.
5. Lesions of the vestibular nerve and semicircular canals: Ménière's disease, middle-ear disease, injury or disease in the external auditory canal causing pressure—*e.g.*, collection of wax, and forcible syringing to remove it.

### MOVEMENT (Muscular Contraction) INCREASED (Spasm).

Spasm, tonic and clonic—Diseases characterized by tonic spasm—  
—Risus sardonicus—Opisthotonos—Emprosthotonos—Pleurosthotonos—Orthotonos—Trousseau's sign—Chvostek's sign—Erb's sign—Retraction of the head—Kernig's sign—Writer's cramp.

Clonic spasms—Fibrillary twitching—Tremor—Rigors—Nystagmus—Convulsions—Summary of the conditions giving rise to spasm.

An involuntary increase in the activity or extent of the muscular contractility over that of a normal subject is due, almost without exception, to disease of the nervous system. The term **Spasm** may be employed to indicate exaggerated muscular action, without necessarily implying the presence of pain or any abnormal sensation.

In the article on Abnormalities of Movement (p. 205) it is indicated that involuntary increase in the movements is the result of increased stimulation or irritation of the nervous structures which preside over the muscular contractions, and is also due to exaggerated reflex conditions, which are probably the result of the removal of inhibiting influences from the motor centres, rather than increased stimulation of those structures. The removal of these inhibiting influences involves the interruption of the motor path, with the result that the power of voluntary movement is impaired; in such cases one finds paralysis combined with spasm. Spasmodic contraction of this reflex nature and the affections in which it may be observed are discussed in the article on Spastic Paralysis (p. 209), and in that on Reflexes (p. 341); in this place are considered mainly those conditions of increased muscular contractility due to excitation of the motor centres.

Spasm occurs either as (A) a continuous, involuntary, tonic contraction, or (B) intermittent or clonic contractions. In either variety there is an irritative lesion of some portion of the motor

tract, as distinguished from the destructive lesions giving rise to paralysis.

A. **Tonic Spasm** is a single continuous contraction of certain muscles or groups of muscles, producing a stiffness and rigidity of the limb or part affected.

The following is a list of the diseases in which one may observe tonic spasm :

**Tetanus**, or **Lock-jaw**, a microbic disease, the symptoms of which appear within a fortnight after infection of a wound. A progressively active contraction of the jaw, neck and face muscles, beginning as a stiffness in the neck and muscles of mastication. The stiffness of the jaw constitutes **lock-jaw**, or **trismus** ; the facial spasm produces the **risus sardonicus** (see p. 353). The body then becomes rigid, and in consequence of excessive contraction of certain groups of muscles it may become curved backwards (**opisthotonos**), forwards (**emprosthotonos**), to one side (**pleurosthotonos**), or the rigid body may remain straight (**orthotonos**).

**Tetany** occurs in children or young adults as a result of peripheral irritation, and may also occur as an epidemic. Tonic spasms, lasting from a few minutes to several hours, cause the following symptoms: Flexion of the hands on the wrists, of the fingers on the metacarpus, with the interphalangeal joints extended, the thumb tucked into the palm of the hand (**accoucheur's hand**) ; the elbow-joint is flexed ; the legs are extended ; the foot is extended on the leg, the toes flexed. In severe cases spasm of many other muscles is found, and general convulsions may occur. Pressure over the nerves or arteries of the affected limb will bring on a recurrence of the spasm, provided the attack is not over (**Trousseau's sign**). A tap over any motor nerve may cause the attached muscle to contract. The facial nerve is particularly sensitive, and when struck (best on or just below the zygoma), the facial muscles contract (**Chvostek's sign**). An increased excitability of the muscles may be observed (**Erb's sign**).

**Spasm of the Glottis** occurs as a result of various irritants (foreign bodies in the larynx, paralysis of the abductors of the cords, laryngitis, œdema of the larynx, hysteria, epilepsy, tetanus, chorea, hydrophobia, rickets, and whooping-cough).

*Symptoms.*—Aphonia, stridor, dyspnœa, asphyxia, violent respiratory efforts.

In children under two years old, and almost invariably the subjects of rickets, spasm of the glottis is known as **laryngismus**

**stridulus (spasmodic croup, child-crowing).** Hoarseness, dyspnoea, an inspiratory crowing sound, and, in severe cases, tetany, are the chief symptoms.

**Strychnine Poisoning** causes tetanic spasms affecting the body and limbs. Opisthotonos is generally marked. The legs are extended, the arms bent, and the hands clenched; the face muscles are contracted into the 'risus sardonicus'; and, lastly, in extreme cases the jaw muscles may be affected (unlike tetanus, where trismus is an early symptom). Between the paroxysms, which are evoked by any trifling disturbance, little or no rigidity may persist.

**Meningitis**, in its varied forms, produces spasms, both tonic and clonic. (1) Cerebro-spinal meningitis exhibits a marked rigidity of the neck and back muscles: the head is drawn backwards till the occiput approaches the shoulder blades, and the body may be in the position of orthotonos or of opisthotonos. (2) Tuberculous meningitis also frequently causes retraction of the head, but clonic or convulsive spasms are here more characteristic. (3) Meningeal infection by pathogenic organisms already active in the body, or introduced by local injury—*e.g.*, typhoid fever, pneumonia, gonorrhœa, malignant endocarditis, influenza, diphtheria, septicæmia, etc. Both tonic and clonic spasms may be observed in any of these affections. As a rule, tonic contraction is more characteristic of basic inflammation, an extreme form of inflammation of the vertex. (4) The non-inflammatory form of meningitis occurring in children under two years of age, described by Gee, Barlow, Lees, and others, may provisionally be considered separately. Tonic contraction of the neck and back muscles is an invariable symptom of the 'posterior-lateral' form, but not of the 'vertical' form. Opisthotonos, while not so frequently observed as head-retraction, is a common symptom, and may be extreme. (5) Serous meningitis, described by Quincke as a non-inflammatory effusion into the ventricles, possibly similar to the angio-neurotic œdematous process in the skin.

**Kernig's Sign.**—In a large proportion of cases of meningitis, and particularly in cerebro-spinal meningitis, a curious spasm of the hamstring muscles may be observed. It is obtained by fully flexing the thigh to less than a right angle, the patient either lying flat on his back or sitting with the legs dependent. An attempt is then made to straighten the leg at the knee, when it is found that the hamstring muscles are in a state

of tonic spasm or 'active contracture,' preventing extension of the leg.

A symptom frequently seen in **Hysteria** and in **Hystero-epilepsy** is spasm, in both the tonic and the clonic forms.

**Professional Spasm (Occupation Neurosis).**—A tonic spasm or cramp in muscles which are habitually overworked. The most frequently observed form is **writer's cramp**, occurring among persons who daily use the pen for hours together. On attempting to write, the muscles employed in holding the pen contract spasmodically. Occasionally the affection takes the form of a tremor, or may, on the other hand, appear as a simple loss of power in the fingers. The affection is seen among pianists, violinists, telegraphists, etc., under similar circumstances.

**Torticollis (Wry-neck).**—The infantile form may be mentioned, as it is apparently due to a tonic spasm of the sterno-mastoid, but is really a shortening of the muscle.

A tonic spasm of a sphincter muscle is frequently observed, the result, as a rule, of reflex irritation—*e.g.*, **blepharospasm**, **vaginismus**.

The familiar 'cramps' occurring in the muscles of the legs, feet, etc., are instances of tonic spasm. They are due, as a rule, to fatigue, varicose veins, and other defects of circulation, or to the use of the limb in a constrained or unusual position. But they may be an evidence of disease—thus, they often occur in the course of cholera, in diabetes, in gout, in alcoholism, and in hysteria.

**Myotonia Congenita (Thomsen's Disease)** is one of the few instances of increased muscular contraction independent of nervous disease.

Spastic forms of paralysis (see p. 209) may, as stated above (p. 249), exhibit an exaggerated muscular contractility or spasm, combined with loss of response to volitional stimulation.

**B. Clonic Spasms** are involuntary, intermittent, muscular contractions, which, however, do not result in as much movement of the part as one would expect from the strength of the contractions, this being due to the opposed action of the affected muscles.

Both clonic and tonic spasms are due to excessive stimulation of some part of the motor tract, the former almost invariably from lesions of the cerebral cortex; tonic spasms, on the contrary, often originate at the base of the brain or in the cord. Conscious-



ness is frequently abolished when the clonic spasms occur, the condition being then termed a convulsion, or convulsive fit. It is desirable that these terms should be restricted to those cases where unconsciousness accompanies the clonic or tonic spasm.

Clonic muscular contractions may be of all degrees of fineness or coarseness, and of regular or irregular recurrence. The finest variety of clonic muscular spasm is the **fibrillary twitching** of very small bundles of muscle fibres, which are insufficient to alter the position of the origin or insertion of the muscle, and which are seen in muscular atrophy, especially when of central nerve origin (in progressive muscular atrophy, bulbar paralysis, and also in the cachexia of phthisis and of cancer).

**Tremor.**—There is no hard-and-fast line separating fibrillary twitching from tremor. The latter term indicates contractions of a larger mass of muscle, which are sufficiently ample to move slightly the insertions of their muscles.

The patient must be examined both while at rest and while performing such movements or acts as will bring into play the various groups of muscles under observation.

Tremor is seen in the following conditions :

(a) **Senile tremor** is a fine spasm, five or six per second, occurs during rest, and commonly affects the limbs and the head.

(b) The tremor of **paralysis agitans** recurs at about the same intervals of time as the senile, also occurs during rest, but is more effectually checked by purposeful acts. It usually begins in one hand, causing a characteristic 'pill-rolling' movement, spreading then to the other limbs, and rarely affecting the head.

(c) In **disseminate sclerosis** the tremor is less regular and fine than those just mentioned. It is essentially a purposeful or 'intention tremor': it is absent when the muscles are at rest, and commences as a fine tremor when any act involving muscular tension is attempted. With the continuance of the act, the clonic contractions become more and more ample and irregular.

(d) The tremor of **exophthalmic goitre** is fine and rapid (eight to ten per second), and may be increased by movements.

(e) In **hysteria** tremor similar to (d) is often seen.

(f) Tremor of the lips and tongue is seen in **general paralysis of the insane**.

(g) Tremor is observed in poisoning by mercury, lead, alcohol, and nicotine. In most of these cases we find an intention tremor, tending to become persistent as the disease advances.

(h) **Rigors** may be regarded as a coarse form of tremor. They may cause rhythmical contractions of all the voluntary muscles, those of mastication producing the familiar 'teeth-chattering,' seen in fevers and septic absorption. (See Rigors, p. 351.)

(i) Physiological causes may suffice to produce tremors, such as intense cold, excessive muscular exertion, fear, or other emotion.

(j) **Nystagmus** is a clonic spasm of the oculo-motor muscles, which may be regarded as a slow type of tremor. It is best observed by directing the patient to look as far as possible to one side, the eyes alone turning laterally, with the face directed forward. A lateral oscillation of the eyeball (two or three per second) is then seen. In some cases a vertical nystagmus is found, best observed when the patient looks up or down. It is thus an intentional tremor, and is found in so many different affections that its diagnostic value is only moderate. It occurs in above 50 per cent. of cases of multiple sclerosis, and in the majority of cases of Friedreich's ataxia. It is frequently seen in cerebellar disease, less commonly in cerebral hæmorrhages and tumours. It is said (Gowers) to occur frequently in ataxic paraplegia, primary spastic paralysis, and hereditary ataxia, sometimes in severe multiple neuritis and syringomyelia, and rarely in progressive muscular atrophy. It is found in cases of congenitally deficient vision, albinism, corneal opacities, cataract, coloboma, and other local affections interfering with sight, and especially in those which date back to early life. In congenital cases the absence of accurate stimuli from the retina interferes with the proper development of the cortical co-ordinating centres for the oculo-motor nerves. Nystagmus is frequently found among coal-miners. Here the darkness and black surfaces prevent accurate retinal impressions; moreover, the constrained positions (*e.g.*, lying on the side) which must frequently be assumed by miners gives rise to the spasm, somewhat after the manner of the professional spasm.

Clonic spasms of a coarser and more ample description than tremor are observed in the various convulsive affections, of which the following may be mentioned:

**Idiopathic Epilepsy.**—This is seen in two forms—the minor affection, or 'petit mal,' which, as a rule, presents few or no motor symptoms, and the convulsive form, or 'haut mal.' In the latter a general tonic spasm, lasting, perhaps, a few seconds, precedes the

convulsion, which is often ushered in by a tremor. All the voluntary muscles participate in a more or less violent manner, without rhythm or regularity. This stage lasts a few minutes, during which the patient may suffer injury to his limbs, head, tongue, etc. The pathology is unsettled, as this form of epilepsy occurs without discoverable anatomical change in the nervous system, and without any observable toxic blood state.

**Jacksonian or Cortical Epilepsy**, due to intracranial lesions causing irritation of the Rolandic area. In this affection the initial rigidity of idiopathic epilepsy does not occur, but the attack generally commences in one particular group of muscles as a twitching which increases in violence and extent, invading, in the order of proximity, the muscle groups controlled by cortical regions adjacent to the centre first affected. Thus, if the face be the earliest part convulsed, the hand and arm will be next involved, then possibly the leg of the same side, and finally, in severe cases, all the remaining voluntary muscles in the body will take part in the outburst. On the other hand, the clonic spasms may be limited to the part first affected. Consciousness is often undisturbed, or may only be lost toward the end of the attack.

**Hysterical Convulsions** resemble in many respects the epileptic fit. The following points will help to distinguish the conditions. The fit of hysteria is observed almost exclusively among females, comes on somewhat gradually, and never during sleep. There may have been some emotional disturbance immediately preceding the attack. Screaming occurs during the fit, not restricted to its onset. The patient may bite her cheeks and tongue, but is more likely to bite her hands, or any part of the bystanders within reach. Consciousness is not completely lost, and she may talk incoherently during the attack. The fit may be brought to an end suddenly by a shock of some description. After the attack (not during it) she may pass a large quantity of pale urine of low specific gravity, while during the attack the abdomen may be distended with flatus. The muscular contractions are a mixture of tonic and clonic spasms. Opisthotonos occurs frequently, and may alternate with clonic contractions. Attacks may recur at short intervals for some days, without the raised temperature and danger to life of the status epilepticus.

Certain **Toxic States of the Blood** may give rise to clonic spasms; of these, uræmia, puerperal eclampsia, and diabetes are common

examples. Asphyxia, opium-poisoning, and the terminal stages of many diseases ending fatally, may exhibit convulsive seizures.

In addition to the typical Jacksonian convulsions, a more generalized convulsive attack may result from intracranial lesions, in which there is no localization of the seat of the lesion. This may occur in meningitis, extensive cerebral, intraventricular, or meningeal hæmorrhage, severe concussion or other extensive injury of the brain.

**Infantile Convulsions** are, like epilepsy, usually initiated by a short period of tonic spasm. The clonic spasms involve all the voluntary muscles, as a rule, producing irregular movements of the face, head, trunk, and limbs.

**Chorea.**—The involuntary movements are irregular and disorderly, sometimes confined to one half of the body (**hemichorea**), but frequently affecting all the voluntary muscles.

Certain **choreiform affections** may be enumerated as further instances of clonic spasms: **Habit spasm**, a twitching of certain groups of muscles, especially the eyelids and other facial muscles; **convulsive tic** (**Gilles de la Tourette's disease**), a similar and more severe disturbance, with pronounced neurotic and even mental disorder; **Huntington's chorea**, a chronic choreiform affection of hereditary character, occurring in adults; **post-hemiplegic chorea** and **athetosis**, involuntary clonic and irregular movements due to cortical irritation, the result of disease or injury occurring, in most cases, in childhood or infancy. **Birth palsies** (**Little's disease**, **infantile spastic hemiplegia**) are in many cases indistinguishable from the last named conditions.

**Myoclonus** (**Paramyoclonus Multiplex**), a hereditary affection of the muscles. Rapid, frequently-recurring contractions of one or more muscles are observed in various parts of the body.

The following is a **summary** of the affections in which is found an involuntary increase of muscular contraction (spasm):

1. Lesions giving rise to irritation of the sensori-motor area of the cerebral cortex. This may be due to—(a) Local affections, such as meningitis, abscess, hæmorrhage, or other vascular lesions, degenerations, tumours, and traumatic injuries, and may be revealed by convulsions, trembling, rigidity, conjugate deviation of the head and eyes, etc. (b) Peripheral irritation—*e.g.*, infantile convulsions, tetany, laryngismus stridulus, blepharospasm. (c) Toxic blood states—*e.g.*, tetanus, strychnine-poisoning, uræmia, diabetes. (d) Emotional and various functional irritations of

uncertain nature—*e.g.*, hysteria, epilepsy, chorea, wry-neck, professional and habit spasms, convulsive tic.

2. Irritation of the lower motor neuron may, perhaps, produce spasm, as in spinal meningitis and in the early stages of multiple neuritis.

3. Spasticity due to degenerative changes in the upper segment of the motor tract, as seen in—

(i.) Primary lateral sclerosis, and in the hereditary lateral sclerosis described by Erb.

(ii.) Secondary sclerosis, due to—

(a) Cerebral lesions: (a) the 'late rigidity' following cerebral palsies; (β) athetosis, epileptiform convulsions, Little's disease, and other affections following cerebral lesions in childhood.

(b) Transverse lesions of the cord, produced by: (a) compression from vertebral caries, tumours, pachymeningitis, aneurism, traumatic lesions; (β) myelitis; (γ) hæmorrhage into the cord.

(iii.) Lateral sclerosis, combined with other lesions of the cord:

(a) Amyotrophic lateral sclerosis.

(b) Ataxic paraplegia.

(c) Primary combined sclerosis (Dana).

(d) Multiple sclerosis (disseminate or insular sclerosis).

(e) Syringomyelia.

(f) Pellagra and lathyrism, due to the ingestion of poisonous cereals and vetches respectively.

(g) Friedreich's ataxia, a hereditary affection with jerky incoordinate movements.

4. Spasticity due to disease of the muscles:

(a) Myotonia congenita, or Thomsen's disease.

(b) Paramyoclonus multiplex of Friedreich.

Tonic spasm occurs in the following conditions: Tetanus, tetany, spasm of the glottis, strychnine-poisoning, meningitis, hysteria, professional spasm, torticollis, blepharospasm, myotonia congenita. In cases of spastic paralysis there may be a combination of increased muscular contractility with loss of power.

Clonic spasm occurs as fibrillary twitching, tremor, or coarser convulsive movements in the following affections: Progressive muscular atrophy, bulbar paralysis, cachexia of cancer or phthisis, senility, paralysis agitans, disseminate sclerosis, exophthalmic goitre, general paralysis of the insane, poisoning by mercury, lead, alcohol, and nicotin, rigors (nystagmus is seen in multiple sclerosis, in Friedreich's ataxia, in cerebellar disease, etc.), idio-

pathic and Jacksonian epilepsy, infantile convulsions, chorea, various choreiform affections—*e.g.*, habit spasm, Huntington's chorea, athetosis, birth palsies, etc.—myoclonus.

### MUCOUS RÂLES.

A large and coarse variety of râle, originating in the larger bronchi, or in pulmonary cavities. They occur in bronchitis bronchiectasis, and phthisis (see p. 419).

**MUREXIDE TEST (for Uric Acid and Urates).** See *Urine Examination of*, p. 538.

### MURMURS.

The use of this word should be restricted to those sounds which are generated by a stream of blood passing into or out of the chambers of the heart, or through the larger bloodvessels; pericardial friction sounds should be excluded from this term.

The varieties of murmurs and their significance are considered in detail in the article on Auscultation of the Thorax, at p. 432.

**MUSCÆ VOLITANTES** (*L. musca*, a fly; *volito*, to flutter).

Floating spots and nodular threads may be seen by persons suffering from digestive disturbances, hysteria, cardiac hypertrophy, cerebral anæmia and hyperæmia.

### MYDRIASIS.

Abnormal dilatation of the pupil. This may be due to excessive stimulation of the sympathetic (irritative or spasmodic mydriasis), or it may result from paralysis of the third nerve (paralytic mydriasis).

The pupil is dilated in the following conditions: (1) In childhood; (2) poisoning by certain drugs (mydriatics); (3) direct irritation of the dilator pupillæ centre or of the sympathetic fibres in some part of their course—*e.g.*, spinal meningitis, disease of the cervical vertebræ, dyspnœa, tumours of the neck, etc.; (4) indirect irritation of the sympathetic system—*e.g.*, emotions, mania, peripheral irritation (worms, etc.), debility; (5) paralysis of the sphincter pupillæ (iridoplegia), as a result of lesion of the pupil-contracting centre or of the nerve fibres supplying the

sphincter, or the effect of defective perception or conduction of the light stimulus. Cerebral lesions affecting the cortical centre of vision in the occipital lobe or its connections with the oculomotor nucleus cause blindness (supranuclear blindness), but as the pupil-reflex arc is undisturbed, the pupil contracts on exposure to light, though sight is lost (see p. 321).

**MYOSIS** (Gr. *μύα*, to close).

An abnormal contraction of the pupil, due to irritation of the third nerve or its central connections, when it is termed irritative or spasmodic myosis; or it may be due to paralysis of the sympathetic innervation (paralytic myosis).

Myosis occurs in (1) advanced age; (2) sleep; (3) certain poisons known as myotics cause the symptom; (4) intracranial lesions which irritate the third nerve; (5) excessive use of the accommodation centre; (6) affections of those parts of the eye supplied by the fifth nerve; (7) paralysis of the sympathetic. Among the last group may be mentioned lesions of the spinal cord damaging the sympathetic and giving rise to **spinal myosis**, seen in locomotor ataxia, etc.

The subject is more fully considered in the article on Abnormalities of the Pupil (p. 320).

**NARCOSIS.** See **Unconsciousness**, p. 498.

**NASAL VOICE.** See **Voice, Abnormalities of**, p. 555.

**NAUSEA** (L. *nausea*, sea-sickness; from Gr. *ναῦς*, a ship).

The feeling of sickness from any cause is closely related to vomiting, and is further referred to in the article on that subject (p. 557).

**NODES ON FINGER-JOINTS.** See **Heberden's Nodes**, p. 163.

**NOSE, Affections of the.**

Abnormal discharges and other disorders of the nose, are, as a rule, the result of local disturbances, and are not of interest in the diagnosis of medical affections. A brief reference to a few points in this connection will suffice.

A broad and coarsely-shaped nose is seen in myxœdema; a depressed or saddle-shaped nose may be due to syphilis if injury can be excluded; redness of the nose, if chronic, may be due to digestive or menstrual disturbances, or to alcoholism.

Active nostrils, dilating with each inspiration, are in most cases evidence of dyspnœa (see p. 131); in pneumonia, especially of children, the sign is of much value. Obstruction to the entrance of air to the lungs, emphysema, cardiac disease, are familiar instances of the symptom.

Epistaxis: Bleeding from the nose is frequently due to local disorders. Occurring in conjunction with headache and pyrexia, it is suggestive of typhoid fever. It is seen occasionally in other fevers (measles, scarlet fever, etc.); it is common in Bright's disease, in cardiac disease, in hæmophilia, in leukæmia, in purpura, and in anæmia of various descriptions. Instances of epistaxis replacing the menstrual discharge (**vicarious menstruation**) have been recorded.

Disorder of the Sense of Smell (see p. 372).

Nasal Discharge: A watery discharge is commonly the sign of coryza; it may be part of a more general catarrh, or may be due to iodism. Purulent and offensive discharges are almost invariably the result of local disease.

### **NYSTAGMUS** (Gr. νυστάζω, to nod in sleep).

A clonic spasm of the oculo-motor muscles, causing an oscillating movement of the eyeball at about the rate of two or three contractions in the second; the movement is usually in the lateral direction, but may be vertical. Its varieties and causes are enumerated in the article on Increased Movements (p. 254).

### **ŒDEMA** (Gr. οἰδέω, to swell).

An increased quantity of fluid in the skin and subcutaneous tissues gives a rounded, full appearance to the part; the wrinkles natural to the region become smoothed out or obliterated, and hollowed portions of the surface are more or less filled up.

This increase in the amount of superficial fluid may be mainly situated in the capillaries, small veins and arteries, or lymphatics, in which case the skin has usually a deeper flesh tint, and is more elastic than normal, and the condition is that of **turgidity**. A further stage in the overloaded subcutaneous tissue results in the



accumulation of the blood fluid in the lymph-spaces, whence it is inefficiently removed by the lymphatics. The effect of this stasis of fluid is to cause the part to present a shiny, swollen, tense, and plump appearance, the normal prominences and hollows being smoothed out; the skin and subjacent tissues lose their natural elasticity, as shown by the length of time required to enable the surface to regain its level after being disturbed by pressure ('pitting on pressure'). Over certain regions (especially the thighs and abdomen) the deeper layers of the skin, together with the lymph-spaces, may give way along lines which show as moderately transparent red or white superficial lines, resembling the *striae gravidarum*; the skin is usually white except in cyanotic and inflammatory œdema, owing to pressure upon the vessels of the region. A generalized œdema is termed *anasarca*.

It may be said that the immediate cause of œdema is inability on the part of the lymphatics to remove the accumulated fluid in the lymph-spaces, the latter being the result of a want of balance between the blood and lymph circulations, brought about by a variety of conditions. The normal flow of lymph is constantly passing from the capillaries into the intercapillary cellular spaces, whence it is carried by the lymphatic vessels back to the blood via the thoracic duct. Its passage through the capillary walls is effected by (a) **filtration**, resulting from the excess of pressure in the capillaries over that in the lymph-spaces; (b) **osmosis**, depending upon the nature and quantity of the substances in solution; (c) the permeability of the capillary walls; and (d) the selective or secretory action of the endothelial cells forming the capillary vessels. In this connection may be mentioned Fischer's work, which goes to indicate that the accumulation of acids within the tissues is the chief cause of œdema, by increasing the affinity of the colloids for water. The fluid lymph which has transuded from the capillaries differs in some respects from blood-plasma: its specific gravity is lowered from about 1028 to 1015; its solid contents are about three-fifths of those of blood-plasma, the chief loss being in proteids; salts are similar in amount and in kind to those of plasma; waste products (urea and carbonic acid) derived from the tissues, are more abundant than in plasma. The further progress of the fluid from the lymph-spaces and along the lymphatics is effected by muscular and external pressure, the proper direction of the current being maintained by numerous valves in the course of the lymphatic vessels. With the accumulation of fluid in the

cellular tissue the pressure there rises; thus the capillaries may absorb again some of the lymph, so assisting the lymphatics in the removal of excessive extravascular fluid.

In various diseased conditions this equilibrium between the blood and lymph circulations may be disturbed, whereby the amount of fluid in the cellular tissue rises or falls. The disorders of the flow may arise from alteration in the respective pressures of the venous and lymphatic systems, from alterations in the constituents of the plasma, or from changes in the degree of permeability of the capillary walls.

An increase in the fluid contained in the lymph-spaces (*œdema*), or in the larger serous cavities (*serous effusion*), resulting from one or more of the above abnormal conditions, may be caused by (a) venous obstruction; (b) an excess in the quantity of blood and a diminution in its solid contents—*i.e.*, a *hydræmic plethora*; (c) inflammation; (d) disturbances of nervous origin.

(a) **Excessive venous pressure** causes an abundant transudation of lymph from the capillaries by mere filtration; long-continued high pressure will also damage the walls of the capillaries, increasing their permeability; the reabsorption of lymph by the capillaries and small veins is also prevented by high pressure in these vessels; the outflow of lymph from the main lymphatic ducts into the large veins is impeded by a similar condition. For all these reasons the amount of fluid passing from the blood vessels to the connective tissue may be greater than the lymphatics are capable of removing, and *œdema* is the result.

An obstruction to the venous return occurs in cardiac affections, in which the right heart is overfull; failure of compensation for valvular defects, or defective contractility from any cause renders the emptying of the *venæ cavæ* a matter of difficulty. Pressure upon the inferior *vena cava* in the abdomen may occur as a result of tumours, enlargements of the liver, pancreas, or bowel, or of ascites; enlarged glands or tumours of the mediastinum and thoracic aneurism may obstruct the superior *vena cava*. In these conditions *œdema* is considerably influenced by gravity; hence its situation varies with the posture of the patient. The region earliest and most markedly swollen is the most dependent part of the body, and *œdema* from obstruction of the *venæ cavæ* will affect both sides of the body. It is otherwise when the obstructed vein is of less general distribution: tumours in the mediastinum, neck, or axilla, thrombosis, and traumatism may obstruct one

subclavian or axillary vein, causing localized œdema of that limb; similarly one or other leg or one side of the head and neck may be the œdematous region.

(b) Another condition which assists in the production of œdema is **Hydræmic Plethora**. Here the volume of blood is increased and its density is diminished by undue retention of water in the circulation. This form of œdema is less affected by gravitation than the preceding, being apparently influenced by local variations in activity of the lymphatic circulation. It is probably an important factor in the production of œdema in Bright's disease; here, however, increased arterial tension may be a factor, as the capillaries must share in the high tension and so suffer increased permeability. That arterial tension alone is insufficient to produce œdema is evident from the fact that dropsy is usually absent in renal sclerosis where a high-tension pulse is the rule: in this condition the gradual development of the disease induces hypertrophy of the heart, with consequent activity of the venous as well as of the arterial circulation, so that venous stasis and obstruction is obviated. In the end, however, such cases present œdema commencing in the most dependent regions, owing to heart failure. The marked œdema which is found in acute nephritis with high arterial tension is partly accounted for by the absence of hypertrophy of the heart, which has not had time to develop. Associated with hydræmic plethora there occurs in many affections (especially in renal disease) a toxic condition of blood which doubtless has some effect in damaging and rendering more permeable the capillary walls.

The œdema of anæmia in all its forms, and of chlorosis in particular, is largely the result of hydræmic plethora; here also cardiac debility, toxæmia, and changes in the capillary walls are in part responsible.

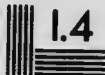
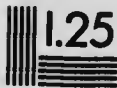
(c) Œdema due to **Inflammation** is of more interest to the surgeon than to the physician; on the rare occasions that a superficial œdema occurs over a deep-seated collection of pus it is of the first importance in diagnosis. This may sometimes be observed in empyema, appendicitis, abscess of the liver, purulent pericarditis, etc. In rheumatism the articular swelling may be the result of œdema quite as much as of arthritis.

(d) **Angioneurotic Œdema**.—Certain rare forms of œdema occur on the face and elsewhere, which are apparently due to disorder of the vaso-motor innervation. The spots affected may vary in



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size up to several inches in diameter; they may be distinguished from urticaria (with the giant form of which they are often confounded) by the absence of itching, burning, or irritation of the surface.

Instances of œdema resulting from nervous disturbance may be seen in the swelling of peripheral neuritis, in the cyanotic or 'blue œdema' of hysteria, and perhaps in the weals of urticaria.

Obstruction of the lymphatics may occur from inflammation or new growths in the lymph-glands, from pressure or other obstruction of the large lymph-vessels, or from blocking of the lymph-vessels by the filaria or its ova. Under these circumstances one might suppose that œdema would ensue; this, however, does not occur, the change of lymph in the affected tissues being evidently effected by reabsorption into the capillaries and venules. If this is insufficient a brawny swelling of the parts may result, which does not pit on pressure—*e.g.*, lymph-scrotum and certain forms of elephantiasis.

**Summary.**—œdema is the presence of an excessive amount of fluid in the cellular tissue; anasarca is the same generally distributed over the body; serous effusion is the collection of fluid in larger serous-lined cavities.

œdema may be the result of one or more of three conditions: increased venous pressure, altered states of the blood-plasma, and increased permeability of the capillary walls.

The swelling is in the most dependent parts in all forms of general œdema, except in the early stages of renal dropsy, when it is often first noticed in the face. In the later stages of renal dropsy the swelling is most marked in the more dependent regions.

Localized œdema usually results from obstruction of a large vein draining the region affected. One arm may be œdematous from obstruction of the subclavian or axillary artery (enlarged glands, tumour, thrombosis). One leg may be swollen from similar conditions affecting the femoral or iliac veins. This is seen in the œdema which sometimes follows parturition (*phlegmasia alba dolens*) or inflammation of the pelvic organs in women; a common cause of œdema in one leg is the blocking of a varicose vein by a thrombus, the result of *phlebitis*. One side of the face may be œdematous from pressure upon the veins in the neck (tumour or enlarged glands), or, in cases of general dropsy, from having lain for some time upon the affected side. Both arms, head, and neck will be swollen if the superior vena cava is

obstructed (mediastinal tumour and thoracic aneurism), while if the obstruction includes the azygos vein œdema will be seen on the thoracic walls.

Swelling of both legs is found in obstruction of the inferior vena cava from:—pressure of new growths; enlargements of the liver, pancreas, spleen, or mesenteric glands; from pressure of inflammatory exudations, or of mere ascitic fluid.

A localized œdema may be the superficial evidence of a deep-seated suppuration, as in empyema, appendicitis, abscess of the liver, purulent pericarditis, etc.

### ŒSOPHAGUS, Examination of.

It is chiefly by passing a sound or tube into the stomach that one is able to obtain information as to the condition of the œsophagus. The method to be adopted in this procedure is described in the article on the Examination of the Stomach (p. 395).

Should difficulty be experienced in passing the instrument, one notes the distance from the teeth at which the obstruction is encountered, bearing in mind that the œsophagus begins about six inches from the incisors, and extends for a distance of about ten inches before it enters the stomach.

Narrowing of the channel is most frequently due to cancer. In this affection the bougie, on being withdrawn, is found to be blood-stained in many cases, and particles of the new growth may even be found adhering to the instrument, especially if a hollow instrument with an eye near its point be used.

Spasm of the canal may in neurotic individuals be a temporary cause of obstruction. A little delay will overcome it.

Stricture of the œsophagus also results at times from syphilis, or from the cicatrix following a burn. Foreign bodies impacted in the canal are an occasional cause of the block.

Passage of the instrument with ease on some occasions, while it is stopped at others, points to a diverticulum.

Pressure on the œsophagus by enlarged thyroid or lymphatic glands, by aneurism of the aorta, or by new growths, may also be the cause of obstruction.

By palpation of the neck one may at times discover a diverticulum, or a tumour pressing on the œsophagus.

Auscultation of the œsophagus is of some little diagnostic value. Six or seven seconds after swallowing one hears normally

a gurgling sound on placing the stethoscope an inch to the left of the spine at the level of the eighth dorsal vertebra. If there be an obstruction, the sound is either lost or delayed.

Should there be any grounds for suspecting an aneurism to be the cause of the stenosis, the bougie or tube must on no account be employed, as it might cause rupture of the sac and a rapidly fatal result.

**OLIGURIA** (Gr. *ὀλίγος*, scanty; *οὔρον*, urine).

A deficiency in the quantity of urine passed in a given time. See *Abnormalities of the Urine*, p. 503.

**OPISTHOTONOS** (Gr. *ὀπισθεν*, behind; *τόνος*, a stretching).

A tonic spasm of the muscles of the trunk, causing the body to be curved backward, so that it rests on the head and the heels. It may be seen in many cases of tetanus, and at times in strychnine-poisoning.

**OPSONIC POWER** (L. *opsono*, to prepare a feast). See **Blood Examination**, p. 81.

**OPTIC ATROPHY.**

A pale, hollowed condition of the optic disc may be observed by means of the ophthalmoscope ('cupped disc'), with shrunken and thick-walled vessels, the result of atrophy of the optic nerve.

This affection may be either a primary disease of the nerve or may be secondary or consecutive to pre-existing disease in the nerve or the brain. Further details regarding the nature and diagnostic value of the symptom will be found in the article on *Disturbances of Vision*, at p. 551.

**OPTIC NEURITIS.**

On examining the fundus oculi by means of the ophthalmoscope we may note changes in the optic disc and surrounding retina. These vary in degree from a slight woolliness of the margin of the disc, with moderate swelling and hyperæmia, to a severe congestion and inflammation of the papilla (papillitis or 'choked disc'), with considerable inflammatory changes in the retina.



This condition arises most frequently as a result of tumours and abscess of the brain (syphilis, tubercle, cancer, glioma); also from meningitis, hydrocephalus, and occasionally in spinal disease—viz., tabes dorsalis and myelitis; also rarely in peripheral neuritis. Tumours of the orbit are an occasional cause. Optic neuritis may also be a symptom of more general disease in which the nervous system takes part—*e.g.*, Bright's disease (associated with retinitis, a neuro-retinitis), chlorosis, rheumatism, lead-poisoning, syphilis (independent of cerebral tumours), suppression of menstruation, and exposure to cold.

The subject is more fully considered in the article on Disturbances of Vision (p. 543).

### ORTHODIAGRAPHY.

Parallel X rays are passed through the body by a special apparatus (Morritz), and the position of any solid mass is mapped out on a screen. Said to be specially useful in demonstrating heart enlargement.

**ORTHOPELTISSION.** See p. 283.

**ORTHOTONOS** (Gr. *ὀρθός*, straight; *τόνος*, a stretching).

A tonic spasm of the muscles of the trunk, which so balance and equally oppose each other that the body is straight and rigid. It may occur in tetanus and in strychnine-poisoning.

### OXALURIA.

Oxalate of lime may be found in the urine as octahedral crystals (the 'envelope' crystal), or more rarely as oval or dumb-bell forms. They are insoluble in weak acids (*e.g.*, acetic) and soluble in strong mineral acids.

Oxalates may be found in the urine of persons susceptible to the effects of certain vegetables (*e.g.*, rhubarb, tomatoes, onions). Injudicious and excessive food, digestive disturbances from any cause, want of exercise, and anxiety are the commonest causes. They are more likely to appear after the urine has stood for some time. At times they occur in diabetes, when the sugar diminishes. Their continued presence causes irritation in the urinary tract, and even albuminuria (see p. 511).

**PAIN.**

Tenderness—Degree of pain—Signs of severe pain—Referred or reflected pain.

Headaches: migraine; neuralgia; dyspeptic; neurasthenic; hysterical; anæmic; of abdominal and pelvic disease; of eye and nose affections; of intracranial disease; syphilitic; of fevers; nephritic; toxic; rheumatic; gouty; diabetic; of disease of the ear and bones of the skull.

Pain in the face—Pain in the neck—Sore throat—Thoracic pain—Pain of the vertebral column—Abdominal pain—Pain in the gluteal region—Pain in the external genitals—Pain at the anus—Inguinal pain—Pains in the arms and legs.

Pain occurs either as a subjective symptom, independently of external interference, or as an objective sign, produced by pressure or other irritation. It is then spoken of as tenderness. Like most other subjective symptoms, pain is not of excessive value as an aid to diagnosis. There are, however, many features in the symptom as it occurs in different localities and in different diseases which require attention.

It is often difficult to determine the amount and the quality of the pain of which the patient complains. With every intention to give an accurate statement of his sensations, the temperament, the habits, and the previous experiences of the sufferer so strongly influence his capacity for bearing pain that an accurate estimate of his sufferings is often wanting. One must be guided to a large extent by the posture, the expression, and the condition of the muscles which are in relation to the affected region.

Very severe pain may cause a condition resembling collapse. The pulse and respirations are rapid, the skin pale and perspiring, the pupils dilated, and a feeling of faintness may be experienced.

It is the situation of the pain and the effect produced upon it by external interference, such as pressure, movement of the part, etc., which are of most value as an indication of the nature and extent of the affection. It must be borne in mind that pain is often referred to a region quite remote from the source of the suffering. In some cases of internal disease, for example, pain may be referred to a superficial spot or area by a sympathetic or reflex process. The stimulus from the diseased organ proceeding to the sensory centre, there overflows, as it were, or irradiates to a neighbouring sensory centre, which may be the perceptive

centre of the skin area to which the pain or other sensation is attributed. The subject has been worked out by Head, who has mapped the skin areas, the afferent nerves of which correspond to those from certain internal organs.

The pain may be generally distributed, or may be confined to one or more regions of the body. The former condition is found in many fevers, and is especially characteristic of influenza and small-pox. It may also be found in acute and chronic rheumatism.

**Pain Regions.**—The various regions of the body in which characteristic pain is felt may be separately considered :

**Headache (Cephalalgia).**—The following is a list of the chief causes and conditions which contribute to pain in the head: Migraine; neuralgia; dyspepsia; neurasthenia; hysteria; anæmia; pelvic or abdominal disease; affections of the eye, nose, and naso-pharynx; intracranial disease; syphilis; fevers; nephritis; alcoholism; plumbism; gout; rheumatism; disease of the bones of the head. The distinguishing features of the different types of headache are discussed at p. 160.

**Pain in the Face.**—In addition to the causes already mentioned under headache, pain in the forehead and brows may be due to affections of the nose and frontal sinuses, of the eyes and of the mouth. Facial neuralgia is usually confined to one side, and may involve one or more branches of the fifth nerve. Pain in the ear, radiating through the head and face, is most frequently due to otitis media, furuncle of the meatus, or mastoid abscess. It may also be caused by carious teeth, tonsillitis, or other mouth affections. In the temporo-maxillary joint pain often occurs in rheumatism (sometimes in the gonorrhœal variety), while mumps gives rise to a painful swelling at and below that region. The pain of mumps is often referred especially to the lower jaw. Disease of the bones of the face—*e.g.*, caries, malignant growths, antrum disease—and of the teeth and tongue, are sometimes the cause of severe facial pain.

**Pain in the Neck.**—In the **front** and **sides** of the neck pain is often caused by inflammatory affections, of which adenitis is the commonest. Tonsillitis, pharyngitis, laryngitis, retropharyngeal abscess, foreign body in the throat, cancer, cervical caries, and myalgia may also be mentioned.

At the **back of the neck** the commonest cause of pain is the so-called muscular rheumatism, or myalgia, nearly related to which

is cervico-occipital neuralgia. Less frequent sources of pain in this region are meningitis, cervical caries, myelitis, tetanus.

**Sore throat** (see p. 479).

**Thoracic Pain.**—The following is a summary of painful conditions of the different regions of the chest and their causes:

The **upper portion of the front of the chest** (clavicular, supraclavicular, and infraclavicular regions) may be the seat of pleuritic pain, in which situation the disease is probably, but not certainly, tubercular. Pain arising from irritation in the large intestine, stomach, or diaphragm is not infrequently referred to this region.

Pain **behind the sternum** is a common symptom of imperfect digestion. It is usually felt as a gnawing soreness and weight, appearing a couple of hours or so after a meal. It also occurs frequently in catarrh of the trachea and bronchi, especially in the early and acute stages. The following affections causing pain behind the sternum are less frequently encountered: Angina pectoris, a paroxysmal oppressive and suffocating pain, usually radiated to the left shoulder and arm. Disease of the sternum or vertebræ (caries, syphilis); pain and tenderness easily localized. Aneurism: the pain is sometimes severe and gnawing in character; it is often seated in the back. Mediastinal new growths or other tumours (*e.g.*, enlarged glands). Pericarditis causes pain in this region, or more commonly to the left (see below).

Pain in the **female breast** may be due to disease of that organ (mastitis, cyst, cancer, cracked nipple). It may also occur in pregnancy and at times during menstruation. Disease of the ovaries and uterus may give rise to mammary pain, and it is also a frequent symptom of hysteria.

The **præcordial region** is the seat of pain in affections of the stomach, heart, and bowels. Gastric catarrh, flatulent distension of the stomach, ulcer and cancer of the stomach, or gastric neurosis, may be the cause. Of heart affections pericarditis is the most likely to produce pain, though endocarditis and myocarditis may be accountable. The pain of pericarditis is often sharp and stabbing in character, and is usually aggravated by pressure over the lower end of the sternum. Of cardiac lesions which cause præcordial pain aortic disease is the commonest, in connection with which, or in the absence of obvious heart disease, the pain of angina pectoris occurs. It is of an agonizing character, with a sense of constriction and compression of the heart. It radiates,

as a rule, to the neck and arms, especially the left, and may be felt at distant parts of the body. Closely simulating the pain of true angina is the pseudo-angina, which occurs in anæmic, hysterical, and debilitated states.

In the **axillary and infra-axillary regions** a common cause of pain is pleurisy. Here the pain is stabbing, aggravated by movement, and compelling the patient to restrict his respiration to abdominal action or the shallowest possible thoracic motion. Pneumonia causes a similar pain, which is really pleuritic. The sharp pain of pleurodynia (muscular rheumatism) and of intercostal neuralgia is also frequently found in the infra-axillary region, the latter oftenest on the left side. Flatulent or fæcal accumulations of the colon and stomach disorders (on the left side) give rise to pain in this region. Herpes zoster is a not infrequent cause of pain here, and it should be remembered that the pain often comes on before the eruption appears.

Pain in the **shoulders** is oftenest due to rheumatism in or near the joint, or in the muscles of the region. Referred pain from lesions of internal organs are not infrequently seated in this part, and may be due to affections of the liver (hepatitis, cancer, gall-stones), of the stomach and bowels, and of the aorta. Pleurisy, apical pneumonia, and phthisis may cause severe pain in the neighbourhood of the shoulder and scapula.

The **infrascapular regions** are the seat of pain resulting from pneumonia and pleurisy. On the right side affections of the liver may cause pain, and on the left the stomach and spleen may be at fault. The lowest part of the thorax behind is often the seat of pain from movable kidney (commonest on the right side), from gastric ulcer (usually close to the left side of the eleventh and twelfth dorsal vertebræ), from renal calculus (one-sided and aggravated by percussion), from lumbago or myalgia, acute nephritis, constipation, flatulent distension of the bowels, and occasionally from pelvic disease.

Pain in the **interscapular region** is most commonly due to stomach affection: —*e.g.*, gastric catarrh, flatulence, gastric ulcer, or cancer. It is a common seat of caries of the vertebræ, causing pain at the site of the lesion and radiating along the course of the spinal nerves involved. Muscular rheumatism may also affect this region, though perhaps not so frequently as the shoulders or loins. A boring, persistent pain in this portion of the back may be due to aneurism of the aorta.

The **vertebral column** in some portion of its length, may be the seat of pain. Lateral curvature (**scoliosis**) occurring in anæmic and debilitated conditions, or resulting from unequal length of the limbs or other deformity, may give rise to pain in this situation. Neurasthenia and hysteria are often the cause. In these conditions pressure over the spines of the cervical and dorsal vertebræ often causes pain. In this connection may be mentioned the irritable condition of the vertebral column known as **railway spine**, a traumatic neurasthenia. Caries of the vertebræ, as already mentioned, may cause spinal pain, which may occur in any portion of its length, but is commonest in the middle or lower dorsal regions. Acute feverish conditions may give rise to pain in this situation. It is often observed in influenza, and in the rarer affection small-pox. Meningitis, myelitis, locomotor ataxia, syringomyelia, and tetanus may be the cause. Abdominal diseases, such as ulcer of the stomach, cancer of the liver, inflammation or cancer of the pancreas, affections of the uterus or other pelvic organs, aneurisms of the thoracic or abdominal aorta, mediastinal tumours, are among the sources of pain in the vertebral column.

The pain may be in many cases elicited by pressure or percussion, or by the application of heat (*e.g.*, a hot sponge applied to the back may cause pain at the seat of the lesion). This is especially the case in inflammatory affections of the spinal column and canal, such as spondylitis, myelitis, meningitis.

Pain over the **sacrum** or **coccyx** is often experienced in cases of rectal and anal irritation—*e.g.*, cancer, piles, fissure and fistula of the anus; in disease or irritation of the other pelvic organs—*e.g.*, sexual excesses, enlargement of the prostate, uterine disease, ovaritis, pelvic cellulitis; sciatica; sacro-iliac disease; hip-joint disease; neuralgia of the coccyx (**coccygodynia**).

**Abdominal Pain.**—In the **hypochondria** pain may be due to movable kidney or renal calculus. The former is worse in the upright position, and is of a dragging, sickening character. The latter is a severe, colicky pain, radiating downwards towards the pubes (or testicles in men). The pain of pleurisy is often felt at a much lower level than the seat of the inflammation. It is not unusual to find the patient refer his pain not only to the hypochondriac, but even to the lumbar, epigastric, or umbilical region. On the right side affections of the liver (active and passive congestion, cirrhosis, cancer, abscess, etc.) and gall-bladder are

frequent causes of pain. In gall-bladder cases, and especially in gall-stones, the pain is often of a paroxysmal and excruciating character, and is felt over the epigastric, umbilical, and lumbar regions. On the left side the pain is often due to gastric catarrh, gastric ulcer, or flatulence. Enlargement of the spleen and perisplenitis cause pain in this and the adjoining regions.

In the **epigastrium** the pain is most frequently due to affections of the stomach. A sharp, cutting pain, felt soon after eating, and consistently referable to one small area, usually in the epigastrium, and with or without a pain of similar character at a corresponding spot at the back, is strongly suggestive of gastric ulcer. The pain of cancer of the stomach is usually more persistent than that of the peptic ulcer, and is likewise aggravated by food. The pain of dyspepsia may be felt at once after eating, or more frequently after some time has elapsed. Affections of the liver and gall bladder may be felt in this region (see above). It is not unusual for the pain of appendicitis to be referred to the epigastrium, especially in the earlier periods of the attack. In this region, also, the pain of pneumonia may be sometimes experienced. Diaphragmatic pain, the result of pleurisy or of violent coughing or vomiting, is felt chiefly in the epigastrium. Disease of the pancreas or of the vertebræ and aneurism may infrequently cause epigastric pain.

**The right lumbar and iliac regions.**—Affections of the first part of the large intestine cause pain in these regions. In the iliac portion the pain may be due to fæcal accumulation in the caecum, to colic, or to appendicitis. In the latter condition the pain may in the first instance be general over the abdomen, or may be chiefly complained of in the epigastrium. On pressure a point midway between the umbilicus and the anterior superior iliac spine will be acutely tender (McBurney's point). In this region, also, intussusception is oftenest found, though it usually extends across the umbilical region, and here the pain is most acutely felt. In typhoid fever it is usual to find a moderate degree of tenderness in the right iliac region. As the right kidney is more frequently movable than the left, it is more customary to find the pain of floating kidney on the right side than the left, and it may extend to the lumbar, iliac, or umbilical regions.

In the **left lumbar and iliac regions** pain may be due to affections of the descending colon—viz., fæcal accumulation or stricture of

the gut, volvulus, colitis. It may be caused by enlargement or inflammation of the spleen, or of its peritoneal covering.

In the **lumbar or iliac region of either side** one finds pain as a result of hernia, varicocele, renal colic, ovaritis, pelvic cellulitis, colitis, tubercular or other ulceration of the bowel, etc.

Pain in the **umbilical region** may be the first sign of appendicitis or of ruptured gastric ulcer. Omental cancer or tubercular infiltration is frequently the cause of pain in this region. Umbilical hernia may give rise to considerable pain in the tumour, more especially if it be incarcerated or obstructed. It is not unusual for the pain of strangulated inguinal or femoral hernia to be mainly located in or near the umbilicus. Gall-stones and floating kidney, as already mentioned, may cause pain in the umbilical region. The colic of intestinal irritation and of chronic lead-poisoning is also chiefly felt in this area. Pelvic tumours—*e.g.*, uterine fibroids, when they ascend into the abdomen—may give rise to pain or tenderness near the umbilicus.

The **hypogastric region** may be the seat of pain from affections of the urinary bladder, of which cystitis is probably the commonest. Here the pain is of a smarting, burning character, and is associated with vesical tenesmus. Stone in the bladder commonly produces suprapubic pain, which is worse after the bladder is emptied, and is often accompanied by a pain at the point of the penis. Tuberculous or malignant disease of the bladder are also the cause of pain in this region. Disorders of the female genital organs are a frequent cause of hypogastric pain. Dysmenorrhœa, uterine inflammation or tumours, and ovarian cysts and inflammation may be mentioned. Pelvic inflammation and pelvic hæmorrhage, including extra-uterine fœtation, give rise to pain in the hypogastric and inguinal regions.

**Generalized pain and tenderness in the abdomen**, usually severe, and accompanied by distension, rigidity, etc., is characteristic of peritonitis from any cause. The pains of gastro-intestinal disease, of hepatic, renal, and other affections mentioned in the foregoing pages as occurring chiefly in one or other of the regions of the abdomen, may under certain circumstances be generally distributed over the whole abdomen. The same may be said of the pains associated with the gastric crises of locomotor ataxia, with the pains due to irritant poisoning, cholera, etc.

**Pain in the Gluteal Region.**—A common cause is sciatica. In



this affection there is a one-sided, dull, boring pain, with acute, cutting exacerbations. It may for a time be confined to the gluteal region or to the more distal regions supplied by the sciatic nerve. On pressure at certain points over the nerve or its branches tenderness is marked (see p. 228). Sacro-iliac disease and hip-joint disease are also the cause of pain in this region. The lightning pains of locomotor ataxia are usually worse in the buttocks and back of the thigh, but may be severe in the leg and foot. Disease of the pelvic organs often causes pain in the gluteal region and back of the thigh. This may occur apart from the pressure of intrapelvic tumours on the sciatic nerve.

Pain in the **external genitals** most commonly indicates local disease. In the testicle, however, pain may point to stone in the kidney or ureter. It is also found as *neuralgia* in neurasthenia. Pain at the point of the penis may indicate local affections, such as gonorrhœa, chancres. It may also be referred pain in case of stone in the bladder.

Pain at the **anus** is usually either piles or fissure. The former is a dull, throbbing, weighty pain as a rule, while the latter is sharp, cutting, or stabbing in character.

Pain in the **groin** may be due to renal colic, to intestinal colic, to hernia, varicocele, enlarged inguinal glands, etc.

Pains in the **arms** and **legs** are very often due to rheumatism. This commonly affects the joints especially. The lightning pains of locomotor ataxia are severe, stabbing pains felt in the legs and sometimes in the trunk by patients suffering from this affection. In the **hands** gout and osteo-arthritis, and certain diseases of the nervous system, are the cause of pain. In most of the nervous diseases pain is of much less diagnostic value than disturbances of the motor, reflex, or trophic functions. See the articles on Movement, Disturbances of, p. 202; Reflexes, p. 334; and Trophic Disturbances, p. 487.

### PALATAL REFLEX.

This consists in the elevation of the soft palate on being touched. (See Reflexes, p. 343.)

### PALPITATION.

When the individual becomes aware of the movements of his heart, and especially if its action be rapid, forcible, and irregular,

he is suffering from palpitation. This is a symptom most commonly of functional disturbance of the heart; it is, therefore, oftenest observed among females, in whom it is frequently a sign of neurotic disturbance, and accompanies hysteria, neurasthenia, emotional disturbances, disorders arising in connection with the onset of puberty or of the menopause. It may be due to anæmia, dyspepsia, the abuse of tobacco, alcohol, coffee, or tea. Excitement and overexertion, especially in those who are not in good physical condition, cause palpitation, as seen in the irritable heart of soldiers after a forced march.

Valvular disease of the heart and disease of the myocardium are occasional causes of palpitation. It is also seen at times in the rapid and weak heart of acute fevers. An extreme degree of palpitation and irregularity is termed **delirium cordis**.

#### PANCREATIC REACTION.

In cases of inflammation of the pancreas, P. J. Cammidge has shown that an osazone is precipitated from the urine by treatment with phenylhydrazin hydrochlorate and sodium acetate. The crystals as seen by the microscope are light yellow, flexible, hair-like, and arranged in sheaves; they form a light yellow flocculent precipitate, which comes down in a few hours. For full details as to the test see the *British Medical Journal*, May 19, 1906.

#### PAPILLITIS (Choked Disc, Stauungspapille).

In cases of severe optic neuritis the papilla is seen (by means of the ophthalmoscope) to be swollen, the veins greatly distended, and inflammatory changes are found in the surrounding retina. The affection is a symptom of disease of the brain (especially tumour), of the orbit, and of various general diseases in which the nervous system takes part. (See Disturbances of Vision, p. 550.)

#### PAPULAR ERUPTIONS. See Skin Eruptions, p. 369.

#### PARÆSTHESIA (Gr. παρά, amiss; αἴσθησις, sensation).

An illusory sensation affecting the perception of tactile sensation as a rule, though pain, cold, or heat sensation may be simulated. The patient may feel a variety of abnormal sensations in the affected region—e.g., tingling, numbness, prickling, cotton-

wool feeling, burning, coldness, a feeling as if insects were crawling over the part (formication), etc. They are most commonly the result of irritation of the nerves at the sensory roots, or nearer the periphery. The lesion may, however, be at any part of the sensory path.

**PARAGEUSIA** (Gr. *παρά*, amiss; *γεῦσις*, the sense of taste).

Perversion of the sense of taste is commonly the result of disease of the mouth, digestive disturbances, or the administration of drugs. It may, however, be a symptom of nervous disorder; thus it is a not infrequent form of the aura of epilepsy. It is often observed in mental affections, and occasionally in hysteria.

### PARAGRAPHIA.

Imperfection in the act of writing, often shown by the omission of words, or by the use of inappropriate words, or by the misplacement of words in the written sentence. It is an evidence of aphasia, due to lesion of the cortical speech centres, or of the nerve fibres in connection with them. (See Disorders of Speech, p. 379.)

### PARALYSIS.

Loss of power in any portion of the body, due to disease of the nervous system, is known as paralysis. If the weakness is not considerable, the term **paresis** is used.

In studying such conditions, one has first to inquire into the condition or tone of the muscle—that is, are we dealing with a **flaccid** or a **tonic** paralysis? Secondly, the situation of the paralysed muscles requires investigation. Is the affection monoplegia, hemiplegia, or paraplegia?

The consideration of the various aspects of this large subject will be found at p. 207 *et seq.*

**PARAPHASIA** (Gr. *παρά*, amiss; *φάσις*, speech).

A disorder of speech, in which the wrong word may be used, or a word may be omitted or displaced to an incorrect position in the sentence. The condition is a form of aphasia, and is due to cerebral lesion affecting the cortical centres of speech and their communicating association fibres. (See Disorders of Speech, p. 379.)

**PARAPLEGIA.**

Lesions of the spinal cord which are sufficient to interrupt the motor tract, cause paralysis of the regions supplied by nerves emerging from the cord at a lower level than the lesion. As a rule the paralysis is bilateral, and is accompanied by anæsthesia and other sensory disturbances. Transverse lesions of the dorsal or lumbar cord affect the legs alone (**paraplegia cruralis**). Lesions at or above the level of the cervical enlargement are less frequently observed, and result in paralysis of all four limbs (**paraplegia totalis** or **paraplegia brachialis**) (see p. 243).

**PARASTERNAL LINE.**

A vertical line drawn on each side of the front of the chest, midway between the side-sternal and mammillary lines. It is used in referring to the topography of the chest. (See Thorax, Shape, etc., p. 464.)

**PARESIS** (Gr. *πάρεσις*, a slackening of strength).

A slight loss of power due to disease of the central or peripheral nervous system is termed paresis (see p. 208).

**PARKINSON'S MASK.**

The dull, expressionless features of a person suffering from paralysis agitans (Parkinson's disease). (See Fig. 48, p. 292.)

**PAROSMIA** (Gr. *παρά*, amiss; *ὄσμή*, smell).

Perversion of the sense of smell is not infrequently observed in mental affections, and (rarely) after head injuries. The aura of epilepsy may assume this form.

**PARROT'S NODES.**

Enlargement of the bony prominences of the frontal and parietal bones, seen in rickety children, especially when combined with syphilis. (See Caput Quadratum, p. 98.)

**PEA-SOUP DIARRHŒA.**

The term is applied to a type of diarrhœa which is characteristic of typhoid fever, and may be found in other forms of intestinal irritation, especially when the diet is mainly milk. The consistence and colour of the stool give rise to the name. (See Fæces, p. 138.)

**PECTORILOQUY** (L. *pectus*, the chest; *loquor*, to speak).

An abnormally clear and articulate quality of the spoken or whispered voice as heard by the stethoscope placed upon the surface of the thorax. An unusually perfect conduction of sound from the larynx (consolidation with adjacent air cavity, superficial phthisical cavity, etc.) is the cause (see p. 415).

**PENDULUM RHYTHM.**

An alteration in the relative duration of the pauses which separate the two sounds sometimes results in the loss of the accent normally placed upon one or other sound. The result is an even series of sounds, such as might be produced by a pendulum swinging truly—the normal rhythm being that of a badly-hung pendulum. The pendulum rhythm may be observed at times in a strongly acting heart with a high arterial tension (*e.g.*, in nephritis), and is probably due to prolongation of the 'closure-time' of the heart, whereby the second sound is delayed.

**PENTOSURIA.**

The presence of pentoses in the urine may at times give rise to error in the search for sugar. The method of detecting them resembles that of glucose. They respond to the reduction tests (Fehling's, etc.), and to the phenylhydrazine test, but they do not ferment. For Bial's orcin test, see p. 537. Their clinical significance is indicated in the article on Urinary Abnormalities (p. 524).

**PERCUSSION SOUNDS : General Principles.**

Resonance, tympanitic and subtympaonic—Pulmonary resonance  
—Resistance—Immediate percussion—Mediate percussion—  
Superficial percussion—Deep percussion—Instrumental percus-  
sion—Orthopercussion.

On striking any portion of the body a sound of some sort is produced. It may be a toneless noise, such as is evoked on striking the fleshy part of the thigh, in which there is but little elasticity. A rather more distinct note is elicited by a stroke upon some bony region, as the tibia or the skull, in which case the bone in question acts as a resounding medium or 'sounding-board,' reinforcing the vibrations in the part struck by sympathetic amplification of the movements in the manner so familiar in our everyday experience of acoustics. The best resounding

medium in our bodies is air, and by striking a region which is adjacent to an air-containing cavity we get a vastly improved note, which may present a greater or less degree of musical quality, or **resonance**, and in which we recognize pitch, duration, and other qualities of musical tone. The clearest or most resonant note is obtained by percussing over a cavity containing a considerable quantity of air under moderate pressure; thus percussion over the stomach or over a pleural cavity containing air (pneumothorax) produces a very clear note of low pitch, which is termed **tympanitic**. A higher-pitched tympanitic note is heard over the intestine, and a somewhat similar sound may occasionally be elicited by percussing over a cavity in the lung. Over healthy lung-tissue a so-called **subtympanitic** note is evoked by percussion, in which the resonance and duration are less marked and the pitch is higher than in the tympanitic sound. This sound is spoken of as the **pulmonary resonance**, and may be modified by (a) the quantity of the air-containing tissues set in vibration by the stroke; (b) the size of the air cavities, and the tension and elasticity of their walls; (c) the conducting quality as to vibrations, and the quantity of the material interposed between the air cavities and the surface of the chest. It is thus obvious that the percussion sounds must vary greatly in the different regions of the chest and abdomen, in both healthy and diseased conditions.

In addition to the sound elicited by the stroke, percussion furnishes occasionally another item of instruction, the sensation imparted to the pleximeter finger—that is, the finger which receives the stroke. The elasticity of the tissues under the spot percussed may be so distinctly reduced as to give to the finger a sense of **resistance** which, once perceived, is easily recognized. This is often observed in cases of pleural effusion.

Constant practice at the bedside is the only way to obtain facility in the use of this valuable means of investigation, and brief reference to the methods employed is all that is called for in this place.

Percussion of the clavicles and the portion of lung over which they are placed is commonly effected by striking the bone directly with the tips of one or two fingers of the right hand. This is **immediate percussion**, which was the original method introduced by Auenbrugger, but which is now practically restricted to percussion over the clavicles. Occasionally a stroke is delivered directly with the tips of several fingers, in order to form a general idea as to the resonance of a large area of the thorax or abdomen; but this procedure is to be discountenanced,

as it encourages laxity in methods of examination. The universally suitable method is that of **mediate percussion**, which is best carried out by striking the first or second finger of the left hand laid, palmar surface downward, on the spot under examination (the **pleximeter** finger). The blow is given by the right middle finger (the **pleissor** finger), so bent that the tip, just in front of the well-pared nail comes squarely down on the dorsum of the pleximeter finger, which rests lightly but immovably on the selected spot. The stroke must be made with a sudden or 'staccato' action, mainly from the wrist, the hand rebounding immediately the blow is delivered. Any dwelling of the pleissor finger on the pleximeter at the end of the stroke would result in a deadening of the note. In case a very light stroke is required, the movement should be almost entirely from the metacarpo-phalangeal joint. Unless the observer has taken pains to acquire a certain degree of dexterity in the execution of this little manœuvre, the resulting note will never be quite satisfactory, and the distinction between doubtfully clear and dull areas will be correspondingly difficult.

As the quality of resonance depends on the sympathetic vibration of adjacent elastic and vibratory structures (which practically means air-containing structures), the *force* of the stroke is of the utmost importance. A gentle stroke will only cause vibrations of a small or weak character, hence the sound will be feeble; a more forcible blow will produce more ample sound-waves and a louder note. It may also be assumed that a forcible stroke sets in movement a more extensive and deeper bulk of resounding tissue (*e.g.*, lung, bowel, etc.) than that affected by a gentle stroke. It will be seen by the accompanying diagrams that, in order to ascertain the boundaries of adjacent solid and gas-containing structures, it is necessary to make use of very gentle percussion (superficial percussion). By employing greater force in the stroke (deep percussion) we lose the power to distinguish the boundaries of organs, but, on the other hand, we gain the power to investigate the character of the deeper structures. Thus we may, by deep percussion, recognize the presence of a solid tumour or organ underlying a superficial air-containing tissue (*e.g.*, a deeply-situated consolidation of the lung, or the inflammatory exudate in appendicitis); or, what is perhaps of less clinical importance, we may note the presence of air-containing tissue below superficial solid structures (*e.g.*, bowel or stomach behind the thin border of the liver).

In comparing the percussion sound in regions which should

normally give a similar note, we must be careful to percuss as nearly as possible over similar structures. Thus, in comparing the right with the left infraclavicular region, one should not place the pleximeter finger on a rib on one side, and over an intercostal space on the other. In both regions interspaces alone, or ribs alone, should be percussed. As a general rule, it will be

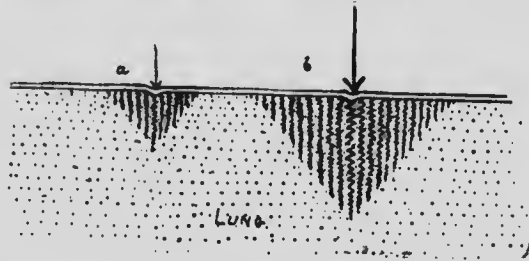


FIG. 39.—DIAGRAMMATIC REPRESENTATION OF LIGHT AND FORCIBLE PERCUSSION.

- a*, Light percussion sets in vibratory motion only a small depth and area of lung. *b*, Strong or deep percussion sets in motion a larger bulk of lung.

found that better results are obtained by restricting percussion as much as possible to the intercostal spaces. The effects of posture must also be borne in mind. For example, when the stomach contains fluid, percussion over that organ while the patient is in the recumbent position will produce different results from those elicited with the patient in the sitting posture. The pleximeter finger must not be too firmly pressed against the surface, as by



FIG. 40.—DELIMITATION OF LUNG BY LIGHT PERCUSSION.

- a*, Light percussion over liver (or other non-resounding structure) adjacent to edge of lung produces an absolutely dull note. *b*, Light percussion over thin edge of lung gives a perfectly clear note.

so doing the vibrations are checked, and an inaccurate estimate of the resonance is the consequence.

Instruments to take the place of the fingers in percussion are used by some clinicians, either with the object of augmenting the sounds, so that they might be heard by a class of bystanders, or from inability to use the fingers with the necessary skill. The



former reason is fallacious, as, in order to render the sounds audible at a distance an unduly forcible stroke is given, and the definition of boundaries of air-containing tissues is quite impossible. There may be conditions in which one of the many forms of mechanical pleximeters is of service, and it is therefore desirable

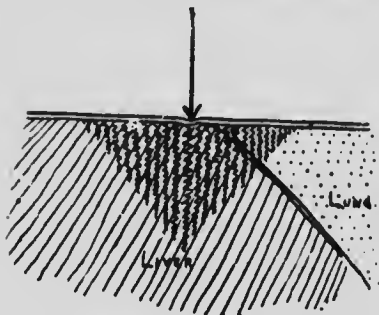


FIG. 41.—Strong percussion over liver close to edge of lung (same spot as *a*, Fig. 40) causes a rather dull note, with a certain amount of added resonance.

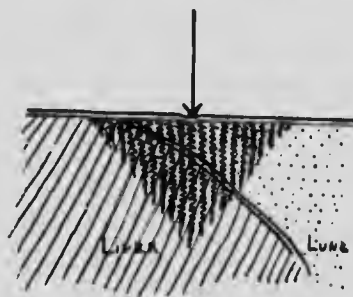


FIG. 42.—Strong percussion over thin edge of lung (same spot as *b*, Fig. 40) gives a relatively dull note not easily distinguishable from that of Fig. 41.

that one should be familiar with their use; but, generally speaking, more accurate and more fruitful results are obtained by employing the fingers alone in percussion.

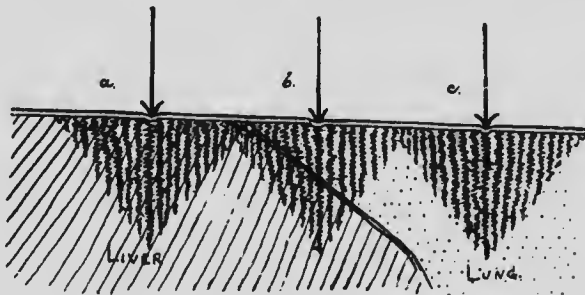


FIG. 43.—STRONG PERCUSSION. *a*, Strong percussion over liver at a spot distant from the lung gives absolute dulness. *b*, Strong percussion over edge of lung (same as Fig. 42) gives a relatively dull note (a dulled resonance). *c*, Strong percussion over lung at a spot distant from non-resounding tissues gives an absolutely clear sound (the pulmonary subtympanic resonance).

**Orthopercussion.**—A method of percussion has been recently advocated by Goldscheider as especially efficacious in determining the size and position of the heart. He claims for it that boundaries, not only of the heart, but also of the great vessels at

its base, and the outline of the diaphragm, can be accurately delineated on the chest surface, and he has found that the boundaries thus obtained correspond fairly accurately to those obtained by Morritz's orthodiagraphic method of radioscopy (see p. 267) Goldscheider's method has been termed **orthopercussion**, and is as

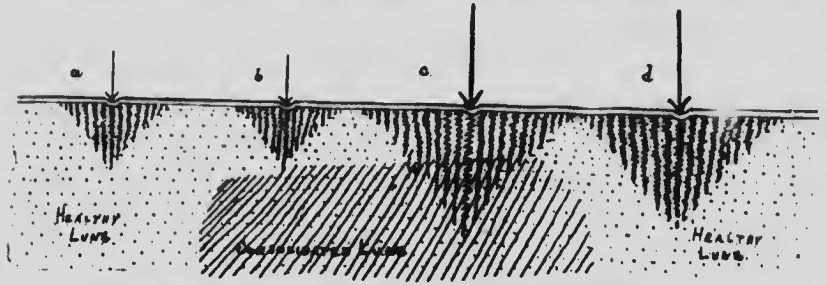


FIG. 44.—LIGHT AND STRONG PERCUSSION OVER A DEEPLY-SEATED PATCH OF CONSOLIDATION OF THE LUNG.

- a*, Light percussion over healthy lung gives normal clear sound. *b*, Light percussion over consolidation, with intervening layer of healthy lung, elicits normal resonance. *c*, Strong percussion causes relative dulness. *d*, Strong percussion over normal lung gives the clear resonance of lung-tissue.

follows: The lightest possible stroke of the percussing finger is employed. The pleximeter consists of the left forefinger bent as nearly as possible in a right angle at the distal joint; the tip only of the pleximeter finger is placed on the chest, and is only placed over the intercostal spaces, the ribs, costal cartilages, and sternum

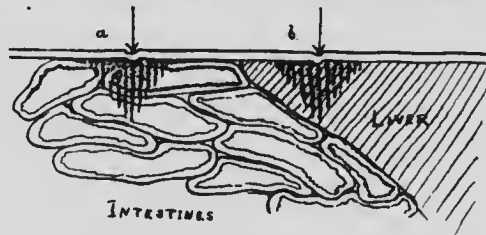


FIG. 45.—LIGHT PERCUSSION OVER LIVER AND BOWEL.

- a*, Light percussion over bowel gives the normal tympanitic note. *b*, Light percussion over thin edge of liver gives absolute dulness.

being neglected. The stroke of the plessor finger is always directed sagittally—that is, strictly in an antero-posterior direction, and not necessarily perpendicularly to the surface, as is usually done. Percussion is commenced in the resonant lateral portions of the chest, the interspaces being followed in toward the sternum.

Only sufficient force is used to elicit a faint sound, which can be heard by listening attentively while perfect silence is maintained in the room. As soon as a point is reached behind which the heart or great vessels lie, even if separated by a layer of air-containing lung, the faint percussion sound is said to disappear. The advantages of this method are stated by Curschmann to be: (1) The small area of chest-wall in contact with the pleximeter, the lateral dispersion of the vibrations being thereby minimized; (2) the projection of the boundaries of the subjacent solid organs

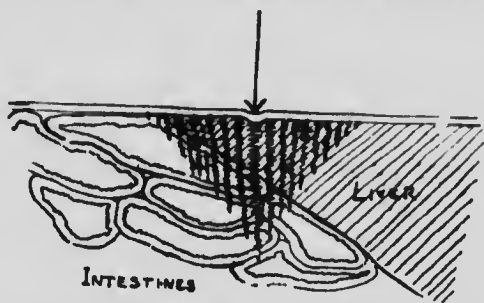


FIG. 45.—HEAVY PERCUSSION OVER LIVER AND BOWEL.

Strong percussion over thin edge of liver produces a certain degree of resonance from the underlying air-containing intestine; this makes it impossible to distinguish the edge of the liver by strong percussion.

on the surface of the chest is true, owing to the sagittal direction of the stroke, unlike that obtained by perpendicular percussion; (3) gentle percussion over the intercostal spaces is sufficient to elicit resonance over the mass of lung-tissue, which resonance is extinguished by the underlying solid organ. The lightness of the stroke has the advantage of minimizing (like the small area of the pleximeter) the lateral dispersion of the vibrations and the consequent indistinctness of outline of the heart.

#### PERISTALSIS, Visible.

The vermicular contractions of the intestine or stomach may under certain circumstances become visible. The diagnostic significance of this occurrence is referred to at pp. 13 and 392.

**PERLES OF LÄNNEC.** See *Sputum*, p. 387.

#### PHARYNGEAL REFLEX.

This consists in the choking and retching on tickling the fauces. It is, like the other superficial reflexes, much better

marked in some individuals than others, but is rarely quite absent in health. It disappears in bulbar paralysis, and often in hysteria. (See Reflexes, p. 343.)

### PHOSPHATURIA.

Calcium phosphate occurs as an amorphous deposit along with crystalline ammonio-magnesium phosphate in alkaline urine. The crystalline forms may be recognized by means of the microscope as stars, 'coffin-lids,' 'knife-rests,' and feathers. These, as well as the amorphous phosphate of calcium, are dissolved on the addition of dilute acetic acid. Their clinical significance is considered in the article on the Abnormalities of the Urine, at p. 511.

### PHOTOPHOBIA (Gr. φῶς, light; φόβος, dread or intolerance).

An excessive sensitiveness of the eye to the stimulus of light is chiefly found in diseases of the eye itself, and most commonly in affections involving the cornea and conjunctiva. It may occur in meningitis, and especially in the cerebro-spinal form, but is also a symptom of functional disturbance of the nervous system—*e.g.*, hysteria, neurasthenia, and migraine.

### PICA (L. *pica*, the magpie).

A perverted or insatiable appetite, in the satisfaction of which the subject will eat unwholesome or even disgusting substances, and often in excessive quantity. The symptom is occasionally seen in pregnancy, in chlorosis, in hysteria, in idiocy, and in dementia. (See Appetite, p. 36.)

### PIGEON-BREAST.

A malformation of the chest, the result of disease in childhood. The sternum and anterior part of the thorax project forward in a narrow, keel-like ridge. (See Thorax, Shape, etc., p. 467.)

### PIGMENTATION OF THE SKIN.

The amount of pigment in the skin is so variable in healthy individuals that only extreme conditions of pigmentation can be termed pathological. The darkening of the areolæ round the nipples and of the lineæ alba in pregnancy is a constant feature of

that state, in which one also observes patches of discoloration on the face and elsewhere, which are known as the **chloasma uterinum** (**masque des femmes enceintes**).

Irritation of the skin from various causes may cause the abnormal deposition of pigment in the skin which may in some cases be permanent—*e.g.*, **vagabond's disease**, due to irritation of dirt, vermin, and scratching. Mustard-plasters and fly-blisters may leave a similar permanent mark.

A **bronzed colour**, which may give a greyish tinge, is seen in **Addison's disease**. Those parts of the skin where pigment is normally deposited are first affected—*e.g.*, the face, axillæ, neck (where the collar has pressed). It extends and grows deeper in colour, through bronze to deep brown, with darker spots scattered over the discoloured surface. The mucous membranes may also be involved, patches being commonly seen inside the mouth. The palms of the hands, the soles of the feet, and the nails escape.

In **phthisis** it is not uncommon to observe a considerable pigmentation of the face and body generally.

**Cirrhosis of the liver** sometimes causes a pigmented or bronzed condition of the skin, which may be distinguished from the tinge of jaundice by the absence of bile from the urine.

A condition known as **diabète bronzée** (bronzed diabetes) is recognized by bronze-coloured pigmentation of the skin, in conjunction with hepatic cirrhosis and diabetes.

In **Hodgkin's disease** the skin is frequently deeply pigmented to a bronze colour.

The prolonged administration of **arsenic** may cause pigmentation closely resembling that of Addison's disease (arsenical melanosis).

The persistent ingestion of **silver salts** (a rare occurrence in modern therapeutics) produces a greyish or even a blue discoloration of the skin and mucous membranes, as well as of the internal organs (**argyria**), which may resemble Addison's disease. The general symptoms of that disease are, however, absent, and the nails share in the discoloration.

**Melanotic sarcoma** may be accompanied by a widespread discoloration of the skin, a dusky grey or even black surface.

Certain skin diseases are characterized by pigmentation. **Syphilitic eruptions** are apt to assume a coloured or dark hue and the cicatrices of healed syphilitic sores are often deeply pigmented.

mented. **Pigmented nævi, pigmented xeroderma,** and other pigmentary abnormalities, are to be observed in skin affections, for descriptions of which see special treatises on dermatology.

**Jaundice** (see p. 169) causes a yellow discoloration due to bile pigments.

### PLANTAR REFLEX.

In normal cases tickling the sole of the foot causes the toes to be flexed, the foot to be dorsally flexed, the knee and hip to be flexed. When the reflexes are increased from any disease of the nervous system these movements are exaggerated; the reflexes are more widespread, contractions of the trunk and even arm muscles taking part. (See Reflexes, p. 343.)

### PLETHORA (Gr. πληθώρα, fulness).

This term is to some extent ambiguous, as it signifies an increase in the total quantity of blood in the body, and is also used to indicate an excessive number of red cells present in a given quantity of blood. For the last condition the term **polycythæmia** is a more accurate designation, but this state of the blood is very commonly spoken of as plethora.

Before the introduction of modern methods of blood examination much attention was paid to the supposed quantity of blood possessed by the patient, and a considerable edifice of loose pathology was built on the habit of the body. It has been shown by experimental physiologists that an artificial increase in the quantity of blood cannot be maintained for any length of time, but there can be no doubt that variations in the bulk of blood do occur, and their clinical significance is of some importance. An increase in the total quantity of blood, with a corresponding reduction in its proportionate cell contents (hædræmic plethora), may be seen in certain anæmic conditions, notably in chlorosis. The researches of Haldane and Lorrain Smith have demonstrated the increased quantity of blood in cases of chlorosis. (See Blood Examination, p. 88.) An increase both in the quantity and in the cell richness of the blood may be seen in persons of great muscular development, and is generally found in individuals whose surroundings and mode of life make for health and energy.

An increase in the number of red corpuscles per cubic millimetre of blood (polycythæmia) is almost invariably an evidence

of the reduction of the quantity of fluid in the bloodvessels, rather than an actual increase of the number of red cells in the body. One expects, therefore, that the removal of fluid from the body, or a restriction of its ingestion, would be followed by a raised blood-count, and this is in accordance with clinical experience. A moderate polycythæmia is found after profuse diarrhoea and vomiting, and this is more marked in cholera.

In heart affections polycythæmia is commonly observed, and may be attributed to two causes, œdema and venous stasis. The first of these, if considerable, will reduce the bulk of blood in the vessels, while the second will favour the accumulation of an undue proportion of the solid elements of the blood in the capillaries and venules from which the blood is obtained for examination.

Polycythæmia has been noted after prolonged cold baths and in cases of coal-gas poisoning. In both conditions the cause is probably venous stasis.

A polycythæmia of less obvious causation, but probably the result of lowered atmospheric pressure, is that observed at high altitudes.

**PLEUROSTHOTOS** (Gr. πλευρόθεν, from the side; τόνος, a stretching).

A condition of tonic spasm of the trunk muscles causing the body to be curved toward one side. It occurs in some cases of tetanus.

**POIKILOCYTOSIS** (Gr. ποικίλος, varied; κύτος, a hollow vessel).

In severe degrees of anæmia, and particularly in pernicious anæmia, the red corpuscles are found to present irregular shapes. Instead of the disc of normal size (about  $\frac{1}{3300}$  inch in diameter, or 7 to 8 microns), varied shapes and forms are seen—pear-shapes, dumb-bells, wedges—and many irregularities occur. Abnormalities in size are known as microcytes, megalocytes, etc. (See Blood Examination, p. 66.)

**POLYCHROMASIA** (Polychromatophilic or Anæmic Degeneration).

Certain anomalies in the staining capacity of red cells in cases of chronic anæmia occur. A blood-film stained with eosin-hæmatoxylin or eosin-methylene-blue shows in normal cases the

red corpuscles coloured eosin-red. In severe anæmias these cells are tinged in various shades between the normal colour and blue or violet. This is regarded by Ehrlich as a sign of degeneration, but some observers regard these cells as the immature or imperfectly developed erythrocytes. (See Blood Examination, p. 67.)

**POLYCYTHÆMIA** (Gr. *πολύς*, many; *κίτος*, a hollow; *αἷμα*, blood).

A condition in which the number of red blood-corpuscles per cubic millimetre exceeds the normal count of about 5,000,000. It is usually the result of a decrease in the total quantity of fluid in the vessels or of venous stasis. (See Plethora, p. 288.)

**POLYURIA** (Gr. *πολύς*, much; *οὔρον*, urine).

The normal amount of about 50 ounces of urine secreted daily by healthy adult kidneys may be considerably increased. This is characteristic of diabetes mellitus and insipidus, of the small red or contracted kidney, and other renal affections. It is also seen in hysteria, and as the result of the administration of certain drugs. Physiological causes may in some cases suffice to produce a copious secretion of urine (see p. 502).

**POSTERIOR AXILLARY LINE.**

A vertical line drawn on the side of the thorax, through the spot where the posterior axillary fold joins the chest-wall, the arm being held out horizontally. It is used in referring to the topography of the chest (see p. 464).

**POSTURE.**

The position assumed by the patient, whether in bed, seated, or standing ('station'), may in some cases convey information as to his condition.

1. **He may lie on his side.** The limbs are flexed at every joint, and he resents any attempt at change of position or at interference of any description. The condition is that of cerebral irritation, and is the result of intracranial lesions of an irritative or inflammatory nature—*e.g.*, meningitis, hæmorrhage, pressure. A similar posture with the head retracted, so that the occiput approaches the neck, is characteristic of basal meningitis. In



renal or hepatic colic a similar flexed posture is not infrequently assumed, though in many painful affections of the abdominal organs the patient is more likely to lie on his back or on his face. He lies on his side, and has obvious difficulty in breathing; this may be pleurisy or pneumonia of the side upon which he is lying. In dry pleurisy he breathes rapidly, because the pain obliges him to be content with a shallow type of respiration, and in order to accomplish the necessary amount of oxygenation he must take more breaths in a given time than a healthy man. In pneumonia and in pleural effusion he also lies on his affected side, and his breathing is laboured and hurried. He is not prevented by pain from taking a deep breath (except in the early stages of pneumonia, when pleurisy usually coexists), but he seems in too great a hurry to be able to draw a good breath.

2. **He lies on his back.** A comfortable posture on the back is assumed by a patient who is not seriously weakened by his malady, or is not in pain. An uncomfortable dorsal posture, with a tendency to slip downwards in the bed, is characteristic of conditions of great weakness, such as the typhoid state in acute and wasting diseases. One leg is drawn up, and the abdominal muscles on that side are more or less rigid and resisting: inflammatory affections of one side of the lower abdomen and neighbouring regions—*e.g.*, appendicitis (right side), hip-joint disease, or inflammation of the female pelvic organs. Both legs are drawn up, and the abdominal muscles are more generally rigid: painful and inflammatory affections involving a larger area of the abdominal cavity—*e.g.*, peritonitis, perforation of the bowel or stomach, pelvic cellulitis, extra-uterine gestation, inflammation of the uterus and its appendages, etc. In cases of cerebral hæmorrhage the patient usually lies on his back, with the head and eyes directed to either side (Conjugate Deviation, *q.v.*, p. 241).

3. **He lies on his face.** This attitude is characteristic of painful and usually non-inflammatory affections of the abdominal organs—*e.g.*, renal colic, intestinal colic, gall-stone colic.

4. **He is unable to lie down, and remains in the sitting posture in bed or in a chair.** This attitude is assumed owing to difficulty in breathing whilst in the recumbent posture (*orthopnoea*), and is observed in cases of cardiac disease, asthma, emphysema, mediastinal tumour.

5. **He stands with knees slightly bent and the body inclined forwards.** The thumbs are generally extended, and the fingers

flexed at the metacarpo-phalangeal joints, but with the phalangeal joints extended, while movements are carried on as if bread were being crumbled. The arms are held out slightly from the sides,



FIG. 47.—PARALYSIS AGITANS.

The posture is characteristic: head inclined forward; knees slightly bent; arms bent at the elbow and forearms carried across the abdomen; fingers flexed at metacarpo-phalangeal joints and in 'pill-rolling' position; face immobile and expressionless. The tremor in this case was also characteristic.

the wrists and elbow-joints are a little bent, and the hands are tilted towards the ulnar side, resting on the abdomen at or near

the waist. . . . The head and neck present a striking appearance, being thrown forwards and rigidly fixed, while the features are motionless and devoid of expression' (Roberts). This is a description of the attitude assumed by a patient suffering from paralysis agitans.

6. In tetanus and in strychnine-poisoning a fixed and rigid posture is assumed, owing to the involuntary spasmodic or exaggerated contraction of the muscles of the trunk. If the different groups of body muscles balance and oppose each other equally the body is straight and rigid, the posture being termed **orthotonos**. Should the contraction of the dorsal groups of muscles prevail, the body is curved backwards, and may be seen to rest on the head and the heels, the middle portion of the trunk being raised from the bed. This is the condition known as **opisthotonos**. If the body is curved forwards by the excessive contraction of the recti and other muscles of the front of the trunk, the posture is termed **emprosthotonos**. Lastly, a curving of the body to one side from similar causes is known as **pleurosthotonos**.

### PRÆCORDIAL PULSATION.

Pulsatile movements of the anterior chest-wall are diagnostic signs of great importance. They are discussed seriatim in the article on the Movements of the Thorax (p. 469).

### PRIAPISM (L. *Priapus*, a licentious deity of the Greeks).

Unduly frequent and prolonged erections of the penis may be the result of local irritation—*e.g.*, enlarged prostate, stone in the bladder, an accumulation of urine in the bladder.

From a diagnostic point of view its interest lies chiefly in the fact that it has been observed in some affections of the nervous system. In these cases it is probable that the centre in the lumbar cord which presides over erection is not only uninjured, but is in a state of reflex excitability without the inhibiting influence of the higher centres, from which the spinal centre has been isolated by the lesion. Priapism has been observed in myelitis, cerebral injuries—*e.g.*, hæmorrhage—and occasionally in locomotor ataxia. In the last-named affection, however, loss of sexual power is more common. For reasons which have not been satisfactorily explained, priapism has been noted in a number of cases of leukæmia.

**PROPULSION (Festination).** See **Gait**, p. 150.

**PRUNE-JUICE SPUTUM.** See **Sputum**, p. 387, and **Hæmoptysis**, p. 157.

**PRURITUS.** See **Itching**, p. 169.

**PSEUDO-ATAXIC GAIT.**

In cases of peripheral neuritis which impair the strength of the muscles of the legs below the knee, and especially those in front of the limb, the gait is a mixture of inco-ordination and paresis, and differs in some respects from the true ataxic gait. The condition is considered in the article on **Gait** (p. 150).

**PSEUDOCYESIS** (Gr. *ψευδής*, false; *κύσις*, pregnancy).

An abdominal tumour may be simulated in hysterical patients by means of flatulent distension of the viscera, together with an arching forward of the lumbar vertebræ (lordosis) and a lowering of the diaphragm. The result is a tympanitic swelling of the abdomen (phantom tumour), which disappears on the administration of an anæsthetic. In such cases it is not unusual for some of the other symptoms of pregnancy to appear—*e.g.*, morning-sickness and swelling of the breasts.

**PTERYGOID CHEST.** See **Thorax, Shape**, etc. p. 468.

**PTOSIS** (Gr. *πίπτω*, to fall down).

A drooping of the upper eyelid due to paralysis of the levator palpebræ superioris (see p. 216).

**PTYALISM (Salivation).**

An excessive quantity of saliva is secreted in the following conditions: Mercury, when administered for too long a period or in too large doses, is a familiar cause of salivation; pilocarpine and the compounds of iodine; inflammation of all descriptions in the mouth; hysteria and other neurotic conditions; pregnancy and hydrophobia may be mentioned among the more important causes.

In facial paralysis, diphtheritic paralysis, and bulbar paralysis, and also in idiocy, there is apparent salivation, but it is really a

mere escape of the fluid from the mouth owing to weakness of the lips and cheek muscles. There may, however, be also an actual increase of the secretion in bulbar paralysis, a possible instance of paralytic secretion.

### PUERILE BREATHING.

An increased loudness of the breath-sounds due to excessive activity of the respiration may be perceived. The exaggerated respiratory efforts may be limited to one lung, in which case they are usually of a vicarious nature, the other lung for some reason being unable to perform its full share of work. In this case the breath-sounds are described as puerile breathing, as they have the energetic quality of those heard over the thin-walled chests of youths. It is to be noted that the increased respiratory sound commonly takes the form of bronchial breathing, rather than a louder vesicular breathing. The latter, however, does occur, and is probably to be explained by the rapidity of the currents in the overworked section of lung setting up audible vibrations in bronchioles which under normal circumstances would be silent. The subject of breath-sounds is generally considered in the article on Auscultation of the Thorax, at p. 408.

### PULMONARY AREA.

This term is applied to that portion of the chest-wall in the immediate vicinity of the second left costal cartilage. In this region those sounds which are generated at the cardiac orifice of the pulmonary artery are best heard (see p. 437).

### PULMONARY REGION.

That portion of the chest-wall which is in contact with the lungs is known as the pulmonary region. It extends from about  $1\frac{1}{2}$  inches above each clavicle downwards in front to the sixth rib in the nipple line, to the eighth or ninth rib in the mid-axillary line, and to the tenth or eleventh rib in the scapular line behind. On the left side the lung usually extends downward a little further than on the right, and the vertical anterior border of the left leaves that of the right at the level of the fourth costal cartilage, receding to the apex-beat, where it joins the horizontal anterior border. These boundaries refer to the condition of the chest in quiet breathing; forcible breathing alters considerably the out-

lines of the lungs. In the mid-axillary line the resonance during full inspiration may extend 2 inches lower than during forced expiration (see p. 451).

The surface of the thorax is subdivided again, for purposes of description, into a number of areas or regions, the majority of which overlie the lungs, and are therefore constituent parts of the pulmonary region. Several of them, however, are in relation to the abdominal cavity. A short description of these areas, twenty-seven in number, will be found at p. 465, and a further reference to the various qualities of sound elicited in the different regions by percussion is made at p. 451.

**PULSE, Alternating (Pulsus Alternans).** See **Arterial Pulse**, p. 310.

**PULSE, Anacrotic.**

A type of pulse curve found in cases of high arterial tension in which the percussion-wave does not reach to the apex of the curve. (See **Arterial Pulse**, p. 303.)

**PULSE, Arterial.**

The object of examining the pulse—Method of examination—Unequal radial pulses—Movable pulse—Tension—Conditions affecting tension.

Tension in various morbid states: Excessive cardiac activity; removal of blood from the arteries; alcoholic stimulation; collapse; fevers; arterio-sclerosis; kidney affections; valvular disease of the heart—The sphygmometer.

The sphygmograph—Sphygmographic tracings—The condition of the walls of the artery—Causes of thickened arteries.

The frequency of the pulse—Causes of slight modifications of the pulse-rate—Tachycardia: its causes—Pulse-temperature rate—Bradycardia: its causes—Stokes-Adams disease.

The volume of the pulse—Pulsus parvus: in conditions of high tension; in conditions of low tension—Pulsus magnus: in the strongly acting heart; in dilatation of the arterioles and capillaries; in aortic incompetence—Corrigan's pulse, pulsus celer, collapsing pulse.

Pulsus alternans—Pulsus paradoxus.

The duration of the pulse: pulsus celer, pulsus tardus.

Information on the following subjects may be obtained by an examination of the arterial pulse: (a) The degree of tension in the systemic arteries; (b) the condition of the arterial walls; (c) the frequency and rhythm of the ventricular contractions;

(*d*) the volume; and (*e*) the duration of the pulse. The information thus acquired, together with that resulting from our examination of the heart, kidneys, and other organs, is of the utmost diagnostic value.

The radial pulse is usually the most convenient, though at times other arteries are selected. Thus the facial or the anterior or superficial temporal may be conveniently palpated by the anæsthetist during the progress of an operation. The carotids may be chosen instead of the apex of the heart to mark the period of ventricular systole, the interval between the carotid pulse and that of the apex being inappreciable to the finger (less than one-twentieth of a second, while the radial is one-sixth of a second later, a distinctly appreciable interval).

The best means of investigating the pulse is by palpation with three fingers—the distal finger to prevent the pulsation of a reflux or anastomotic wave, the proximal finger to cause obliteration of the pulse by pressure, and the middle finger to observe the character of the pulse-wave. By this means the degree of arterial tension can be fairly accurately ascertained by the educated finger. Transverse and longitudinal rubbing movements detect abnormal hardness of the vessel's walls, which should be impalpable between the pulse-waves.

**Unequal Radial Pulses.**—A comparison between the two radials should generally be made. Inequality of the pulses is commonly due to want of symmetry in the distribution of the arteries to each hand. It may, however, result from disease of the vessels, or mechanical obstruction to the arterial circulation. Thus an aneurismal dilatation of the vessel supplying the arm at any part of its course after it leaves the aorta, or interference to the blood-current in a similar situation from pressure of a tumour, thrombotic or embolic plugging, rupture or other injury to the vessel, will diminish or abolish the radial pulse on the affected side.

The pulse may also be examined by **inspection**. Visible pulsation of the radials denotes a greater amplitude of the wave than is found in health as a rule. If the arteries are tortuous and the pulsations considerable, a condition which may occur in aortic incompetence, the artery may be seen to be actually displaced with each beat, constituting the so-called **movable** or **locomotive** pulse. Tortuosity of the artery, without actual movement of the vessel, is usually a sign of thickening and degeneration

of the arterial wall. The temporal artery, however, may be visibly tortuous, though the artery is healthy.

The arterial pulse is also investigated by means of the sphygmograph, the sphygmanometer, and the stethoscope (see below).

(a) **Tension.**—As stated above, the arterial tension may be observed by the educated finger placed upon the radial artery. As a rule this method suffices for clinical purposes, and it has much to recommend it over the use of instruments for the same purpose. Three finger-tips should be placed over the vessel—the distal one firmly pressed to prevent reflux pulsation, while the middle finger is used to observe the effect upon the pulsation of increasing pressure exerted by the proximal finger. In cases of high arterial tension the pulse is only obliterated by considerable force, while with low arterial tension a moderate digital pressure suffices to abolish the pulse. Careful comparisons of low-tension pulses show that an ample pulsation-wave (such as may be observed in the low-tension pulse of fevers) is more difficult to obliterate than a smaller low-tension pulse. In other words, the **systolic** tension of the former may be high, while the **mean** or **diastolic** tension may be low.

It is often desirable to preserve a record of the arterial tension more accurately than can be effected by noting one's impressions from digital examination. Here the sphygmanometer (see p. 300) is of some value, as a daily chart can be formed, showing more or less accurately the mean arterial tension in millimetres of mercury. By means of the sphygmograph one may also record the degree of tension (see p. 301). A low-tension tracing shows a vertical up-stroke, a sharp summit wave, and a well-marked dicrotic wave, while the tidal or elasticity waves are ill-marked or absent. A pulse of high tension produces a more oblique up-stroke, a blunt summit wave and more gradual fall; the elasticity waves are well and the dicrotic wave is ill marked.

The average mean normal arterial tension of adults, as tested by the sphygmanometer, may be stated at about 120 mm., and may vary 10 to 20 mm. above or below this pressure. It depends on (a) the strength of the ventricular contractions, (b) the volume of blood in the arteries, (c) the peripheral resistance, and (d) the elasticity of the arterial walls. Any departure from the normal of one or more of these factors will alter the arterial tension. Increased cardiac activity from exercise or emotions raises the



tension, while rest has the contrary effect. The removal of blood from the arteries, either by its transference to the abdominal veins (a common result of reflex irritation) or by hæmorrhage, lowers the pressure if the other conditions are undisturbed. In hæmorrhage, however, the pressure is usually maintained up to a certain point by contraction of the peripheral vessels, the resistance being thus increased. The increased cardiac activity resulting from alcohol is largely neutralized by relaxation of the peripheral vessels. In shock and collapse there is vaso-motor paralysis, which lowers the peripheral resistance, together with transference of blood from the arteries to the vessels of the splanchnic area; hence a marked and dangerous low tension results. In fevers the diminished peripheral resistance lowers the tension, in spite of the frequent and usually excited cardiac action. Excessive elasticity of the arterial walls is seen in infancy and childhood, when the tension is low. Diminished elasticity occurs in arterio-sclerosis, with a consequent rise of arterial pressure. In kidney affections, and especially in chronic renal sclerosis, the increased peripheral resistance resulting from contracted, and sometimes sclerotic, arteries, as well as cardiac hypertrophy, cause in most cases an increase in the arterial tension. In such cases the artery seems to fill slowly and against strong opposition (*pulsus tardus*; see below). Cardiac cases show more variety. Loss of compensation almost always lowers the tension, but if combined with arterio-sclerosis a fairly high tension may be maintained. Valvular lesions with compensatory hypertrophy of the ventricles are also as a rule accompanied by low-tension pulse, in spite of the strength of the ventricular contraction, the systemic arteries being kept in relative slackness by the valvular defect (mitral incompetence, aortic obstruction or incompetence). In the case of aortic incompetence the powerfully contracting left ventricle causes a momentary systolic high tension (*pulsus celer*), followed by mean and diastolic low tension, the so-called *collapsing, water-hammer, or Corrigan's pulse*. When this lesion is accompanied by mitral disease or by aortic obstruction, the systolic high tension is to some extent abolished. A decrease in the considerable difference which usually exists between systolic and diastolic tension in aortic incompetence is therefore a fair measure of the degree in which this lesion is complicated by other valvular affections. In mitral obstruction, whether pure or accompanied by other valvular lesions, the

amount of blood which reaches the arteries with each beat is diminished, and the pulse is, in consequence, of low tension.

**The Sphygmanometer.**—In order to arrive at a more accurate estimate of the arterial tension than can be accomplished by digital examination, the sphygmanometer has of late been found useful. Several forms of the instrument have been devised, the principle in all being a record of the amount of pressure registered by a manometer which is found necessary to abolish the circulation in the parts distal to the obstruction.

Riva-Rocci's instrument is perhaps the most serviceable. It consists of a rubber sleeve forming an air-tight bag, and covered by an inextensible leather or canvas band. In the original Riva-Rocci instrument this sleeve was too narrow, but as now modified it is about 5 inches in width. It is fitted loosely on the upper arm just above the elbow, the outer cover being adjusted to comfortably encircle the arm. The rubber bag inside is connected by a tube with a pump, by which it can be inflated, and a side-branch from this tube, controlled by a valve, places the inflated armlet in direct communication with a mercury manometer. The patient's arm being supported in an easy position about the level of his heart, air is pumped into the apparatus until the radial pulse is lost. The fingers of the observer being retained on the radial artery, air is allowed to escape gradually by means of a tap for the purpose, and the height at which the pulsations return is carefully noted, and is regarded as the **systolic pressure**. The column of mercury is seen to rise and fall with each heart-beat, and the amplitude of the pulsations must be carefully noted. The pressure is now further reduced by successive small escapes of air, and after a fall of variable amount the mercurial excursions grow distinctly less. At this point the height of the column is again noted, and is taken as the **diastolic tension**. Midway between the two readings is the **mean tension**.

While it cannot be claimed that these results are as accurate as those obtained by intra-arterial observations in the lower animals, they are sufficiently consistent to afford information of some value.

Of other forms of sphygmanometer it is only necessary to mention the original instrument of v. Basch, in which an air-pad pressed on the artery was used instead of the sleeve to obliterate the pulse, and Gärtner's, by means of which the amount of pressure is measured which will just prevent the re-entry of

blood to a finger whence the blood had been previously expressed.

Normally the pressure registered by Riva-Rocci's instrument is about 120 to 140 mm. of mercury. If the observations are to be of any practical value they should be made methodically for a definite length of time once, or better twice, daily, and the results noted in chart form.

**The Sphygmograph.**—By means of this instrument one secures a permanent record of some of the features of the pulse, and in the hands of many clinicians it has proved of real service. It is unnecessary to describe the instrument, many forms of which are in use. In this country Dudgeon's is the favourite. With a little practice in adjusting the pressure of the pad upon the artery, fairly consistent and reliable tracings can be obtained. The method of using the instrument requires no explanation here, but some consideration of the tracings marked by the lever may be useful.

The sudden expansion of the vessel producing the up-stroke is due to a rapidly travelling wave, not to the passage of the blood injected by the ventricle into the already full aorta. The retraction of the vessel by means of its elasticity causes the lever to fall, and so mark the down-stroke. This, however, is a more gradual process than the heart-beat which occasions the up-stroke, and produces a series of elastic recoil waves. The wave causing the up-stroke is known as the **primary** or **percussion wave** (*p*, Fig. 48). Of the series of elevations (katakrotic elevations) forming the down-stroke, the best marked is usually the **dicrotic wave** (*d*), which follows immediately on the closure of the semi-lunar cusps, and is the result of recoil from the valves, causing a further expansion of the vessel's walls. It may often be perceptible to the finger—*e.g.*, in fevers. The elevation preceding this (*t*) is the **tidal**, **secondary**, or **predicrotic wave**, which coincides with the end of the ventricular systole. One or more **postdicrotic waves** may follow the dicrotic elevation. While the dicrotic wave is doubtless the rebound from the closed aortic valves—for it is increased in low tension of the arteries with an active heart-beat—the remaining or secondary elevations are possibly of the same nature, but may be in part the result of oscillations in the instrument.

A pulse of low tension (see Figs. 49 and 50) produces a sharp-pointed percussion elevation, with a steep down-stroke. The

dicrotic wave is distinct, for the recoil wave from the valve will produce a marked effect upon the comparatively relaxed arterial walls. The secondary elevations are ill-defined or absent, owing to the relaxation of the walls and the rapidity with which the



FIG. 48.—PULSE OF LARGE VOLUME.

*b, b, b*, Respiratory or base line; *p*, primary or percussion wave; *s*, secondary, tidal, or predicrotic wave; *d*, dicrotic wave; *a*, dicrotic or aortic notch. (Monro.)

lever falls. When the notch preceding the dicrotic curve reaches the base line the pulse is said to be fully dicrotic. If the dicrotic



FIG. 49.—PULSE OF LOW TENSION (PNEUMONIA).

A fully dicrotic pulse.

wave be excessive, or if it occur so late as to form part of the succeeding upstroke, the pulse is hyperdicrotic. Dicrotism is



FIG. 50.—HYPERDICROTIC PULSE (TYPHOID FEVER).

characteristic of fevers, where the tension is low and the heart's action vigorous.

High arterial tension gives a tracing of a different character (see Fig. 51). The up-stroke is somewhat sloping; the line is sus-

tained at its height, forming a blunt curve, the apex of which may be a plateau, or may even rise higher than the percussion-wave. When the latter condition is found, the pulse is termed **anacrotic**. The dicrotic wave is indefinite or imperceptible, while the secondary waves are increased in amplitude and number.



FIG. 51.—PULSE OF HIGH TENSION (NEPHRITIS).

The comparison of the radial pulse with that of the apex, the carotid, or the jugular vein has yielded information which has been of assistance in elucidating the phenomena of arrhythmia.

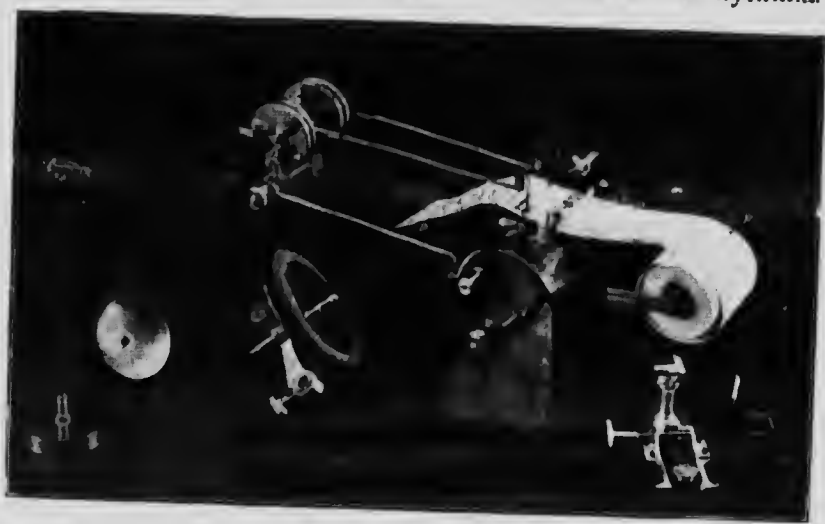


FIG. 52.—MACKENZIE'S INK POLYGRAPH.

The instrument gives three ink tracings of any desired length; they represent the movements of any two pulsating surfaces, and the excursions of a time lever. (S. Shore, Padigham.)

By means of recording tambours the exact moment at which the various acts occur can be ascertained, and pulse-tracings may be rendered more instructive. The sphygmograph has been modified by Mackenzie so as to perform efficiently all that the more elaborate instruments accomplish, while the still further improved

'clinical polygraph' which he introduced at the Toronto meeting of the British Medical Association should render the scientific investigation of abnormalities of the pulse practicable to clinical observers (see Fig. 52).

(b) **The Condition of the Walls of the Artery.**—In healthy individuals the radial artery cannot be felt when the pulsation is abolished by pressure higher up the arm. In elderly subjects, however, the vessel becomes progressively more palpable, owing to senile changes of a degenerative nature. Careful palpation of the artery may show the vessel to be hardened in persons whose age does not justify that condition. In such a case it is probable that the arteries elsewhere in the body have also undergone a change similar to that observed in the radial. On the other hand, the radial may be apparently healthy while degenerative changes exist in some of the other arteries of the body or limbs. The degenerated arteries grow in length as well as in thickness, so that they are thrown into bends and turns (tortuous arteries). The loss of elasticity of the arterial walls under these circumstances causes the arterial tension to be raised (as stated above). It may therefore be difficult in some cases to decide whether the distinctness with which such a radial artery may be felt is to be attributed to disease of its walls or to heightened intra-arterial pressure. If the vessel can be felt after its pulsation is checked by pressure, the walls are thickened.

Premature thickening of the arterial walls is due in the majority of cases to arterio-sclerosis. This condition of vessels is often associated with chronic sclerotic or inflammatory changes in the kidneys, whereby the arterial tension is still more raised. Syphilitic arteries may also cause thickening of the arteries, which is usually observed in younger subjects than arterio-sclerosis. The sphygmographic tracing of arterio-sclerosis resembles in some respects that of the high-tension pulse. The up-stroke is short and inclined to slope upwards, the apex is blunted, and the waves of the down-stroke are ill-marked.

(c) **The Frequency and Rhythm of the Pulse.**—The adult pulse beats normally rather more than once every second. An average rate is from 70 to 75, but very considerable departures from these limits are consistent with health. Any emotion may quicken the pulse; the mere entrance of the physician may add a number of beats, so that it is not advisable to count its frequency immediately one sees the patient. Bodily exercise increases it,

and in sensitive persons a very slight amount of exercise is enough to produce an effect. Thus the pulse may count more in the sitting than in the recumbent position, or in the standing than in either the sitting or lying positions.

A slight increase in the frequency is observed during digestion. A similar effect is produced by coughing or by forced expiratory effort with the glottis closed ('Valsalva's experiment').

The age of the individual has a relation to the frequency of the pulse. In the new-born infant it beats at 120 to 140 per minute; in adult life it is usually 70 to 75, decreasing slightly up to the age of sixty, after which it again increases in frequency.

The average frequency is a few beats more in women than in men of the same age. It is quicker in short than in tall persons. A lessened arterial tension is usually accompanied by a frequent pulse, hence in renal disease the pulse is commonly infrequent, while in all forms of heart affection, with the exception of aortic stenosis, the pulse is frequent as a rule (see Bradycardia, p. 307). In aortic stenosis the obstructed orifice causes the same impediment to the expulsion of the ventricular contents as does high arterial tension, with consequent infrequency of the ventricular contraction.

Individual peculiarities in the pulse-rate must be borne in mind. An abnormally slow pulse may be natural to a perfectly healthy man; a pulse as slow as 40 may exceptionally be habitual in health. On the other hand, 80 or more per minute may be the standard rate of some individuals, especially in neurotic subjects.

**Tachycardia.**—When the pulse-rate is considerably accelerated, up to or beyond 120, the condition is termed **Tachycardia** (Gr. *ταχύς*, swift; *καρδία*, the heart).

One of the commonest causes of a rapid pulse is **Fever**. As a rule, the pulse and temperature rates rise and fall together. Departures from this parallel curve of pulse and temperature may be observed in several febrile affections. In cerebral disease accompanied by fever the pulse may be slowed by the onset of intracranial pressure, while the fever is maintained or is increased. This is commonly seen in tubercular meningitis and cerebral abscess. The contrary condition of a low temperature with a frequent pulse is seen in **collapse**. An increase of the pulse-rate out of proportion to the elevation of temperature is characteristic of **scarlet fever**, but not especially so of other feverish conditions

with which it might be confused. In **typhoid fever**, on the other hand, the pulse-rate is usually less increased than the degree of pyrexia would lead one to expect. This may help us to distinguish typhoid from acute tuberculosis (especially the miliary form) and septicæmia, for which affections it may often be mistaken. In these the pulse-rate is raised proportionately as much as, or even more than, the temperature. This parallelism of the two curves is as a rule a favourable sign in febrile conditions. An increase of the pulse-rate without a corresponding increase of temperature is of more serious import than a moderate pulse-rate with a high fever. A sustained high rate of pulse while the patient is at rest is unfavourable for prognosis.

Tachycardia without pyrexia may indicate exophthalmic goitre, endocarditis and its resulting valvular disease, pericarditis, chlorosis and other anæmias, hysteria and other nervous disturbances. It also results from the abuse of tobacco, alcohol, tea, and from general debility. In addition to alcohol, atropine is the drug which most actively increases the pulse-rate. A 'paroxysmal tachycardia' is occasionally observed, in which the pulse may rise to over 200. The attack lasts as a rule an hour or two, and may occur at irregular intervals for years. It is said (Martius) to be due to sudden temporary dilatation of the heart, to epileptiform stimulation of the accelerator nerves (Sahli), to temporary absence of the vagus stimulation, to some sudden change in arterial pressure, etc. Nothing definite is known, however, of the pathogeny of the condition. The term tachycardia is by some authorities (*e.g.*, Allbutt) restricted to this paroxysmal affection, but it is by many used in the more general sense defined above—simply as a convenient and brief reference to the symptomatic frequency of pulse arising from any cause. Analogous use of a short descriptive word to indicate some general symptom due to a variety of causes is common—*e.g.*, pyrexia, hæmatemesis, dyspnœa, neuralgia, etc.

**Bradycardia.**—Infrequency of the pulse is known as Bradycardia (Gr. βραδύς, slow; καρδία, the heart). Provided one regard the slow pulse as a mere symptom of various conditions, and in no way elevate it into an entity of disease, the use of the term is equally unobjectionable with that of tachycardia.

A pulse of 50, or even less, is sometimes found in healthy persons. The debility of cachexia, especially that from exhausting disease which interferes with nutrition—*e.g.*, cancer of the



oesophagus and starvation—are commonly accompanied by a slow pulse. Valvular disease of the heart, as stated above, generally gives rise to a frequent pulse. Stenosis of the aortic orifice (a rare affection) is, however, often the cause of a slow pulse. In this condition the effort of the left ventricle to expel its contents is more prolonged than normal owing to the obstruction. For the same reason the pulse is slowed in high arterial tension, as is frequently exemplified in kidney affections. Occasionally the pulse is slow in fatty and sclerotic changes in the heart muscle and coronary arteries.

Intracranial pressure slows the pulse, as seen in meningitis, cerebral hæmorrhage, tumour, etc. If the pressure is gradually developed, it has no decided effect on the pulse. A diminution in frequency is often observed in melancholia, mania, general paralysis of the insane, epilepsy, sunstroke, and in myxædema.

The presence of certain poisons in the blood may render the pulse less frequent. Of these digitalis, opium, carbon monoxide, and lead, bile, urea, and other waste products of metabolism, are the most commonly occurring.

A form of slowed pulse, which is further referred to in the article on Arrhythmia (p. 40), is a condition known as Stokes-Adams disease. The prominent symptoms are syncope, epileptiform attacks, and vertigo, while the radial pulse and ventricular contractions are very infrequent, perhaps as low as 20 or 30 in the minute. At the same time, the auricles are contracting twice or three times for each ventricular beat, as shown by the venous pulse in the neck. The causes of this 'heart-block' are uncertain, and may be associated with arterio-sclerosis, or without obvious pathological changes in the heart or vessels.

Abnormalities in the rhythm of the heart-beat are considered in the article on Arrhythmia (p. 37).

(d) **The Volume of the Pulse** depends on the tension of the arteries and capillaries and the strength of the ventricular contractions. A small pulse (*pulsus parvus*) is often a pulse of high tension. In this case the excursions of the arterial wall are diminished in amplitude by the constricted or inelastic condition of the vessels and the high pressure of their fluid contents. On the other hand, a small pulse is often associated with low arterial tension, and may be the result of weak action of the left ventricle. Here the pulsation wave is of small bulk, owing to deficient propulsive power. This occurs in heart failure (e.g., in pneumonia,

typhoid fever, or failing compensation in valvular disease), or in mitral stenosis in which the left ventricle has not become hypertrophied. Again, a similar small volume of pulse is found even in cases where the left ventricle is hypertrophied, but where the quantity of blood finding its way into the aorta is comparatively small. This is the case in mitral incompetence, as a portion of the blood ejected by the left ventricle escapes back into the

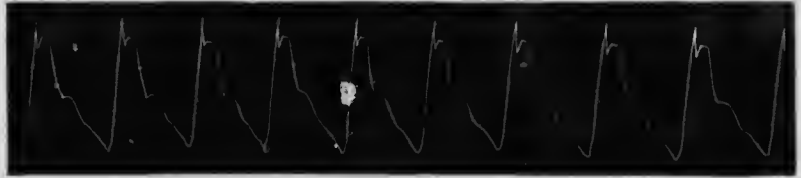


FIG. 53.—PULSE OF SLIGHT AORTIC REGURGITATION WITH FAIR COMPENSATION. (MACKENZIE.)

auricle, and in the rare condition aortic stenosis, owing to which a mechanical obstruction hinders the outflow into the aorta. Here also the arterial tension is low. Lastly, the ventricular power may be sufficient and the quantity of blood expelled by it at each stroke be ample, and still the pulse may be small. This occurs in consequence of an obstruction in the course of the artery between the heart and the radial artery, whereby the

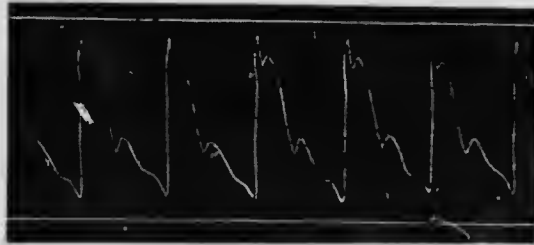


FIG. 54.—AORTIC REGURGITATION WITH GOOD COMPENSATION (COLLAPSING PULSE).

pulsation wave is checked and diminished in amplitude. Aneurism of the aorta, innominate or subclavian arteries, is the not uncommon cause of this condition.

A large or bounding pulse (see Figs. 49 and 54)—*pulsus magnus*—is found in those conditions which favour the transmission of an ample wave from the heart to the periphery. They are: a strongly acting heart, a low arterial tension, elasticity of the

arterial walls, and an ample supply of blood passing without obstruction to the periphery. These conditions are present—in part, at least—in very different states of the heart and blood-vessels. In a healthy subject whose left ventricle is acting powerfully (from exercise or emotion) the pulse has this character. Here the force of the ventricle and the natural elasticity of the coats of the vessels are enough, even with a normal arterial tension, to cause unusually ample pulsations of the radial. Dilatation of the capillaries and small arteries occurring in fevers, and as a result of alcohol or other drugs, lowers the tension, and if the heart is acting with due force, will permit the systolic wave to assert itself as a large and bounding radial pulse. Should the heart muscle weaken, as it so frequently does in severe fevers, the pulsations become feeble, and the radial pulse grows small and thready. Incompetence of the aortic valves produces a markedly large pulse. In this condition the left ventricle is hypertrophied and dilated, so that at each contraction a large

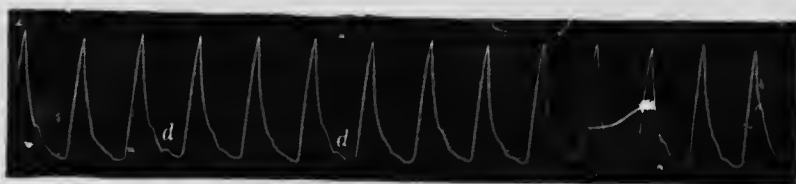


FIG. 55.—PULSE OF EXTREME AORTIC REGURGITATION. (MACKENZIE.)

volume of blood is thrown with unusual force into the arteries, which, owing to the regurgitation backward into the ventricle during diastole, are in a condition of low tension. In spite of the fact that in many cases of aortic incompetence the arteries have lost much of their elasticity, the association of low tension, powerful ventricular contraction, and a large bulk of blood entering the vessels with each beat of the heart is quite sufficient to produce a characteristic type of bounding pulse. It seems to spring up under the examining finger suddenly to a considerable height, and as quickly to fall away again. The excursions of the vessel wall with each pulsation are much greater than usual, and the pulse is variously known as the **Corrigan's pulse**, the **collapsing pulse**, the **water-hammer pulse**, and **pulsus celer**.

The volume of the pulse may, like its rhythm, be irregular. Indeed, the two forms of irregularity are very commonly associated. When a strong and a weak beat alternate, the pulse

is known as **pulsus alternans**. The weaker beat is usually preceded by a shorter diastole than the stronger, so that it assumes the bigeminal character. The shorter diastole ensures a weaker beat, as a less amount of 'irritable material' has accumulated. (See Arrhythmia, p. 39.) During inspiration the pulse may be observed in some cases of adherent pericardium to diminish in volume and become almost or completely imperceptible (**pulsus paradoxus**). This abnormality of pulse has less frequently been observed in pleurisy, valvular disease of the heart, pneumonia, and in obstruction of the air passages.

(e) **The Duration of the Pulse** is of less interest. Briefly stated, a large, bounding pulse is a rapidly advancing and rapidly receding pulse-wave. A small pulse of high tension, on the contrary, is a slowly advancing and slowly receding pulse-wave. The former is known as **pulsus celer**, or the **quick pulse** (which must not be confounded with the frequent pulse), and is found in fevers, in aortic incompetence, etc. (See Pulsus Magnus above.) The **slow pulse**, or **pulsus tardus** (not the infrequent pulse), is found in those conditions characterized by high tension (see above, p. 298).

**PULSE, Bigeminal.** See Arrhythmia, p. 42.

#### **PULSE, Capillary.**

On rubbing the skin of the forehead gently, or drawing the finger-nail across it, the red mark which is thus produced may in cases of excessive arterial pulsation be seen to flush and pale synchronously with the systole and diastole of the ventricles. A similar ebb and flow of the pink colour seen through the nails may be observed under like conditions. It is almost exclusively in aortic incompetence that this symptom is noted.

#### **PULSE, Collapsing (Corrigan's Pulse, Pulsus Celer, Water-hammer Pulse).**

These terms are used to indicate the type of pulse found in one condition—viz., aortic incompetence. It is a pulse of high systolic and low diastolic tension, and gives the examining finger the impression of a rapidly filling and rapidly emptying vessel. The pulse-wave seems to strike the finger with a distinct tap, while

the sphygmographic tracing shows a sudden up-stroke and an almost equally sudden down-stroke, with ill-marked or absent dicrotic wave. (See Arterial Pulse, p. 308, and Fig. 55.)

**PULSE, Dicrotic.** See Arterial Pulse, p. 302.

**PULSE, Intermittent.** See Arrhythmia, p. 42.

**PULSE, Large (Bounding Pulse, Quick Pulse, Pulsus Celer, Pulsus Magnus).**

By these terms is indicated the sensation imparted to the examining finger as if the arterial wall were moved through a greater distance than usual with each wave of the pulse. Also it conveys the impression that the distension of the vessel by the advancing wave is accomplished quickly and without serious hindrance being offered by the tension of the artery. It is observed in those conditions which favour the transmission of an ample wave from the heart to the periphery—viz., a strongly acting heart, a low arterial tension, elasticity of the arterial walls, and an ample supply of blood passing without obstruction to the periphery.

The subject is further discussed in the article on the Arterial Pulse (p. 303).

**PULSE, Movable.**

If the arteries are tortuous and the pulsations considerable in amplitude, the artery may be seen not only to pulsate, but actually to move out of its position with each beat. This is best seen in cases of aortic incompetence (see p. 297).

**PULSE, Paradoxical (Pulsus Paradoxus).**

By this term is indicated the diminution in volume, or even the disappearance, of the pulse during inspiration, which is sometimes observed in adherent pericardium and adhesive mediastinitis. It occasionally occurs in valvular heart affections, in pleurisy, in pneumonia, and in obstruction of the air passages.

**PULSE, Small (Pulsus Parvus).**

The sensation imparted to the examining finger is that the pulse-wave is restricted in height, and that the blood-stream is

small in bulk. It is often a pulse of high tension, but may be observed in cases of low tension which results from weakness of the left ventricle, from mitral incompetence, or from aortic stenosis. (See Arterial Pulse, p. 307.)

### **PULSE TENSION.**

The arterial tension is tested at the radial artery by digital examination, by the sphygmograph, and by the sphygmomanometer. Tension depends on—(a) the strength of the ventricular contractions; (b) the volume of the blood in the arteries; (c) the peripheral resistance; and (d) the elasticity of the arterial walls. The subject is further considered in the article on the Arterial Pulse (p. 298).

**PULSE, Trigeminal.** See Arrhythmia, p. 42.

### **PULSE, Unequal (Pulsus Inequalis).**

An inequality in the volume of the two radial or other arteries. It may be due to a want of symmetry in the distribution of the vessels, or to an obstruction in the course of one of the arteries—*e.g.*, an aneurism. (See Arterial Pulse, p. 297.)

### **PULSE, Venous.**

The flow of blood through the veins is not normally intermittent or pulsating. Under certain conditions the veins are seen to pulsate, and a careful study of this symptom is very instructive.

In conditions of excessive arterial pulsation, such as may be observed when the arteries are much relaxed, and where the conditions are favourable for the production of a large and bounding radial pulse (see p. 308), the arterial pulsations may persist through the capillaries, and become evident in the more distant and smaller veins (**penetrating pulse**).

Of greater clinical interest is the pulsation which may be seen best in the veins of the neck under a variety of circumstances, but always as a result of the action of the **right** side of the heart.

The contraction of the heart is of a peristaltic nature, commencing at the mouths of the great veins which open into the auricles. (See Arrhythmia, p. 38.) The first act in the contraction

is the more or less complete closure of the orifices of these vessels, so that as the wave of contraction sweeps over the auricles towards the ventricles but little of the contents of the auricles is forced back into the venæ cavæ and pulmonary veins. There is, therefore, no perceptible back-travelling wave in these vessels, unless the veins are unusually distended.

In certain conditions of disease, as mentioned below, the openings of the veins are inefficiently guarded, and the contracting heart propels a wave back into the veins, which is most conveniently studied in the vessels of the neck.

The internal jugular vein, when its movements can be observed, is very suitable for examination. Its position in the neck prevents one from seeing the movements of its walls, but its pulsations raise the tissues superficial to it to a sufficient degree to enable one to make observations. The patient should be in the recumbent position, if possible. Some means must be used to assist one to time the pulsation—that is, to ascertain with which period of the cardiac cycle it corresponds. Light levers, flags, or strips of cotton-wool may be so fastened to the skin over the vessel that its movements are amplified and rendered more visible. It is necessary, if useful information is to be obtained from the pulsating veins, to obtain a graphic record of its movements. By means of recording tambours such a record can often be secured, and the modification of the sphygmograph designed by Mackenzie (see p. 303 and Fig. 52) is a simplification of the more elaborate instruments used in the laboratory. Pulsation of the internal jugular vein is often difficult to distinguish from that of the carotid artery. The latter is more abrupt in its pulse stroke and more gradual in the falling away of the wave than the jugular vein. The retraction of the vein after its wave has passed is usually sudden, often forming a definite recession over the vessel. If the pulsation were in the carotids, similar excessive pulsation would almost certainly be observed in the radials and other peripheral arteries; the majority of pulsating jugular veins are due to conditions which show feeble pulsations in the arteries. Pulsation in the superficial veins of the neck does not occur so readily as in the internal jugular or subclavian, but is more easily recognized. By means of a graphic record, simultaneously taken, of either the apex-beat, carotid, or radial artery, along with the internal jugular vein (Mackenzie prefers the radial), it may be possible to learn much as to the condition of the right heart.

Before proceeding further, the reader should consult Fig. 56, which displays a comparative record of the tension in the chambers of the heart and in the aorta during the heart's action.

The pulsation may be presystolic in time. This is not neces-



FIG. 56.—DIAGRAM SHOWING THE PULSE-CURVES PRODUCED AT THE APEX-BEAT (A.B.), THE CAROTID ARTERY (CAR.), THE JUGULAR VEIN (JUG.), AND THE RADIAL ARTERY (RAD).

The perpendicular lines represent the time of the following events: 1, the beginning of auricular systole; 2, the beginning of ventricular systole; 3, the appearance of the pulse in the carotid; 4, the appearance of the pulse in the radial; 5, the closure of the semilunar valves; 6, the opening of the bicuspid valves. (John Hay.)

sarily an evidence of disease, as moderate distension of the veins (as in holding the breath or making a strong muscular effort) may be enough to induce slight pulsation. Hence it is often spoken of as the normal or physiological venous pulsation. It is obviously due to the contraction of the right auricle. Its



presence is a proof that the walls of that chamber possess a fair measure of contractility, but when well marked this pulse suggests that the orifice of the vena cava is so little contracted during the auricular systole that the pressure in the large veins is practically identical with that in the right auricle. Tracings of this type of jugular pulsation (see Fig. 57) show first the elevation due to the auricular systole (*a*), followed by the fall (*x*), resulting from the closure of the tricuspid valve. A gradual rise of the lever ensues, due to the filling of the auricle by the venæ cavæ during the ventricular systole (*v*), followed by another fall (*y*) as soon as the ventricular diastole begins, after which the curve again rises for the next auricular systole. Figs. 58 and 59, tracings of venous pulses, show also the auricular or presystolic wave.

In some cases the elevation just referred to as synchronous with the ventricular systole assumes greater prominence, and may exceed the auricular systolic curve, which in many instances is not to be seen on the tracing. The ventricular systolic eleva-



FIG. 57.—SIMULTANEOUS TRACINGS OF THE JUGULAR AND RADIAL, AND CAROTID AND RADIAL PULSES.

*E* and  $\dot{E}$ , auricular outflow, right and left, through arterial orifices; *c*, carotid wave; *v*, ventricular wave; *c*, carotid wave; *x*, auricular depression; *y*, ventricular depression. The dotted lines between *a* and *x* indicate probably the true venous pulse-wave. (Mackenzie.)

tion under these circumstances is the result, not of the normal filling of the left auricle from the venæ cavæ alone, but of a regurgitation through the tricuspid orifice. The presence of the 'ventricular venous pulse' (Mackenzie), then, indicates tricuspid incompetence, resulting as a rule from the raised tension and dilatation of the right ventricle consequent on mitral disease, or may be a sign of dilated right heart in acute fevers, in pulmonary disease, or in cachectic conditions. So long as the right auricle retains some tone and contractility, the 'auricular venous pulse' will be discernible; but as the auricular wall grows weaker and more dilated by the raised tension in the right heart, the presystolic wave becomes feebler and more delayed, and eventually disappears. It is this ventricular systolic venous pulse which is

often termed the positive or pathological venous pulse. Such an advanced state of auricular paralysis is only likely to occur if the right ventricle is fairly active, even though it is so much dilated



FIG 58 —TRACING OF A LARGE VENOUS PULSE OF THE AURICULAR FORM (MACKENZIE.)

as to cause incompetence of the tricuspid valve. Hence the disappearance of the presystolic or auricular venous pulse is usually



FIG 59 —SIMULTANEOUS TRACINGS OF JUGULAR AND RADIAL PULSES, SHOWING A LARGE WAVE (a) DUE TO THE AURICLE. (MACKENZIE.)

an evidence of hypertrophied as well as dilated right ventricle, and is observed in mitral disease, but not in anæmia or fatty degeneration of the heart muscle.

### PUPILS, Abnormalities of the.

Anatomy and physiology of the pupillary reflexes—Methods of examination—Hippus—Direct and indirect pupil reflex—Accommodation—Skin or pain reflex.

The pupil in disease—Myosis—Mydriasis—Accommodation—Wernicke's hemiopic pupillary reaction—Argyll-Robertson pupil—Westphal's pupil reaction—Unequal pupils—Irregular pupils—Increased hippus—Skin reflex.

Considerable variations in the size and movements of the pupils are noticed under normal circumstances. In disease, and especially in disease of the nervous system, the variations are more definite and characteristic, and furnish us with valuable aid in diagnosis. In order to interpret fairly the information they convey, we must first consider briefly the mechanism by which changes in the pupils are brought about.

**Anatomy and Physiology.**—The iris is a vascular and muscular screen interposed between the lens and the cornea with the object of controlling the amount of light admitted to the retina. It is composed chiefly of bundles of plain muscular fibres, concentrically arranged (the **sphincter pupillæ**), innervated by a branch of the third nerve which reaches the sphincter via the ciliary ganglion and short ciliary nerves. There are also radially disposed fibres (the **dilator pupillæ**), which are probably muscular, and are supplied by the sympathetic nerve which finds its way to the dilator in the ophthalmic division of the fifth nerve and its nasal branch, then by the long ciliary nerves to the eyeball. Contraction of the sphincter reduces the size of the pupil; relaxation of the sphincter permits the pupil to dilate to a moderate extent by the elasticity of the radially disposed fibres. These latter are believed, as above stated, to be capable of contraction in obedience to stimuli via the sympathetic nerves. Contraction of the pupil occurs physiologically as the motor part of a reflex act, the **light reflex**, and also in association with convergence of the visual axes in the accommodation of vision to near objects. The latter function is not so much a purely reflex act, but rather an involuntary factor in the action resulting from stimulation of an 'accommodation centre,' which is initiated by volitional impulses. The reflex arc concerned in the light reflex is made up as follows (see Fig. 60): The sensitive end-organ receiving the necessary stimulus (light) is the retina. The impulse is conveyed centrewards by fibres in the optic nerve, which are believed to be distinct from those conveying impressions of light to the sensorium. Moreover, it is now assumed by some physiologists and clinicians (Bechterew, Sahli) that the centripetal fibres of the light reflex leave the optic tract before its arrival at the primary optic centres (external geniculate bodies, corpora quadrigemina, or pulvinar)—that is, behind the chiasma, traversing the grey matter adjoining the third ventricle to reach that portion of the oculo-motor nucleus controlling the sphincter pupillæ. The **iris nucleus** lies at the anterior end of the third nerve nucleus, in the floor of the Sylvian aqueduct, and in close proximity to that of the internal rectus, which produces the associated convergence in accommodation. The efferent impulse to the sphincter leaves the iris nucleus by one of the anterior rootlets of the third nerve on its way to the orbit.

While sufficient dilatation of the pupil for the purposes of the

light reflex is probably effected as a rule by mere inhibition of the sphincter contraction (by force of the simple elasticity of the radiating fibres of the iris), a more extensive enlargement of the pupil occurs as a result of contraction of the dilator pupillæ through the sympathetic. The course pursued by the dilator impulses is from a centre adjacent to that of the constrictor centre (see Fig. 72, p. 546), downward through the pons and medulla to the cord, in the lower cervical and upper dorsal regions of which

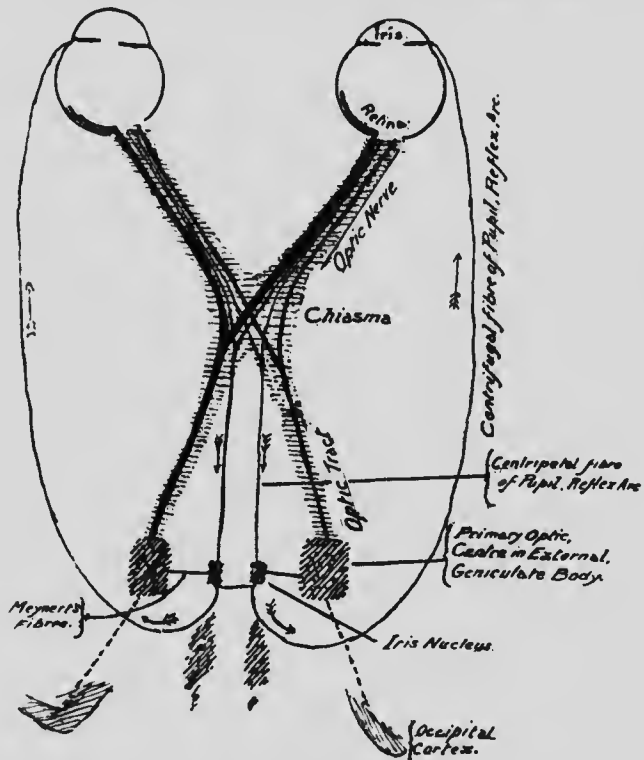


FIG. 60.—DIAGRAM INDICATING THE NERVOUS MECHANISM OF THE LIGHT REFLEX.

a series of subsidiary centres are believed to exist, the 'cilio-spinal' centres. The dilator fibres leave the cord by the second dorsal nerves, proceeding by the rami communicantes to the inferior cervical ganglion of the sympathetic chain, thence by the carotid plexus to the fifth cranial nerve, in the ophthalmic division of which they are conveyed to the orbit, and reach the iris through its nasal branch and the long ciliary nerves.

Movements of the pupil have for their object—(a) the control

of the amount of light permitted to reach the retina; (b) the exclusion of those rays of light directed towards the periphery of the lens, which, owing to increased refraction at that portion of the lens, would cause blurring of the image if allowed to fall on the retina ('spherical aberration'); (c) the narrowing pupil gives support to the ciliary muscle in its contraction during accommodation. The first of these three functions ('light reflex') is accomplished by the retinal light stimulus proceeding directly to the iris centre (probably without communicating with the primary optic centres in the anterior corpora quadrigemina), and returning as a motor impulse to the sphincter pupillæ. Contraction for accommodation is initiated by a volitional stimulus to the accommodation centre, which produces a combined contraction of the internal recti sufficient to converge the visual axes upon the object; of the ciliary muscles, whereby the suspensory ligament is relaxed and the lens becomes more convex owing to its inherent elasticity; and, finally, of the sphincter pupillæ, thereby supporting and assisting the contraction of the ciliary muscle.

The size of the pupil varies considerably in different individuals; its average diameter is 4 millimetres. It is smaller in elderly and in hypermetropic persons than in young emmetropic subjects. In myopic adolescents it is commonly dilated.

**Method of Examination.**—In examining the action of the pupil it is necessary to observe separately the effects upon it of light and of efforts of accommodation.

**Light Reflex.**—First, direct the patient to fix his eyes on a moderately bright light, not too close in front of him, such as a candle or a window on which the sun is not directly shining; cover both eyes with the hands; after a few seconds uncover one eye. The pupil should now be dilated, but when an appreciable interval after being exposed to light has elapsed (about half a second) it contracts excessively, relaxes somewhat, contracts again to a less degree than at first, and so oscillates until it comes to rest. This oscillation is termed **hippus** (see p. 325). The reaction is the **direct pupil reflex**. If the eye which has been covered during this procedure be carefully examined while the reflex is in action, care being taken to admit as little light as possible to the covered eye, the pupil of the latter will be observed to react in a similar manner, owing to the bilateral innervation of the eyes. This is the **indirect, crossed, or consensual reflex**. Any imperfection, sluggishness, or loss of the reflex must be duly noted.

**Accommodation.**—Next, the patient is made to fix his eyes on any dark object at a distance of not less than 20 feet; the examiner's finger is then quickly interposed at a distance of 12 to 15 inches in front of the patient's eyes, and he is directed to concentrate his vision upon it. The pupils should be found to contract with the act of accommodation.

**Pain Reflex.**—Pinching or pricking the skin of the neck by stimulating the cervical sympathetic causes the pupil to dilate. The same result is obtained in many cases from pain arising from injury of any region or organ of the body.

During this examination the observer will have an opportunity of noting any abnormality in the pupils, such as inequality, irregularity of shape or size, or unexpected mode of action.

**The Pupil in Disease.**—Abnormalities of the pupil are the result of disease of the eye, affections of the central, peripheral, or sympathetic nervous system, or poisons acting locally or internally. The following abnormalities may be mentioned:

(a) **Myosis**, or abnormal contraction of the pupil, is the result of irritation of the third nerve or its nucleus, and is then spoken of as **irritative** or **spasmodic myosis**; or it may be due to paralysis of the sympathetic nerve or of its nucleus—**paralytic myosis**; or both these causes may combine to produce a maximum degree of myosis.

The conditions giving rise to myosis are briefly the following:

1. Advanced age.
2. Sleep.
3. Certain poisons known as **myotics**. Eserine, or physostigmine, stimulates the endings of the third nerve and paralyzes those of the sympathetic in the iris. Pilocarpine, muscarine, and nicotine act similarly to eserine, but less forcibly (Swanzy). Opium and its derivatives taken internally are myotics, but have no such effect when applied locally. Chloroform, when inhaled, first stimulates the pupil-dilating centre, but soon reduces the irritability of this nucleus and stimulates the contracting centre, causing an extreme or 'pin-hole' contraction of the pupil. Further administration of the vapour may paralyse the pupil-contracting nucleus, causing the pupil to dilate rapidly, a sign in chloroform narcosis of formidable import, the patient being in imminent danger.
4. Intracranial lesions which irritate the third nerve or its nucleus. Thus myosis is observed in meningitis of all forms

when intracranial pressure is absent, as in the early stages of the affections. In cerebral hæmorrhage it is frequently observed, especially in the pontine lesion; in intracranial tumours or injuries which, from their nature or position, do not destroy but merely stimulate the function of the third nerve; and at the commencement of an attack of epilepsy or of hysteria.

5. Excessive use of the accommodation centre causes myosis, as may be seen in the case of engravers, watchmakers, etc.

6. In many affections of those parts of the eye supplied by the fifth nerve a reflex myosis may occur.

7. Paralysis of the sympathetic may be due to lesions in the central or peripheral portion of its course. The former may be observed in lesions above the level of the second dorsal segment of the cord. This condition, termed **spinal myosis**, is sometimes seen in the early stages of tabes dorsalis. At a later period of that affection the pupil may be contracted down to a pin-point dimension, when it may be assumed that the third nerve or its nucleus is irritated. An imperfect light reflex may coexist with spinal myosis, but, on the other hand, it may be absent. (See Argyll-Robertson Pupil below, p. 324.) Spinal myosis sometimes occurs in general paralysis of the insane, as well as in chronic inflammations or injuries of the cervical cord and medulla—*e.g.*, progressive muscular atrophy, bulbar paralysis, myelitis. The peripheral portions of the sympathetic may be the site of a lesion causing paralytic myosis; this is most liable to occur in the neck, as from aneurism, tumours, inflamed glands, or injury.

(b) **Mydriasis**.—The pupil may be abnormally dilated, the condition known as **mydriasis**. And here, as in myosis, there may be an **irritative mydriasis** due to excessive stimulation of the sympathetic, and a **paralytic mydriasis** resulting from paralysis of that portion of the third nerve or its nucleus which supplies the sphincter of the iris.

Dilatation of the pupil is observed under the following conditions:

1. In children the pupil is usually larger than in adults.
2. Certain poisons act as **mydriatics**. Belladonna and its derivative atropine paralyse the third nerve and stimulate the sympathetic. Duboisine, hyoscyamine, daturine, and scopolamine act in a similar manner. Cocaine irritates the sympathetic nerve locally. Strychnine and curare act indirectly by the retention of

carbonic oxide in the blood. Chloroform narcosis, in its earliest and in its latest stages, dilates the pupil, as stated above, under Myosis.

3. Direct irritation of the dilator pupillæ centre or sympathetic fibres in some part of their course—viz., spinal meningitis, hyperæmia of the cervical cord, sunstroke in its early stages, the early stages of cervical caries, and of tumours of the spinal canal or intracranial cavity which do not give rise to pressure symptoms; dyspnœa (excess of carbon dioxide in the blood); a premonitory sign of locomotor ataxia.; inflammations or tumours in the neck which irritate but do not destroy the continuity of the sympathetic path.

4. Indirect irritation of the sympathetic system. In powerful emotions, neurasthenia, hysteria; melancholia, mania; reflex peripheral irritation—*e.g.*, intestinal worms, severe pain; debility, as in anæmia and convalescence from acute and exhausting diseases.

5. Paralysis of the sphincter pupillæ (**iridoplegia**) from (i.) Lesion of the pupil-contracting centre or of the motor nerve fibres supplying the sphincter: cerebral or meningeal hæmorrhage; thrombosis (especially when it involves the cavernous sinus); tumour and abscess of the brain; orbital tumours pressing on the ciliary ganglion or nerves; glaucoma; intra-ocular tumours. (ii.) Deficient perception of light in the eye, and defective conduction of the stimulus to the pupil-contracting centre: atrophy of the optic nerve (*q.v.*, p. 551) frequently occurs in tabes dorsalis and general paralysis of the insane, more rarely with lateral sclerosis and multiple sclerosis. Optic neuritis in some cases interferes with vision, but less frequently than one would expect considering the nature and position of the lesion. In these cases mydriasis may occur.

It will thus be seen that paralytic mydriasis or iridoplegia occurs when the light reflex arc is interrupted. Those cerebral lesions affecting the cortical centre of vision in the occipital lobe, or its connections with the oculo-motor nucleus, cause blindness (**supranuclear blindness**), but the light reflex remains intact; the pupil of the unseeing eye responds to the stimulus of light, and maintains a moderate degree of contraction. Nuclear and infranuclear blindness (lesion of the nucleus or centripetal infranuclear fibres), on the contrary, produces paralysis of the sphincter, with mydriasis and loss of light reflex.



(c) **Accommodation.**—Abnormalities in accommodation consist in deficiency in the contracting power of the ciliary muscle (**cycloplegia**), and in that of the muscles concerned in convergence—viz., the internal recti. The former may be part of a widespread paralysis of the oculo-motor apparatus from an affection of the third nerve. It may result from the specific action of a toxin—*e.g.*, diphtheria—or from the local or general action of a poison, as seen in the effects of atropine or duboisine. Deficient convergence may also be part of a general oculo-motor paralysis. It may be produced by a lesion in the pons; by myopia, when it is accompanied by fatigue, double vision, and slight divergent strabismus for near vision. It is sometimes seen in hysteria; in exophthalmic goitre failure of convergence, or even slight divergent strabismus may be observed, and is known as **Moebius's sign**.

(d) **Abnormalities of the Light Reflex.**—Under a variety of pathological conditions the pupils fail to react to light—viz.:

1. States of profound unconsciousness, especially when produced by cerebral pressure. Here the defect is bilateral.
2. The various poisons mentioned above which produce mydriasis. When applied locally the defect may be unilateral, but internal administration gives a bilateral result.
3. Lesions interrupting the pupil reflex arc in its central course (intracranial hæmorrhage, chronic degenerative or inflammatory changes in the third nucleus, tabes dorsalis, etc.).
4. Peripheral oculo-motor paralysis (see p. 216).
5. Affections of the retina interfering with efficient perception of light.
6. Optic atrophy, and occasionally optic neuritis, if sight has been lost. It is, however, possible that the loss of light perception may be very considerable while the light reflex is retained, if it be admitted that the afferent fibres of the pupil reflex are distinct from those conveying impressions to the visual centre (see Fig. 60, p. 318).

Loss of the pupil reflex usually results in dilatation of the pupil, which may be easily recognized in the unilateral affection.

(e) **Wernicke's Hemiopic Pupillary Reaction.**—This is a phenomenon of some diagnostic importance observed in certain interruptions of the pupil reflex arc. Injury to the afferent fibres from the retina causes loss of light reflex (as well as of vision). If the lesion is in such a position in the optic nerve, chiasma, optic

tract, or iris nucleus as to involve one half of the retina in one or both eyes, light stimuli falling on the insensitive portion of the retina should give rise to no reflex contraction of the pupil. Under ordinary circumstances light entering the pupil is distributed by diffusion and reflection to all the retina; hence the light reflex is not interfered with by want of function of one half of the retina (**hemianopsia**) (see *Vision, Disorders of*, p. 545), the active region sufficing for its due performance. It has been shown by Wernicke that by carefully focussing a cone of light upon the retina it is possible to stimulate one portion of the retina to the exclusion of the rest of the sensitive surface. By reference to Fig. 72 it will be seen that an interruption at *c* will render the temporal half of the left retina and the nasal half of the right insensitive to light. If, then, a beam of light be focussed upon either of these regions, there will be no responsive contraction of the pupil in the eye under examination, nor in the other eye. The ray of light is then directed to the sensitive half of the same retina, with the result that the sphincter pupillæ contracts in both eyes—*i.e.*, we find both the direct and the crossed pupil reflex. If the lesion should be in the situation *b* (Fig. 72), dividing the chiasma antero-posteriorly, the nasal half of each retina will be blind (**bitemporal hemianopsia**). By diffuse light the direct and indirect pupil reflex occurs, but on stimulating the nasal halves of the retina by Wernicke's method both pupils are immobile. A lesion at *c* in the optic tract interrupting both the visual and light reflex centripetal fibres causes, as stated above, blindness of the corresponding half of each retina (**homonymous hemianopsia**). Stimulation of this portion of the retina alone produces no contraction of the pupil in either eye (**crossed and direct homonymous hemiopic immobility**), while stimulation of the sensitive portions of the retina gives the ordinary direct and indirect pupil contraction.

(*f*) **Argyll-Robertson Pupil**.—An abnormality of the pupils observed chiefly in locomotor ataxia and also in general paresis, first described by Dr. Argyll-Robertson, is absence of pupillary contraction to light, while accommodation produces the customary contraction. This is commonly attributed to interruption of Meynert's fibres (see Fig. 60), connecting the oculo-motor nucleus with the primary optic nucleus in the superior corpus quadrigeminum, etc. This lesion, however, would not account for those somewhat rare cases in which the Argyll-Robertson

pupil is unilateral, and it is unnecessary so to account for the symptom if we admit the existence of centripetal pupil reflex fibres proceeding direct to the iris nucleus, independently of the visual fibres. Under this assumption a lesion at *d* (Fig. 72) will destroy the pupil reflex, while the accommodation mechanism is undisturbed. It may be that this afferent limb of the light reflex is especially vulnerable in tabes and in general paralysis of the insane, for it is in these two affections almost exclusively that this phenomenon is observed.

On rare occasions a reversal of the light reflex has been observed in tabes and general paresis, the pupil dilating on exposing the eye to light, probably from fatigue.

(*g*) **Westphal's Pupil Reaction.**—Westphal has described a contraction of the pupil as occurring on the production of 'Bell's phenomenon' (*q.v.*, p. 222). The patient's eyelids are held apart while he attempts to close the eye; the eyeball turns up behind the upper lid, and the pupil, in some cases of tabes and general paresis, is seen to contract.

(*h*) **Inequality of the Pupils (Anisocoria;** from Gr. *ἀνισος*, unequal; *κόρη*, the pupil) may be found in health in persons with unequally refracting eyes, and in unilateral dilatation or contraction of the pupils from some of the affections mentioned above under **Mydriasis** and **Myosis**—*e.g.*, the unilateral action of mydriatics and myotics; lesions of the third nerve or of its nucleus; general paresis and tabes dorsalis; lesions of the sympathetic; iritis, keratitis, glaucoma, and other local eye affections.

(*i*) **Irregularity of the Pupils** is most frequently caused by adhesion of an inflamed iris to the lens (**posterior synechia**; from Gr. *σύν*, *ἔχειν*, to join together), or by inclusion of the iris in the cicatrix of a healed perforated ulcer of the cornea (**anterior synechia**).

(*j*) **Increased Hippus.**—An excessive degree of oscillation of the pupil in contraction (see above, p. 319) is seen at times in multiple sclerosis, in post-apoplectic spastic conditions, in meningitis, and in neurasthenia. As a rule, it coincides with increase of the reflexes generally.

(*k*) **Skin Reflex.**—Dilatation of the pupil on stimulating the sympathetic by pricking or pinching the skin of the neck is by no means constantly observed. If it is seen on one side and not on the other its absence may be of diagnostic value, and may indicate local eye affections (synechia, glaucoma), a lesion of the upper

nerve roots, or of any portion of the peripheral sympathetic fibres.

**Summary of Pupillary Disturbances.**—The following abnormalities may be observed in the condition and action of the pupils:

(a) **Myosis.**—Excessive contraction of the pupil may be **irritative** (third nerve) or **paralytic** (sympathetic), or both.

*Causes.*—Advanced age; sleep; myotic drugs; intracranial lesions which merely irritate the third nerve—*e.g.*, meningitis without intracranial pressure, cerebral hæmorrhage (especially pontine), tumours of the brain and base of the skull; spinal lesions interrupting the sympathetic fibres (**spinal myosis**)—*e.g.*, tabes dorsalis, general paralysis of the insane, progressive muscular atrophy, bulbar paralysis, myelitis, etc.; cervical lesions—*e.g.*, aneurisms, tumours, inflamed glands, trauma.

(b) **Mydriasis.**—Excessive dilatation of the pupil may also be **irritative** (sympathetic), **paralytic** (third nerve), or both.

*Causes.*—Youth; mydriatic drugs; irritation of sympathetic—*e.g.*, spinal meningitis, sunstroke, early stages of spinal caries and tumour; dyspnœa; irritative lesion in neck; direct sympathetic stimulation, as in emotions, hysteria, mania; peripheral irritation—*e.g.*, intestinal worms, pain, debility, anæmia, convalescence from debilitating diseases; paralysis of the sphincter pupillæ (iridoplegia) from nuclear or infranuclear lesions—*e.g.*, cerebral and meningeal hæmorrhages or thrombosis, tumour or abscess of the brain (supranuclear lesions cause blindness, but do not interfere with light reflex); diseases of the eye—*e.g.*, glaucoma, intra-ocular tumours; deficient perception of light, as in optic atrophy.

(c) Deficient contraction of pupil during accommodation may be due to certain mydriatics—*e.g.*, atropine; toxic blood-states—*e.g.*, diphtheria; a widespread oculo-motor paralysis from any cause—*e.g.*, pontine lesion; exophthalmic goitre (Moebius's sign); hysteria.

(d) Failure of light reflex may be observed in lesion of the central portion of the reflex arc from injury, inflammatory and degenerative changes—*e.g.*, hæmorrhage, tabes dorsalis, ophthalmoplegia interna; lesion of the peripheral portion of the reflex arc, as may occur from intracranial tumours, hæmorrhage, or exudate, and orbital lesions involving the optic and oculo-motor nerves; also optic atrophy, local eye affections, and the use of mydriatics; coma from toxæmia or intracranial pressure.

(e) Other pupillary abnormalities may be enumerated :

1. Argyll-Robertson pupil occurs in tabes and in general paresis.
2. Wernicke's hemiopic pupillary reaction occurs in lesions of the optic tract and of the chiasma; rarely in those of the optic nerve.
3. Westphal's reaction in tabes and in general paresis.
4. Inequality of the pupils in unequal refraction of the eyes; in unilateral, central, or peripheral affections causing myosis or mydriasis.
5. Irregularity of pupils in diseases of the eye.
6. Increased hippus in multiple sclerosis, in spastic conditions after hæmorrhage, in meningitis, and in neurasthenia.
7. Absence of the skin or pain pupil reflex may, if unilateral, indicate an interruption in the sympathetic path, or else a local disease of the eye.

#### **PUPIL REFLEX (Light Reflex).**

The stimulus of light falling on the retina sets in action a reflex which has for its object the regulation of the amount of light admitted into the eye. This is accomplished by contraction of the sphincter iridis, in obedience to stimuli proceeding via a branch of the third nerve from the iris nucleus in the floor of the Sylvian aqueduct. This is the **direct pupil reflex**. The **indirect or consensual pupil reflex** is the contraction of the pupil of an eye which is protected from light, the movements occurring in obedience to light stimulation falling on the retina of the **other** eye (see p. 319).

**PURIN BODIES.** See **Urine, Abnormalities of**, p. 510.

**PUSTULAR ERUPTIONS.** See **Skin Eruptions**, p. 368.

**PYREXIA** (Gr. πῦρ, fever heat; ἔχω, to have).

Elevation of the body temperature above the normal range. The varieties to be observed in feverish temperatures and their causes are discussed in the article on **Temperature of the Body** (p. 403).

**PYROSIS.** See **Waterbrash**. p. 562.

**PYURIA** (Gr. πύον, matter; οὔρον, urine).

The presence of pus in the urine may be recognized by means of the microscope; the white cells with divided nuclei are easily recognized. On the addition of some liquor potassæ to urine containing pus, the deposit becomes clotted and difficult to pour out of the vessel. The diagnostic significance of pyuria is considered in the article on Abnormalities of the Urine (p. 506).

**QUINQUAUD'S SIGN.**

The patient is directed to press the tips of his outspread fingers against the palm of the observer's hand. Normally the pressure is quite steady, but in cases of chronic alcoholism a tremulous knocking sensation is felt in the observer's hand, which may not occur for the first few seconds, but soon becomes observable.

**RACHITIC CHEST (Ricky Chest).**

A sub-typical chest, showing a vertical groove on each side of the sternum; the deepest part of the groove is generally the junctions of the ribs with the costal cartilages. It is due to a yielding of the chest-wall at its weakest part, owing to an abnormal softness of the walls, and the diminished atmospheric pressure inside the thorax, especially during the act of inspiration. The disease occurs in childhood, and when this type of chest is seen in an adult the walls are fixed in their abnormal form by the subsequent hardening of the bones and cartilages (see p. 467).

**RACHITIC ROSARY (Ricky Rosary).**

As a result of rickets the ends of the ribs, like those of other long bones, become enlarged; the junctions of the ribs with the costal cartilages show this enlargement in a marked manner, and the successive swellings are seen and felt as a row of beads down each side of the front of the chest. This is known as the rachitic rosary.

**RADIOGRAPHY.** See **X-Ray Diagnosis**, p. 565.**RÂLE** (Fr. *râle*, a rattling sound).

This term is by some authors applied to every kind of adventitious sound occurring in the lungs; it seems more rational to restrict the term to all those adventitious sounds which are not at

all or only very slightly musical in character. We therefore separate into a different class the so-called **musical râles**, or **rhonch** and describe râles as **crepitant**, **sub-crepitant**, **mucous**, and **gurgling**; any of these varieties may, in addition, have the qualities referred to as **consonating**, **metallic**, or **cavernous**. The subject is more fully considered in the article on the Auscultation of the Thorax (p. 418).

**RAY FUNGUS.** See **Sputum**, p. 388.

### REACTION OF DEGENERATION.

The behaviour of muscles in connection with damaged nerve structures, when stimulated with the continuous and with the interrupted currents, differs in certain particulars from that of muscles innervated by normal nerve fibres proceeding from normal nerve nuclei. Briefly stated, in the case of healthy nervous tissue and muscles the following phenomena occur: The interrupted or faradic current applied to a motor nerve produces a contraction in the corresponding muscle, which varies directly in amplitude with the strength of the current. If applied directly to the muscle it produces similar results, not by its effects on the muscle itself, but by the stimulus acting on the nerve fibrils and nerve terminations which are present in the muscle. The continuous or galvanic current, applied to the nerve or to the muscle, produces no result while the circuit is complete and the current passing; but the act of breaking a circuit, and the act of closing a circuit which had been open, gives the necessary stimulus to either the nerve or the muscle. Galvanism produces its effect not only upon nerves, but upon the muscle fibres themselves.

The galvanic current is applied by placing one pole over some part of the central nervous system, say on the cervical vertebræ. A large electrode is used, for the purpose of completing the circuit with as little resistance as possible. The other pole, employed for its polar effect—and for convenience it is a small electrode—is placed upon the muscle to be examined or on its nerve. It is found that the closure of the circuit by placing the negative pole (**kathode**) upon the muscle is followed by the best-marked results; that is, a muscular contraction is more readily produced by a current delivered in this manner than by one originated in any other way. The muscular contraction so produced is called the **kathodal closing contraction (K.C.C.)**. If the poles are now reversed and

the circuit is again closed, a muscular contraction follows, but a rather stronger current is necessary in order to produce the contraction than that used in the first case. The positive pole (**anode**) in the latter experiment was placed in the muscle, and the contraction is called the **anodal closing contraction (A.C.C.)**. With the poles in the same position the circuit is broken, and again a contraction occurs, the **anodal opening contraction (A.O.C.)**, but this time a still stronger current is required. Lastly, with the poles changed back to the original position, with the kathode on the muscle, and the circuit opened, a contraction sometimes occurs, the **kathodal opening contraction (K.O.C.)**, but this is only effected by a very strong current. The galvanic current being gradually increased in strength, the four different methods of applying the current being employed with each augmentation of the current, the contractions will occur in the following order: First K.C.C., then A.C.C., A.O.C., K.O.C.

In lesion of the lower segment of the motor tract the reaction of the muscles involved differs from that above described, and is called the reaction of degeneration (R.D.). This may be complete or incomplete.

**Complete R.D.**—The nerve is insensitive to both faradic and galvanic stimulation. The muscle does not respond to the faradic stimulus, but does so more powerfully but more sluggishly than in health to the galvanic current. Instead, however, of the kathodal closing contraction appearing first, the anodal closing contraction may be as easily, or even more easily, produced. This may be expressed in symbols: A.C.C. = or > K.C.C.; also we may find K.O.C. = or > A.O.C. The changes in susceptibility of the muscle are termed **qualitative** or **polar** changes.

**Incomplete R.D.**—The polar changes are found, but the excitability of the muscle to faradism and of the nerves to either form of current is not abolished, but only diminished. Decrease or increase in this susceptibility is known as **quantitative** changes.

R.D. occurs in cases of paralysis produced by lesions of (a) the anterior horns of the grey matter of the spinal cord; (b) the anterior nerve roots and the motor nerves proceeding therefrom; and (c) the cerebral motor nerves and their nuclei. It is found, therefore, in the following conditions: infantile paralysis; progressive spinal muscular atrophy; amyotrophic lateral sclerosis; bulbar paralysis; syringomyelia; Landry's paralysis; progressive



neural muscular atrophy (peroneal form); neuritis in its various forms affecting motor nerves; traumatic lesions, hæmorrhages and tumours, involving the grey matter of segments of the spinal cord and similar lesions interrupting the continuity of motor nerves.

The presence of R.D. in either the complete or the incomplete degree is strong evidence against the lesion being cerebral, or in the pyramidal tracts of the cord; these lesions do not as a rule involve the peripheral neurons of the motor tract. Further, the presence of R.D. contra-indicates muscular dystrophies and hysterical paralysis. (See Trophic Disturbance, p. 487).

### RECTAL REFLEX.

The retention and evacuation of the contents of the rectum are carried out mainly by a series of reflex acts and conditions, resembling in many respects those governing the bladder. The subject is considered in the article on Defæcation (p. 116).

### RECTUM, Examination of.

The omission of this procedure in cases where it is at all likely to be a source of information is unjustifiable.

In the majority of instances the examination is best carried out with the patient lying on his left side, and with his legs well drawn up on his abdomen. Occasionally it may be found that the dorsal position is more suitable, as by this posture a bimanual examination can be better accomplished; the knee-elbow may also be employed with advantage in some cases, especially when a speculum is being used.

The rectum is explored by the finger or by some form of speculum; in most cases the former is sufficient to obtain all the information possible. The right index-finger is lubricated, its nail pared short; the crevices under and round the nail being filled with soap or vaseline, the tip of the finger is gently insinuated into the bowel, following carefully the direction of its axis—that is, slightly forward through the sphincter, then back toward the sacrum, and finally upward and toward the left. The finger is then swept round the walls of the rectum, and when one is familiar with the feeling of the normal bowel, any abnormality in its shape, or the presence of any tumour or other disease condition can be readily verified. In addition one may ascertain if the sphincter has the normal amount of tone, and may observe the condition of the rectum as to irritability and temperature.

By means of the speculum one obtains a view of the interior of the rectum—not a very satisfactory survey, as a rule, though modern instruments have brought not only the upper parts of the rectum, but even the sigmoid flexure, into view.

Immediately inside the sphincter it may be possible to feel **internal piles**, though these are by no means easy to distinguish from folds of mucous membrane, unless they have become hardened by thrombosis. In this situation **syphilis** (commonest in women) is the cause of a growth which causes a hardening and narrowing of the passage; it may be mistaken for **carcinoma**, which, however, is more likely to form a funnel-shaped, ulcerated stricture, situated higher in the rectum as a rule than the syphilitic growth. **Tubercular** or **dysenteric ulcers**, when their bases are thickened and infiltrated, may be easily felt, but the more superficial inflammatory erosions are not readily distinguished by touch. **Rectal polypi, hardened fæces, foreign bodies**, or a presenting **intussusception**, may be detected by the finger. The condition of the structures outside the rectum may also be investigated by this procedure; thus one can observe **enlargement of the prostate, tumours or displacements of the uterus, inflammatory infiltration** in the pelvis or in the lower regions of the abdominal cavity.

A rectal examination in cases of intestinal obstruction may be sufficient to explain the stoppage; if the rectum is found to be dilated, even if empty, there is a probability that **fæcal accumulation** higher up may be the cause of the obstruction, for the enlarged rectum indicates that the bowel has in the past been habitually overloaded. The presence of fæcal masses in the rectum under the same circumstances would be a more reliable indication that the obstruction was due to fæcal accumulation.

In the absence of a tumour in the rectum, pus, blood, and mucus are suggestive of inflammatory affections in the colon and rectum (**colitis, dysentery, tubercular ulceration**), and this is especially the case when the digital or instrumental examination is attended with much pain.

In diseases of the nervous system it may be of importance to observe if the sphincter is able to contract forcibly on the examining finger, and also if there is the usual amount of sensitiveness in the mucous membrane of the rectum.

Other means for examining the interior of the lower bowel have been attempted, but the practical results are not encouraging; for example, distension of the rectum with air and with water as a

measure of its capacity, or as a means of detecting stricture or other change in the shape of the organ, has been recommended; attempts have been made to sound the more deeply-seated regions of the bowel by means of flexible bougies. In both these procedures the results are disappointing.

For further consideration of the subject see the articles on *Fæces* (p. 137), and *Defæcation* (p. 116).

### REDUPLICATION OF THE HEART-SOUNDS.

On listening to the heart one may hear in certain conditions of disease in the heart or lungs three sounds, or perhaps even four, instead of two in each cardiac cycle; this is due to a doubling of one or other of the heart-sounds. The varieties and significance of the symptom are discussed in the article on *Auscultation of the Thorax* (p. 428).

### RED VISION.

A red discoloration of all objects is often perceived by persons suffering from neurasthenia.

### REELING GAIT.

A form of ataxic gait in which loss of power to maintain the equilibrium is a prominent feature. It is seen as a staggering or reeling mode of progression, resembling that of alcoholic intoxication, and is observed in affections of the outer, middle, and internal ear, of the cerebellum, and of the eyes. The articles on *Vertigo* (p. 539) and *Gait* (p. 150) deal with the subject in more detail.

### REFLEX ARC.

In order that reflex acts may be efficiently performed, a series of structures must be intact. These are: (1) a sensitive receptive organ; (2) an afferent nerve; (3) communicating branches (by means of synapses) between the afferent nerves and (4) the ganglion cells in the anterior cornua of the cord; (5) an efferent nerve; (6) muscle (or, more rarely, gland). These six factors form the **reflex arc**, which may be divided for convenience' sake into (a) the **peripheral reflex arc**, consisting of all of it outside the brain or spinal cord, and (b) the **nuclear reflex arc**, which forms the shortest pathway between the afferent and efferent nerves in the central nervous organs (brain or spinal cord) (see p. 335).

**REFLEXES.**

Nature and objects of reflex actions and conditions—Reflex arc—Anatomy and physiology of reflexes—Deep reflexes: Knee-jerk, ankle-jerk, ankle clonus, wrist-jerk, elbow-jerk, scapulo-humeral reflex, jaw-jerk.

Diminished reflexes from—(a) Lesions of the peripheral nerves; (b) lesions of the central nervous system: tabes dorsalis, progressive muscular atrophy, infantile paralysis, hereditary ataxia, Landry's paralysis.

Increased reflexes from—(a) Transverse interruption of the spinal cord; (b) sclerotic changes in the upper segment of the motor tract; (c) intoxications; (d) functional disturbances of the nervous system.

Superficial reflexes: Plantar reflex, cremaster reflex, inguinal reflex, abdominal reflexes, gluteal reflex, interscapular reflex, corneal reflex, palatal and pharyngeal reflexes.

Visceral reflexes: Bladder, rectal, and uterine reflexes, pupil reflex, vasomotor reflex.

Reflex action consists chiefly in the production of purposeful muscular contractions independently of volition, and in obedience to stimuli originating in the periphery. Glandular reflexes resulting in modification of the function of a secreting gland are of less diagnostic interest, and will not be further referred to. The proper maintenance of the muscular reflex condition and action is essential to our safety and efficiency in relation to the external world; our muscles are by this means kept in a state of vigilance or 'tone,' ready to combine promptly in the carrying out of any movements, especially those of self-defence. Thus, blows or injuries in the neighbourhood of joints would result in serious damage to ligaments, synovial membrane, etc., were it not that the surrounding muscles are immediately and automatically placed on guard. Irritation or painful stimulation of the surfaces (skin or mucous membrane) produces a co-ordinated series of muscular contractions directed to the removal of the irritation—as, for example, on tickling or pricking the sole of the foot the toes are flexed, and the foot and leg withdrawn; a touch on the cornea causes the eyes to be promptly closed; strong light falling on the retina produces an immediate contraction of the pupil; a particle of food 'gone the wrong way' or an undue amount of mucus in the bronchial tubes gives rise to coughing.

In disease of the nervous system the reflex functions may be faulty, and a knowledge of the physiology and of the pathology of reflexes is essential to a correct appreciation of the morbid condition.

A reflex act or state requires for efficiency (*a*) a sensitive organ capable of receiving and correctly interpreting stimuli from external sources; (*b*) healthy afferent nerve fibres to convey the stimuli to the central nervous structures; (*c*) uninterrupted functional communication between the afferent nerves and the ganglion nerve cells in the anterior cornua in the case of the spinal reflexes, and the motor nuclei in the medulla, pons, and crura, in the case of reflexes depending upon cranial nerves; (*d*) an active condition of those ganglion cells in the anterior cornua; (*e*) healthy nerve fibres (axons) proceeding from the ganglion cells to the muscles—*i.e.*, healthy efferent nerves; and lastly (*f*) active and healthy muscles. These six factors form a chain which is termed the **reflex arc**, and, as stated above, must be unbroken if reflex action is to be maintained. It may be divided conveniently for clinical purposes into two parts: the **peripheral reflex arc**, consisting of those portions of the arc to be found outside the spinal cord (or brain in the case of cranial reflexes), and identical with the motor, sensory, and mixed nerves and their roots; the remaining portion of the reflex arc—*viz.*, the shortest pathway in the central nerve organs (brain and spinal cord) between the afferent and efferent nerve fibres—may be termed the **nuclear reflex arc**. This central portion of the reflex arc corresponds to what has been styled the **reflex centre**. The existence of definite centres in the cord for the control of reflex acts is, however, now generally denied, so that the somewhat less concise but more accurately descriptive term **nuclear reflex arc** might be used to express what was formerly known as the 'reflex centre.'

While it is certain that integrity of the reflex arc is essential to the due performance of reflex acts, it is probably only the simpler phenomena of this description (if any) that are independent of influences brought to bear upon the nuclear reflex arc from other regions of the central nervous system. These influences may be cerebral or cerebellar, and are exercised to secure the necessary co-ordination, control, or inhibition of the activity of the reflex arc, which is at the mercy of every chance stimulus reaching the sensory end-organs. This controlling influence is interposed at (*e*) (see above). The afferent impulse does not necessarily follow the shortest path between the sensory nerve, where it enters the cord (or brain in cranial reflexes) and the motor nerve cells. There are three possible paths (in spinal reflexes) for the impulse to pursue (see Fig. 28, p. 204): (i.) The first set of branches from the afferent

nerves in the cord communicate by their terminal divisions coming almost into contact with the branched processes (**dendrons**) of the anterior cornual nerve cells, or with intermediate cells in the posterior cornua; the impulses, therefore, are transmitted from one neuron to the other without actual contact (by synapse). This is the shortest path which reflex impulses can follow (**nuclear reflex arc**). (ii.) Another series of branches from the afferent nerves communicate with the cells of Clarke's column, whence the impulses pass to the cerebellum; the anterior cornual nerve cells are influenced by the cerebellum possibly by a direct nerve tract to the anterior cornua, and more probably via the cerebrum. (iii.) The rest of the sensory nerve, reaching the cord by the posterior root, ends in the medulla by arborizations round the nerve-cells of the nucleus gracilis or nucleus cuneatus, of the same side; thence by relays via the opposite optic thalamus and association neurons to the pyramidal nerve cells in the cerebral cortex.

From the cortex efferent fibres proceed by way of the pyramids to the nerve cells in the anterior cornua of the spinal cord; the effect of these cerebral influences is to regulate and co-ordinate the automatic motor functions. The views held by Munk, Mott, Bastian, and others, and accepted by many physiologists, that all sensory impulses from the skin and muscle are directed to the Rolandic or motor area of the cerebral cortex (named by them the sensori-motor or kinæsthetic area) places the reflex function in a more important position. In this light, voluntary movements partake largely of a reflex character. 'What really occurs in the Rolandic area is a sense of movement, and this acts as a stimulus via the pyramidal tracts to the true motor centres, which are in the opposite anterior horn of the spinal cord' (Halliburton).

It may be assumed that all the six factors in the reflex arc enumerated above are essential to the production of a normal reflex. An interruption at any part of the pathway will interfere with the due performance of the act, causing decrease or loss of the reflex. Increased activity, or the appearance of reflexes not found in health, may arise from interruption by disease to the passage of nerve impulses to or from the brain—by the pathways (ii.) or (iii.); see preceding paragraph—whereby the co-ordinating, and presumably inhibiting, influence of the higher centres is removed. Another theory, advanced by Dr. Hughlings Jackson, attributes increase of reflexes to cerebellar stimulation. In

practice it is found that lesions which separate the brain from that segment of the cord which contains the anterior cornual nerve cells involved in the reflex produce increased activity of that reflex. Bastian has shown, however, that complete transverse lesions of the cord frequently result in abolition of the subjacent reflexes (a point in favour of Jackson's view as to cerebellar stimulation); but this has been attributed by Sahli and others to damage received by that portion of the cord lying below the lesion from hæmorrhage, shock, or vasomotor disturbance. When these conditions have been recovered from, the reflexes may again be observed. Increased reflexes may also be the result of direct stimulation of the reflex arc. This is said to occur at times in the early stages of neuritis. Lastly, irritation of the cord from toxic or other causes may produce exaggerated reflexes, as in strychnine-poisoning, tetanus, hydrophobia, and hysteria.

It is sometimes difficult to ascertain the condition of the reflexes as compared with those of a healthy individual, since the variations in health are considerable. We must, therefore, compare the reflexes of one side of the body with those of the other, noting any difference in strength or extent of the movement. The information obtained in this manner is in many cases of the highest value, but some of the phenomena, especially those due to skin stimulation, are of but little aid to diagnosis.

Three main varieties of reflexes are recognized, viz. :

1. **Deep Reflexes**, also known as **Tendon Reflexes**, **Tendon Jerks**, **Periosteal** or **Fascial Reflexes**.
2. **Superficial** or **Skin Reflexes**.
3. **Visceral** or **Complex Reflexes**.

Many examples of reflex action might be given in each of these three classes. Only those which are of some help in diagnosis will be mentioned. After the description of the varieties of reflexes an indication is given of the diseased conditions characterized by abnormal reflexes.

1. **Deep** or **Tendon Reflexes** are elicited by stimulating a muscle to contract either by means of a blow struck on its tendon or by putting the muscle on the stretch. The result is a very prompt contraction or series of contractions. That this movement of the limb is not a true reflex act is now generally conceded. The involuntary contraction produced by the stroke is only possible after the muscles have been put in a condition of

**reflex readiness.** This condition has been termed **myotatic irritability** by Gowers, and is brought about by extending or stretching the muscle.

The following are the chief deep reflexes of diagnostic interest :

(a) **The Patellar Reflex (Knee-jerk)** is almost always present in health. The muscular irritability, or reflex readiness, is effected by simply flexing the knee. For a patient able to sit up the most convenient method is to direct him to cross one leg over the other, then with the ulnar edge of the hand, or with the earpiece of a single stethoscope, a sharp stroke is delivered on the patellar tendon just below the knee-cap. The result is a sudden contraction of the extensor muscles of the leg. When the patient is restricted to the recumbent position, the thigh and knee should be semiflexed, and supported by the examiner's left hand in the popliteal space, while the patellar tendon is struck. The spinal portion of the reflex arc (the nuclear reflex arc) is situated in the lumbar enlargement of the cord.

The inhibitory influence of the cerebrum upon any reflex, and upon the patellar in particular, may interfere with the due appearance of this symptom. In order to avoid erroneous conclusions, the patient's attention should be distracted, either by engaging his mind with questions or conversation, or by directing him to clasp his hands in front of his chest and endeavour to pull them asunder, as suggested by Jendrassik, and termed by him **reinforcement of the reflexes**. Absence of the knee-jerk is sometimes referred to as **Westphal's Sign**.

This reflex is more generally useful to the diagnostician than any other. Its absence at once excites suspicion as to the integrity of the reflex arc, and its exaggeration suggests an interruption in the pathway from the brain to the central reflex arc in the lumbar enlargement. Less frequently a distinct increase in the patellar reflex indicates direct stimulation of the reflex arc or functional nervous disturbance.

Occasionally when the knee-jerk is increased a repeated or clonic contraction of the quadriceps extensor cruris is observed. This is best evoked by supporting the leg in an extended position, grasping the patella between the thumb and forefinger, so that the upper border of the patella lies against the web connecting the thumb and forefinger. Pressure is then made firmly and persistently, displacing the patella downwards towards the foot, thereby extending the extensor muscle. Clonic contractions of



the muscle may then occur, and may continue as long as the muscle is kept on the stretch. This is known as **patellar clonus**, and indicates an exaggerated reflex condition.

(b) **Ankle-jerk, or Achilles' Tendon Reflex.**—Like the patellar tendon reflex, the ankle phenomenon is seen both as a single muscular contraction and as a series. The single jerk is frequently observed in health, but less commonly than the knee-jerk. It is obtained by dorsal flexion of the foot so as to stretch the calf muscles, by which means they are brought into the condition of reflex readiness already referred to. The tendo Achillis is then struck a little above its insertion, and the calf muscles contract. Instead of a single jerk of the foot, a rhythmical succession of extending movements of the foot may in cases of exaggerated reflexes be produced. In rare instances a genuine **ankle clonus**, as the phenomenon is termed, may occur in persons free from disease of the nervous system. One must not, however, mistake the few ineffectual contractions of the calf muscles seen in neuroses and in fatigue for the real ankle-clonus of organic disease of the nervous system. This is a symptom, then, of definite disease of the central neurons, and is obtained by supporting the patient's heel with the left hand, the knee being slightly flexed. The right hand then firmly pressing on the sole of the foot flexes it dorsally, with the result that the foot makes rhythmical movements of extension so long as flexing pressure is maintained. In some cases of exalted reflexes a very slight degree of flexion is enough to produce the clonus; in others mere pressure is insufficient, and the flexion must be done in a jerking, abrupt manner, so as to administer a shock to the already stretched calf muscles.

(c) **Arm-jerks** of all the available tendons may be produced by putting the muscle on the stretch, then tapping it with the fingertip. The best examples are: The **wrist-jerk**, elicited by flexing the hand (wrist-drop), and tapping on the extensor tendons, when an extending movement of the hand follows. A similar flexing jerk is seen when the hand is hyperextended and the flexor tendons are struck. **Wrist clonus** may sometimes be produced by extending the hand firmly. So long as pressure is maintained on the palm clonic contractions of the flexors continue. **Elbow-jerk** is obtained by flexing the joint and percussing the triceps tendon, when extension of the arm occurs. A flexing jerk of this joint also may be produced by striking the tendon of the stretched

biceps muscle. The arm reflexes are inconstant in their appearance; any of them may be found in health, but they are often absent in normal subjects. In conditions in which the reflexes generally are exaggerated the above and many other tendon reflexes may be seen in the arm. Another reflex which may be included among the arm-jerks is the **scapulo-humeral reflex**, elicited by striking the spinal border of the scapula just above the inferior angle. The result is in most normal cases an external rotation of the arm, due to involuntary contraction of the spinati muscles. Lesions causing obstruction in the motor tract above the cervical enlargement not only produce increase of this reflex, but also give rise to new features in it—viz., the arm is abducted, the shoulder raised, the forearm flexed at the elbow, and the fingers extended. These movements are caused by reflex contraction of the deltoid, of a part of the trapezius, of the biceps, and of the forearm muscles.

(d) **Jaw-jerk** is occasionally seen in health, but more commonly in cerebro-spinal disease with exalted reflexes. It is obtained by percussing the finger laid across the lower teeth, or just above the chin, with the mouth open. An involuntary contraction of the masseters jerks the lower jaw upwards, and on pressure being kept up clonic movements of the jaw may in extreme cases occur. The nuclear reflex arc in this case is situated in the pons in connection with the nucleus of the fifth cranial nerve.

**The Influence of Disease upon the Deep Reflexes** must now be considered; but it must not be forgotten that those reflexes which are normally present may, even in health, vary greatly from time to time even in the same individual.

**Diminished or Lost Reflexes.**—As stated above, reflexes of all descriptions depend for their production upon the integrity of the reflex arc. Any interruption in its course abolishes the reflex act in that particular group of neurons. If, then, a sufficient number of arcs is annulled, the reflex act will be not merely diminished, but abolished. The lesion causing the interruption may be due to—

(a) **Disease of the Nerves** (see table, p. 227).—Inflammation of the nerves is a common cause of diminished reflexes. A localized neuritis is rarely a cause of abolished knee-jerk (which is the only deep reflex of value as an index of decrease in the reflex condition). Inflammation of the anterior crural and obturator nerves usually occurs as part of a multiple or polyneuritis. This affection

is attributed to the following causes: (1) Alcohol; (2) metallic poisons—arsenic, lead, and mercury; (3) microbic poisons—diphtheria, beri-beri, leprosy, and occasionally scarlatina, typhoid fever, small-pox, etc.; (4) morbid blood states—gout, rheumatism, syphilis, diabetes, debility and cachexia from any cause, as in cancer, tuberculosis, or anæmia; (5) acute neuritis, due to cold and exposure, with which may perhaps be associated Landry's paralysis. (See Movement, Decreased, p. 214.)

(b) **Disease of the Nuclear Reflex Arc**—that is, of that portion of the spinal cord (or brain in cranial nerve reflexes) forming the shortest pathway for nerve impulses between the point of their arrival in the cord by the posterior nerve roots and that of their departure by the anterior roots of the nerves involved in the reflex (see above, p. 335). The afferent fibres, with their branching terminations, or the nerve cells in the anterior cornua, with their processes, may be the seat of the lesion, giving rise to the following diseases:

(i.) *Tabes Dorsalis (Locomotor Ataxia, Posterior Sclerosis)* is a progressive degeneration of the posterior nerve roots, and of their projections in the posterior columns of the cord. The reflex arc is interrupted by this disease, and, in consequence, the patellar reflex is abolished.

(ii.) *Progressive Spinal Muscular Atrophy* produces decrease in the deep reflexes, owing to the lesion of the nerve cells in the anterior cornua of the spinal cord.

(iii.) *Infantile Paralysis (Poliomyelitis Anterior Acuta)*, from acute disease of the same region, shows loss of deep reflexes.

(iv.) *Landry's Paralysis (Acute Ascending Paralysis)*, a rare affection of the spinal cord or peripheral nerves, of which the pathology is still uncertain, may be mentioned as an instance of abolished deep reflexes.

(5) *Friedreich's Ataxia (Hereditary Ataxia)*, a disease of the dorsal and lateral columns of the cord, occurring in families, in which the most prominent symptom is inco-ordination. The deep reflexes are absent, while nystagmus and other characteristic symptoms are fairly constant.

**Increase of the Deep Reflexes** is observed (as above stated) in diseases which interrupt the passage of impulses from the brain to the nerve cells in the anterior cornua, or in the case of cranial nerve reflexes, to the cranial nerve nuclei. There are, in addition, certain affections, chiefly of a functional nature, and

presumably without organic change, in which the reflexes are exaggerated, probably in consequence of some toxic irritation of the reflex arc. The following is an enumeration of the affections associated as a rule with increased reflexes :

(a) **Transverse Interruption of the Cord** above the level of the nuclear reflex arc (see above, p. 335) in most cases results in an increase in the deep reflexes. The loss of reflexes in complete transverse obstruction, referred to above at p. 337, has to be borne in mind.

The interruption may be caused by—

(i.) Compression of the cord by caries of the vertebræ, tumours, pachymeningitis, aneurism, injuries from violence.

(ii.) Myelitis.

(iii.) Hæmorrhage into the meninges or cord.

(b) **Sclerotic Changes in the Upper Motor Segment.**—(i.) Primary lateral sclerosis or primary spastic paralysis; a degenerative change in the lateral columns, without obvious causative lesion higher up in the motor tract.

(ii.) Hereditary spastic paraplegia of children.

(iii.) Lateral sclerosis, with other complications of the cord : amyotrophic lateral sclerosis; ataxic paraplegia; combined sclerosis (Dana); multiple sclerosis; syringomyelia.

(iv.) Intracranial lesions of the upper segment of the motor tract : hæmorrhage; thrombosis; embolism; tumours; birth palsies (Little's disease); meningitis in its earlier stages—in its later developments the reflexes disappear. The cerebellar type of hereditary ataxia, as described by Marie, differs from Friedreich's disease in having the deep reflexes retained and exaggerated in the later age at which the disease appears, and in the absence of deformities. Abscess of the brain and injury by violence may occasionally give rise to increased reflexes. General paralysis of the insane usually causes the reflexes to be exaggerated, but in some cases they are abolished.

(c) **Intoxications**, which apparently act as direct irritants to the reflex arc, as in tetanus and strychnine-poisoning.

(d) **Disturbances of the Nervous System not attributable to any Organic Lesion** (Hysteria, 'Railway Spine,' Tetany).—In all these affections the reflexes are usually exaggerated, and in epilepsy the same condition may often be observed after a fit.

**Superficial Reflexes (Skin Reflexes)** may be of use in localizing spinal lesions (see table, p. 227). They are inconstant

in their appearance, both in health and in disease, but when definitely increased they indicate a lesion of the upper segment of the motor tract. They are obtained by stroking or tickling the appropriate surface with some blunt object, such as the unpointed end of a pencil or the tip of the finger. In some cases the response is better elicited by pricking or pinching the skin. The more important are:

**Plantar Reflex.**—On tickling the sole of the foot the toes are flexed on the sole; the foot may be flexed on the leg and drawn up, owing to flexion of the knee and hip. In diseases of the upper motor segment these movements are often exaggerated, and the reflexes may be more widespread, contractions of the trunk and even arm muscles taking part in the phenomenon. In this condition, and especially in affections of the lateral columns, an abnormal reaction was first observed by **Babinski**. The sole of the foot toward its inner side is irritated by pricking or gently scratching with a moderately sharp instrument. In suitable cases the great toe is flexed on the dorsum, and the others are flexed on the sole of the foot.

The **Cremaster Reflex** is produced by stroking or pinching the skin of the inner side of the thigh, when the testicle of that side is retracted, care being taken not to mistake the movements of the dartos muscle for those of the cremaster. In females (and also in males) similar irritation produces a contraction of some of the fibres of the internal oblique muscle, above and along Poupart's ligament. This is known as the **Inguinal Reflex**.

**Abdominal Reflexes** are obtained by stroking the side of the abdomen and lowest part of the thorax, when the oblique and recti muscles of the same side contract in part, causing a depression of the region involved and traction of the umbilicus toward the side irritated. The reflexes are named **epigastric**, **Umbilical**, or **Hypogastric**, according to the level of the contracting region in the abdominal wall.

The **Gluteal Reflex** (contraction of the glutei muscles on irritating the skin over the buttocks) and the **Interscapular Reflex** (adduction of scapula when its spinal edge is stroked) are less constant than those already mentioned.

Among the superficial reflexes we include the **Corneal Reflex**, depending on the integrity of the fifth cranial nerve, and perhaps also of the seventh; the **Palatal** and the **Pharyngeal Reflexes**, depending upon the ninth, tenth, and eleventh nerves. The first

is produced by lightly touching the cornea, when the eyelid is promptly and involuntarily closed. The second consists in the elevation of the soft palate on being touched, and the third is the choking and retching caused by tickling the fauces. The absence of the last two reflexes is noted in bulbar paralysis and also in hysteria.

The absence of any of the superficial reflexes, with the exception, perhaps, of the corneal, does not necessarily denote an organic lesion of the nervous system; but the presence on one side of the body and absence on the other of any of them, and especially of the cremasteric, inguinal, and abdominal, indicates definite changes in the peripheral or central nervous organs.

Having enumerated the more important of the deep and superficial reflexes, we may revert to their physiology for a moment. The deep reflexes, as already stated, are not to be regarded as reflex acts, but rather as a condition of reflex readiness of the muscles to respond promptly to local stimuli. This reflex readiness, or 'myotatic irritability,' as it is called by Gowers, is brought about by stretching the muscles in question, and by relaxing their opponents; for it has been shown by Sherrington that muscular tone, or, in other words, reflex readiness, is to a large extent dependent upon impulses originating in the muscles and passing thence to the centres. He has shown that the contraction of one group of muscles causes inhibition of contraction in the opposing group, and, conversely, relaxation of a muscle permits a more active contraction of its opponent. The act of flexing the knee in order to elicit the knee-jerk relaxes the hamstrings and stretches the extensors of the leg, the latter being thus placed in the most advantageous circumstances for efficient action. It would thus appear that 'myotatic irritability' or 'reflex readiness' is merely an active and efficient state of 'muscular tone.' These phenomena of deep reflex action are believed by certain observers to be purely spinal reflexes—that is, they may be carried out by means of the spinal and peripheral mechanism alone, subject, of course, to modification and control from the cerebral centres if they are available. The same observers (Jendrassik, Sahli, etc.) regard the superficial reflexes as primarily cerebral—that is, the central portion of their reflex arc is not usually the short pathway in the segment of the cord with which the sensory and motor nerves involved in the reflex are connected [see (i.), at p. 335], but habitually follows one of the higher paths (ii.) or (iii.). In cases

of hemiplegia the lesion, being cerebral, interrupts the efferent limb of the arc at (ii.) or (iii.). The result is abolition of the superficial reflexes, with retention or exaggeration of the purely spinal deep reflexes. In cases of transverse lesion of the cord we sometimes find that the superficial reflexes, which, according to the above theory, ought to be lost, are, on the contrary, normal or even increased. This is explained by Sahli as follows: In complete interruption of the cord above the spinal reflex arc, the afferent impulses reaching the cord from the peripheral region involved in the reflex cannot pass upwards by their usual route to the brain, but take the path already formed for the deep or purely spinal reflexes, straight across by the shortest path to the anterior horns. Moreover, the obstruction to the upward passage of the sensory stimuli results in a 'damming-up' of these stimuli, which therefore may burst into the deep reflex path with such energy as to cause the skin reflexes to be exaggerated, or even to cause the appearance of new or pathological reflexes. This interesting hypothesis explains to a large extent the inconstancy of the skin reflexes. Their presence in any given region is the chief point to be ascertained, as it proves the integrity of the peripheral and, at any rate, of the lowest spinal portion of the reflex arc.

**Visceral or Complex Reflexes.**—The functions of many of the muscular viscera are performed by means of a complicated reflex mechanism, and abnormalities of these reflexes may be of considerable help in diagnosis. They resemble in many respects the skin reflexes, with special provision for the more complex action required; but it is probable that the sympathetic nervous system takes part in the reflex, though to what extent is still an unsettled question.

In several instances the visceral reflexes furnish us with valuable diagnostic information, the more important of them being the following:

(a) **The Bladder Reflex** is in reality a combination of several reflex acts and conditions. It is considered in detail in the article on Micturition (p. 195).

(b) **The Rectal Reflex** resembles in many respects that of the bladder, and is also discussed in a separate article. (See Defecation, p. 116.)

(c) **The Uterine Reflex** consists in the production of involuntary contractions of the muscular fibres of the uterus in response to

peripheral stimulation. The stimulus which initiates the reflex may be received in a variety of ways. It is chiefly in the uterus and other neighbouring organs that the impression is received. Thus, uterine contractions may be evoked by the presence of a foreign body or a tumour in the womb, by intra-uterine injections, by rectal injections or operations, by putting the child to the breast, etc. The controlling centre, situated probably in the lumbar region of the cord, may also be stimulated by drugs which reach it through the blood, and by impulses travelling down from the brain, as is seen by the influence of emotions on the uterine contractions. The cerebral influence in this reflex is slight. The automatic action has been well demonstrated in cases on record, in which a fairly normal parturition occurred in spite of a complete transverse lesion of the cord.

The diagnostic value of this reflex consists in the fact that its presence indicates an uninjured lumbar enlargement of the cord.

(d) The **Pupil Reflex** is of great diagnostic value in a variety of medical affections, and is separately considered in the article on the Pupil (p. 316).

(e) **Vasomotor Reflex**.—The regulation of the blood-supply to the various organs and tissues of the body depends upon a series of reflexes, which occasionally prove useful for diagnosis. The function is presided over by a centre situated in the floor of the fourth ventricle, from which fibres pass down by the lateral columns to terminate by arborizations round the cells of the subsidiary spinal centres, which are probably the cells of the inter-medio-lateral tract. Thence axis-cylinder processes proceed as **vaso-constrictor** or **vaso-dilator** nerves, via the anterior roots and white rami communicantes, to the various sympathetic ganglia, whence a fresh neuron or relay continues the path to the muscular fibres in the artery walls. Afferent impulses reach the centre by the sensory nerves, and exert an influence there which gives rise to vaso-constriction (so raising the arterial tension), or to vasodilatation (causing a fall of the arterial tension). The vasomotor centre may be affected by cerebral stimuli, as by emotions; by poisons, as digitalis, which raises the arterial tension by constricting the peripheral vessels; and by venous blood, as shown by the rise of pressure in asphyxia.

Disturbances of the vasomotor system, while in the main reflex phenomena, are so combined and associated with other disorders, and their results are in many cases so similar to those produced



by non-reflex vascular and nervous affections, that this would not be an appropriate place to discuss them all in detail. Thus, certain forms of **cyanosis** are purely reflex vasomotor in origin, while others are due to respiratory disorders, and others, again, are the result of gross lesions of the heart and bloodvessels; they are therefore considered together at p. 114. To disturbance of the vasomotor arrangements must also be attributed **Raynaud's disease**, the most prominent symptom of which, in the fully-developed disease, is a symmetrical gangrene of the extremities (see p. 48). **Oedema** is in many cases an affection of the vasomotor system, and is discussed at p. 260. Abnormal sweating is referred to at p. 401.

**Tache Cérébrale.**—Tache cérébrale may be mentioned. On gently stimulating the skin, as by lightly scoring it with the finger-nail or the blunt end of a pencil, the portion of the skin touched soon becomes bright red, and may be raised in a wheal. This phenomenon (known also as 'dermographism' and 'Trousseau's spots') is due probably to abnormal irritability of the vasomotor nerves, and is seen in meningitis and other diseases of the brain and cord. It is also found in fevers, in functional nervous affections, and may occur in health. Its diagnostic value is therefore small.

**Priapism.**—Erection of the penis is seen in transverse lesions of the cord and lesions of the medulla. It is doubtless due to a vasomotor reflex, whose centre is in the lumbar enlargement of the cord (see p. 93).

In henioplegia due to cerebral lesion vasomotor disturbances may be present. At first an increased warmth is found in the affected limb, which later becomes cold and cyanotic, the earlier condition being possibly due to paralysis of the bulbar vasomotor centre, the functions of which are later assumed to some extent by the subordinate centres in the cord. The coldness and blueness of the paralysed limb may be in part the result of imperfect vasomotor control, and in some measure due to want of muscular contractions.

Transverse lesions of the cord may produce very similar vasomotor disturbances to those just mentioned, in the regions affected by the lesion.

The vasomotor reflex originating in stimuli received by the digestive organs, and resulting in increased arterial tension of the systemic vessels, with dilatation of those in the splanchnic

area, has been credited with the principal share in the causation of **angina pectoris** (Russell).

Reflexes may be observed, both in health and in disease, in connection with the functions of many other viscera, as, for example, vomiting produced by peripheral irritation of various kinds (see Vomiting, p. 557); coughing produced in a like manner (see Cough, p. 106); hiccough (p. 164), etc. The diagnostic value of these symptoms is discussed at the places referred to.

**Summary.**—A reflex act or condition can only occur if the respective reflex arc is intact. The latter consists of a peripheral and a nuclear portion. A co-ordinating and inhibiting influence is probably exerted upon the nuclear portion of the reflex arc by higher centres.

Three groups of reflexes are observed: (1) Deep reflexes; (2) superficial reflexes; (3) visceral reflexes.

Of these the most important for diagnostic purposes are:

1. **The Deep Reflexes**, which may be **diminished** or **abolished** in:

(a) Interruption of the peripheral reflex arc—*e.g.*, peripheral neuritis and sometimes locomotor ataxia.

(b) Interruption of the nuclear reflex arc—*e.g.*, progressive spinal muscular atrophy, infantile paralysis, locomotor ataxia, Landry's paralysis, Friedreich's ataxia.

The deep reflexes may be **increased** and **amplified** in—

(a) Transverse interruption of the cord above the level of the nuclear reflex arc involved. This may be due to (i.) compression of the cord by inflammatory products, tumours, aneurism, injuries, etc.; (ii.) myelitis; (iii.) hæmorrhage.

(b) Sclerotic changes in the upper segment of the motor tract—(i.) Primary lateral sclerosis; (ii.) hereditary spastic paraplegia of children; (iii.) sclerosis of the lateral columns, combined with other lesions of the cord—*e.g.*, amyotrophic lateral sclerosis, ataxic paraplegia, combined sclerosis of Dana, multiple sclerosis, syringomyelia; (iv.) intracranial lesions of the upper segment of the motor tract—hæmorrhage, thrombosis, embolism, tumours, meningitis.

(c) Intoxications: tetanus and strychnine-poisoning.

(d) Functional nervous affections: hysteria, 'railway spine,' tetany, and sometimes after an epileptic fit.

2. **Superficial Reflexes.**—Diminution or loss of these is not of much diagnostic value. Retention of the reflex indicates integrity of the cord at the level of the nuclear reflex arc involved. A

distinct difference in the force of corresponding reflexes on either side of the body indicates central or peripheral nerve lesions. Babinski's sign usually denotes changes in the lateral columns.

3. **Visceral Reflexes.**—(a) Pelvic reflexes, when intact, denote an active and normal function of that region of the spinal cord in which their nuclear reflex arcs are situated, namely, the lumbar enlargement. They are the vesical, rectal, and uterine. Incontinence of urine or of fæces indicates involuntary relaxation of the respective sphincters. This may be due to paralysis of the muscle, from interruption of the reflex arc, or to distension of the viscus, causing the active muscle to give way (this applies especially to the bladder). (See Micturition, p. 195, and Defæcation, p. 116.) (b) Pupil reflex. Various defects may be observed in the pupils' action and condition, which are of assistance in diagnosis—*e.g.*, myosis, mydriasis, failure of light and accommodation reflexes, etc. (see p. 316).

## RESISTANCE.

In the act of palpating the various regions of the body by the hand, as a means of examination, a certain degree of resistance to pressure is observed. In some cases this is the result of undue consolidation of the tissues, as may be seen in cases of inflammatory infiltration or malignant tumours. It is in the examination of the abdomen that the question of resistance is of chief diagnostic interest. Here it is mainly dependent on the amount of contraction found in the abdominal muscles. In some instances this produces a board-like rigidity of the anterior wall of the abdomen, which renders negative any attempt to palpate the contents of the abdomen. This may be seen in neurotic individuals, in peritonitis, and in tetanus. Any painful condition of the intra-abdominal contents may cause an involuntary contraction of these muscles, which thus place themselves on guard over the tender spot. A localized area of resistance of the abdominal muscles should therefore suggest the presence of a painful or inflammatory lesion behind it—*e.g.*, appendicitis, lesion of the gall-bladder, stone in the kidney, gastric ulcer, etc.

A different sort of resistance is that felt by the pleximeter finger while percussing the thorax over a pleural effusion. Here the underlying fluid gives a curious sense of support to the finger, especially if it is placed on an intercostal space.

**RESONANCE.**

A sound, by whatever means produced, may be strengthened and may have various qualities added to it—*e.g.*, tone, pitch, duration, etc.—by the proximity of a **resounding medium**. This in the case of musical instruments is supplied by air cavities as a rule, and in the human voice by the same means. In medical examination of the body it is chiefly the modifications of resonance as produced by percussion of the surface that is of interest. This is considered in the articles on Percussion, General Principles (p. 279); Thorax, Percussion-Sounds of (p. 450); Abdomen, Percussion-Sounds of (p. 15).

By auscultation one also observes varieties in resonance. (See Auscultation, General Principles, p. 50; Thorax, Auscultation Sounds, p. 415; Abdomen, Auscultation Sounds, p. 19; Auscultatory Percussion, p. 52.)

**RESPIRATION, Abnormalities of.**

In the article on Dyspnoea (p. 125) the causes and nature of respiratory disturbances are considered. The respiratory movements of the chest and of the abdomen in disease are discussed in the sections on the examination of those regions, at pp. 10 and 469.

**RETENTION OF URINE.**

The inability from any cause to expel the contents of the bladder. In medical cases this is commonly due an interruption in the course of the nerve impulses from the cortex to the lumbar enlargement, which presumably exercise an inhibitory action on the sphincter vesicæ. In the case of such a lesion the sphincter remains tightly closed until the pressure of the accumulated urine overcomes its strength, and a dribbling overflow ensues (paradoxical incontinence). Retention may be due to atony or weakness of the bladder, the detrusor being unequal to the task of emptying the organ. Mechanical obstructions to the outlet of the bladder may be responsible for retention—*e.g.*, stricture of the urethra, enlarged prostate gland. (See Micturition, p. 197.)

Retention must be distinguished from suppression of urine (*q.v.*, p. 400), in which condition the kidneys are inactive and the bladder is empty.

**RETINITIS.**

There may be observed in the fundus oculi (by means of the ophthalmoscope) a diffuse cloudiness, especially in the central regions; the papilla may be congested, swollen, and indistinctly outlined; the retinal veins may be engorged; bright or dark red hæmorrhagic patches and white exudations and degenerations may be observed. These changes, which are usually bilateral, indicate retinitis or, when the papilla shares in the inflammation, neuro-retinitis. The subject is more fully discussed in the article on Disturbances of Vision, at p. 552.

**RETRACTION OF INTERCOSTAL SPACES.**

Various movements of retraction of some portion of the chest-wall and of the epigastrium, unconnected with respiration, are of diagnostic importance. They are considered in the article on the Movements of the Thorax, at p. 469 *et seq.*

**RHONCHI** (Gr. ῥόγκος, a snoring).

Musical sounds heard over the lungs in catarrhal and other affections of the organs. Every variety of pitch and quality may be heard, depending on the degree of narrowing which exists in the bronchi of different sizes, as a result of thickened mucous membrane, plugs of mucus, etc. (See Thorax, Auscultation, p. 417.)

**RICE-WATER STOOL.**

In Asiatic cholera the stools often consist of serous fluid of a dirty, opalescent, whitish appearance, the so-called rice-water stool. (See Fæces, p. 120.)

**RIGORS.**

The patient complains of chilliness, and a trembling, more or less active, affects his limbs and body. His teeth chatter, and the tremor may be so violent as to cause the bed to shake. The skin of the face and body is pale or livid, and the patient looks and feels ill. The temperature of the surface is low. If taken on the skin at this stage it may be subnormal, but in the mouth, or, better, in the rectum, it is raised above normal. After the lapse of a variable length of time—it may be only a few minutes or it may last longer—the cold stage passes away, and if the fever

be considerable a sense of heat and burning succeeds. Many affections characterized by a chill at the outset with fever are intermittent, the hot stage being followed ere long by sweating and defervescence. This is characteristically the case with malaria, and is to a less marked degree seen in many hectic conditions—*e.g.*, tuberculous abscess, pyæmia, empyema, hepatic and other abscesses.

It is, perhaps, in pneumonia that rigors most consistently mark the onset of acute inflammation. In septic processes of all kinds and in any locality rigors are very often observed, and are especially ominous when they mark the onset of infection of the blood with the organisms of septicæmia or ulcerative endocarditis. It must, however, be remembered that rigors are not in any way a measure of the severity of the attack. As a rule, a rigor means a sudden onset of inflammatory disease, but not necessarily a very severe one.

In addition to the affections already named, rigors often signal the onset of acute rheumatism, variola, influenza, typhus fever. Occasionally any of the other infections which commence fairly suddenly—*e.g.*, chicken-pox, scarlet fever, mumps—may be initiated by a chill. Those affections which in adult life would provoke a rigor are apt to produce convulsions in childhood.

Inflammation of serous membranes, as peritonitis, meningitis, may cause rigors; but these are rarely seen in pleurisy, pericarditis, or joint affections unless there be purulent effusion in the cavity.

Pyelitis resulting from stone or from the presence of the colon bacillus often causes rigors.

Conditions closely resembling rigors are sometimes seen which are in no way connected with inflammatory processes nor with septic absorption: the shivering and teeth-chattering of cold and of emotions (fear, suspense, anxiety); the tremor so constantly observed immediately after parturition is completed. These conditions may be accounted for by disturbances of the nervous mechanism rather than by inflammatory processes. The tremor does not differ in any essential respect from that entering into the group of symptoms known as rigor. It is not, however, accompanied by the other phenomena—*viz.*, there is no paleness nor lividity, the surface temperature is not lowered, and the internal temperature is not raised.

Another group of cases must be mentioned—namely, those

instances of rigor which are caused by the presence of a foreign body in the urethra, the ureter, or the bile-duct. At times it is found that the passage of a catheter through the urethra, especially if some force have been employed, is followed promptly, often within an hour or two, by rigors, with the usual rise of temperature. This was formerly regarded as an example of nervous disturbance. This view is still held by some clinicians, who consider that the appearance of the rigor so soon after the obvious cause is only consistent with a nervous origin of the symptom. Many observers are of the opinion that this form of rigor is in no way different from that which occurs at the onset of other pyrexial states, and is due to the action of micro-organisms and their toxins. The passage of gall-stones through the bile-duct and of a calculus through the ureter may similarly be accompanied by rigors, which have been variously regarded as neurotic or as toxic in their origin. These two latter instances are, however, even more likely than the urethral example to be produced by the absorption of micro-organisms or their toxins.

### **RISUS SARDONICUS.**

A tonic spasm of the facial muscles causing the eyebrows to be raised and the angles of the mouth to be drawn back in an anxious, unmirthful grin. This occurs characteristically in tetanus and in strychnine-poisoning. In the former disease it is an early symptom, the muscles of the jaws and face being affected before those of the trunk and limbs. In strychnine-poisoning, on the contrary, the trunk muscles are first affected, and the face only at a later stage, should the patient survive.

### **ROMBERG'S SIGN.**

Inability to maintain the balance without the aid of sight. The patient is instructed to stand upright with his feet close together, and unassisted by contact with any object. He is then to close his eyes firmly, and if he is defective in co-ordinating power he will sway unduly, and even fall. The diseased conditions giving rise to this symptom are enumerated in the article on Inco-ordination. (See *Movements, Disorderly*, p. 247.)

**ROSEOLAR RASH.** See *Skin Eruptions*, p. 369.

**ROTCH'S SIGN.**

The right border of superficial cardiac dulness normally joins the upper border of liver dulness at a right angle. In cases of moderate pericardial effusion the line limiting the cardiac dulness on the right side tends outward to the right in its lower part, owing to the fact that the pericardial sac is dilated laterally at its lower part by the fluid. The result is that there is dulness in the fifth right interspace near the sternum, and the angle above mentioned is increased to an obtuse angle.

**RUSSO'S METHYLENE BLUE REACTION.**

As a test for typhoid fever this reaction is of considerable value (Rolleston, Gardner Robb). A solution of 1 part methylene blue in 1,000 of water is prepared, of which 4 drops are added to 4 or 5 c.c. of filtered urine. The tube is now shaken, and if the patient is suffering from typhoid an emerald green colour is developed; in negative cases there is little or no colour. This reaction cannot usually be obtained after the commencement of lysis.

**RUSTY SPUTUM.** See *Sputum*, p. 387, and *Hæmoptysis*, p. 157.

**SALIVATION.** See *Ptyalism*, p. 294.

**SALOMON'S TEST.**

The presence of albumin in the stomach contents some hours after it has been thoroughly emptied by lavage. This is suggestive of early cancer of the stomach.

**SCANNING SPEECH.**

A slow, deliberate, measured style of speaking, in which each syllable seems to receive an undue attention; the mode of speech suggests that the speaker is engaged in counting the number of feet in a verse (scansion). It is in multiple sclerosis that this speech defect is best observed; a less typical form of scanning speech is to be seen in the rare affection hereditary or Friedreich's ataxia. (See *Speech, Disorders of*, p. 383.)



**SCAPHOID ABDOMEN (Boat-shaped Abdomen).**

This term is applied to an abdomen retracted in the epigastric and umbilical regions, giving it a hollowed or grooved appearance. This condition is found in cases of meningitis and of cerebral tumours, also in colic, especially in that due to chronic lead-poisoning.

**SCAPULAR LINE.**

A vertical line drawn on the back of the thorax, passing through the inferior angle of the scapula, the arm hanging by the side. It is used in referring to the topography of the chest (see p. 464).

**SCAPULAR REGIONS.**

That portion of the back of the thorax lying between the vertebral border of the scapula and the posterior axillary line; it is divided into a supraspinous region, extending from the spine of the scapula to the apex of the lung, and an infraspinous region, from the spine of the scapula to the level of the inferior angle of the scapula. The abnormalities to be found in these regions are considered in the articles on the Shape, etc., of the Thorax (p. 464), Percussion (p. 450), Auscultation (p. 407), and Pain (p. 271).

**SCAPULO-HUMERAL REFLEX.**

The spinal border of the scapula is struck just above the inferior angle; in most normal cases an external rotation of the arm takes place, as a result of reflex contraction of the supra- and infraspinati muscles. In conditions of exaggerated reflexes this reflex is increased, and, in addition, more extensive movements of the arm occur—viz., abduction of the arm, raising of the shoulder, flexing of the forearm at the elbow, and extension of the fingers (See Reflexes, p. 340.)

**SCOLIOSIS** (Gr. *σκολιός*, crooked).

A lateral curve of the spinal column, combined with rotation of the vertebræ, most commonly the result of a vicious habit of standing or sitting, the supporting muscles of the back being

weakened by debility or want of exercise. A marked degree of scoliosis is often due to rickets, or may be the result of tilting the pelvis to avoid pain or to overcome defects of motility in paralysis, hip-joint disease, or sciatica.

It may result from chronic pleurisy or empyema, or from fibroid plithisis. In these conditions the lung becomes retracted and shrunken, causing one side to fall inwards, and the spine curves laterally, with the concavity toward the affected side. In cases of recent copious pleural effusion the affected half of the thorax may be increased in size, and curvature of the spine may be observed, with the convexity toward the effusion. (See Thorax, Shape, etc., p. 468.)

**SCOTOMA** (Gr. *σκότος*, darkness).

On examining a patient's field of vision certain areas may be found in which the vision is lost or indistinct; these are known as scotomata. In accordance with the position of the blind area in the field of vision, the terms nasal, temporal, peripheral, or central scotoma are employed. (See Vision, Disturbances of, p. 543.)

**SCYBALA** (Gr. *σκήβαλον*, fæces).

A term (used in the plural) to indicate masses of constipated fæces, rounded or irregular in shape. (See Fæces, p. 137, and Defæcation, p. 116.)

**SENSATION, Disorders of.**

Here we shall consider only symptoms indicating disturbances of the organs concerned in the production of sensory phenomena. For further remarks on the diagnostic value of pain see p. 268.

Common and special sensation are brought about and maintained by (a) a suitable organ at the periphery for the reception of impressions from without; (b) nerve fibres which efficiently convey to the sensorium those impressions received by the peripheral organs of sense; (c) an active and sensitive condition of those portions of the brain which receive the sensory impulses. Disturbance of one or more of these parts of the sensory apparatus is followed by disorders of sensation.

The end-organs (a) vary in structure and situation, according

to their function. They are found in the skin, muscles (voluntary and involuntary), tendons, internal organs, serous membranes, retina, internal ear, mucous membrane lining the nasal and buccal cavities, etc. The sensory paths by which the impressions travel to the centre are, firstly, the mixed and sensory nerves entering the cord by the posterior roots, and the cranial nerves of similar character (including the nerves of special sense); secondly, the afferent paths through the brain and cord. On entering the cord by the posterior nerve roots, some of the fibres proceed at once to the substance of Rolando, but the bulk of them ascend by the postero-internal column, giving off numerous collateral branches to the grey matter of the same side, and some via the posterior commissure to the opposite side. Relays of fibres, connected by means of the grey matter with the posterior root fibres, proceed upward in the other ascending columns (the anterior and posterior cerebellar tracts and antero-lateral tracts). The posterior column fibres, conveying impressions of common and muscular sensibility, reach the nucleus gracilis and nucleus cuneatus of the same side in the bulb, thence by the fillet to the opposite corpora quadrigemina and optic thalamus. From these centres fibres reach the cortex, passing through the internal capsule. The cranial nerve impulses, from the respective centres in the medulla, pons, and mesencephalon, pursue a similar course to the cortex. Another pathway, consisting of a multitude of relays of neurons in the grey matter of the cord, is probably the track followed by impulses produced by pain, heat, and cold. The remaining tracts mentioned above form the pathway for impressions reaching the cerebellum.

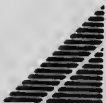
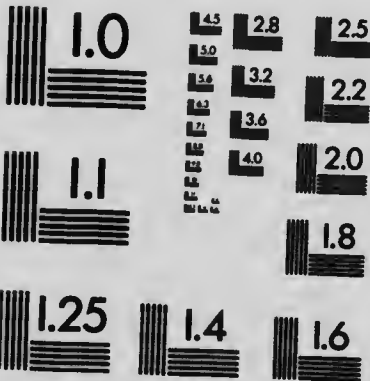
**Varieties of Sensation.**—The end-organs referred to above give rise to sensory impulses of different qualities, the perception of which results in (*a*) tactile and pressure sensibility, (*b*) the sense of pain, (*c*) thermal sensibility, and (*d*) the sense of movement. In addition, the organs of special senses transmit their respective impressions, but their abnormalities are considered elsewhere. (See Disturbances of Vision, p. 543; Smell, p. 372; Taste, p. 402.)

A few terms commonly used to describe sensory symptoms may first be mentioned. Loss of sensibility is termed **anæsthesia**; diminution of sensibility, **hypæsthesia**; increase of sensibility, **hyperæsthesia**, or, if the hyperæsthesia amounts to pain (as it usually does), **hyperalgesia**; a single pin-prick may be felt as



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several painful impressions, **polyæsthesia** (successive or simultaneous); subjective sensations which do not correspond to any stimulus from the outside, **paræsthesia**, such as tickling, cotton-wool feeling, crawling of insects (**formication**), etc.

(a) **Tactile Sensibility** is best tested by closing the patient's eyes and touching him lightly with the tip of the finger or with a dry camel's-hair brush. He should be directed to say 'Now' the moment he feels the touch. His sense of location, which is not the result of separate end-organs or special nerve fibrils, should at the same time be tested by asking him where he is being touched. An instrument for testing the acuteness of common or tactile sensibility, called the 'æsthesiometer,' may be used. It consists of two points, which can be separated measured distances by means of a sliding scale. Both points are placed upon the skin, with any desired distance between the points, and the patient is required to say if he feels one or two points touching him. Normally each point can be distinguished, when only a few millimetres apart, by such sensitive surfaces as the tip of the tongue, the palmar surface of the finger, or the lip, while on the less sensitive localities—*e.g.*, the neck, back, or thigh—the points must be two or three inches apart to be separately felt. Any important deviation from the normal can then be noted in actual measurements, but it must be admitted that the advantage of such an instrument is more apparent than real.

**Pressure sensibility** is practically a form of tactile sensation, and is tested by supporting the limb or other part under examination. Objects of the same size, but of different weights, are then placed on the skin, preferably over a supporting bone, and the patient tells which is the heaviest.

Diminished or lost sensation may be observed in lesions of the sensory apparatus, obstructing partially or completely the passage of sensory impulses. Hence it may be diminished in neuritis (even when pain and tenderness are at the same time present) or injuries of the nerves, and in partial destructive lesions of the cord and brain. Anæsthesia occurs in the more complete destructive lesions of the sensory tract, as in transverse myelitis or pressure paraplegia, and traumatic or inflammatory lesions of nerve trunks. Variable degrees of diminished tactile sensibility are found in other central nervous diseases—locomotor ataxia, cerebral hæmorrhage or softening. In syringomyelia the tactile sensibility is preserved, unless the lesion (cavity) invades the

posterior columns. An interesting instance of anæsthesia and analgesia is the painless trophic affection of the joints (Charcot's joints) found in tabes dorsalis and in syringomyelia. The differential diagnosis between the affections producing anæsthesia depends mainly on the anatomical distribution of the symptom, which in these organic lesions will strictly follow the nerve supply (see table at p. 227). Quite the contrary is the case in the anæsthesia of hysteria. This is one of the commonest symptoms of that disease, and is often restricted to one half of the body (hemianæsthesia). The areas affected do not coincide with the nerve distribution. Another affection, often hysterical, in which anæsthesia may occur is the traumatic neurosis termed 'railway spine'; but the sensory symptoms in functional nervous affections are so variable as to render these signs of very little use for diagnostic purposes.

(b) **The Sense of Pain.**—Increased common or tactile sensibility (hyperæsthesia) must be distinguished from increased **sensibility to pain** (hyperalgesia). The former is found in functional nervous affections, as neuralgia and hysteria, but in these conditions hyperalgesia is more in evidence. The latter is seen typically in neuralgia, in neuritis, in lesions of the spinal cord and its meninges, whereby the posterior nerve roots are irritated, giving rise to the girdle sensation (hyperæsthesia) or to the girdle pain (hyperalgesia). Here belong the lightning pains of tabes dorsalis, referred to the peripheral distribution of the lower dorsal and lumbar spinal nerves, and resulting from disease of the posterior columns of the cord. In hysteria and neurasthenia, hyperalgesia and hyperæsthesia, also paræsthesias may be seen. In some cases of internal disease pain is an important symptom, and may be referred to a superficial spot or area by a sympathetic or reflex process. (See Pain, p. 268.) Many phenomena of this sympathetic or reflex nature are familiar, and a few of the more important may be here mentioned: Parietal pain in middle-ear disease and in some stomach affections; pain in external auditory meatus and over the temporo-maxillary joint in diseases of the tonsils, tongue, and upper molars; tendency to cough in irritation of the external auditory canal; pain in any or all of the branches of the fifth nerve, from a carious tooth or from disease of the frontal sinuses; pain in the shoulder (usually the right) from liver affections; pain in the left shoulder from spleen diseases; pain in the arm (usually the left) from angina pectoris; pain in the back

in stomach diseases; tickling in the nose from intestinal worms; pain in the epigastric or umbilical region from affections of the uterus or vermiform appendix; pain in the knee from hip-joint disease; sensitiveness to pressure of the skin of the præcordial region in heart disease.

Tenderness, or increased sensitiveness to pressure, may exist without subjective pain; it is, however, merely a form of hyperalgesia, and is best seen among nervous diseases in peripheral neuritis. The subject is further considered in the article on Pain (p. 268.)

Delayed sensibility to pain is sometimes observed in tabes dorsalis and in peripheral neuritis; a pin-prick on the toe is felt first as a touch, and after perhaps as long as ten seconds the patient becomes aware of the pain. Here the complicated and interrupted pathway for painful impressions through numerous relays of neurons in the grey matter of the cord is evidently more easily obstructed than the simpler track followed by the impressions of tactile sensibility, through the posterior columns. This is still more marked in the case of syringomyelia. Here the passage of painful (and thermal) impulses through the diseased grey matter may be actually stopped, while the messages of tactile sensibility passing through the comparatively healthy posterior columns may be unaffected. This condition (loss of sensibility to pain, heat, and cold, with retention of touch sense) was called by Charcot **dissociated anæsthesia**.

Inability to localize pain (**allocheiria**) is occasionally seen in locomotor ataxia, multiple sclerosis and hysteria, in which diseases the illusory sensations mentioned above as paræsthesias are sometimes observed.

(c) **Thermal Sensibility** has been incidentally referred to above. It may be tested most conveniently by placing in contact with the patient's skin test-tubes filled with hot and with cold water, requiring him to state whether he feels them to be hot or cold. A less suitable method is to blow upon the patient's skin, with the mouth either widely open (hot) or nearly closed (cold).

Inability to distinguish heat from cold is most typically seen in syringomyelia, where dissociated anæsthesia frequently occurs; in multiple sclerosis, in hysteria, and in tabes dorsalis, a loss of thermal sensibility may occasionally be found.

(d) **Sense of Muscular Power**.—The judgment of the amount of



muscular effort which is at any moment being exerted, or 'feeling of innervation,' may be tested by placing weights in a sling, suspended by a broad band from the hand and forearm of the patient; he tells when he recognizes a difference in the weights he supports. This sense of strength or of innervation is present in healthy individuals, who can by practice discriminate very slight differences in weight. It is lost or diminished in locomotor ataxia, and in other conditions producing inco-ordination (see p. 246).

(e) **Sense of Movement and Position.**—What has commonly been termed the **muscle sense** is in reality a combination of the last-mentioned variety, the **strength sense**, with the **touch sense** (skin, joint surfaces, etc.); it is therefore better named the **kinæsthetic sense**. By it we become aware of the movements and of the position of our limbs and body. This perception is to be tested by directing the patient to perform certain movements with his limbs while his eyes are closed, and to describe their position after active or passive movements have been accomplished.

Diminution or loss of this sense is the chief feature in ataxia (see p. 246), and is observed in locomotor ataxia, cerebellar diseases, etc.

(f) **Stereognosis.**—The recognition of the form and qualities of an object is also a combination of senses; the perceptions of touch, temperature, muscular action, are combined in the brain, with the aid of experience, to produce the judgment of shape, form, etc.

(g) **Sensation of Vibration.**—It has been shown recently by Egger that vibrations produced by a tuning-fork placed over a bone give rise to a perception which is independent of cutaneous tactile sensibility, but is really a bone sensibility. An osseous anæsthesia is described as occurring in tabes dorsalis, syringo-myelia, myelitis, and other affections, and osseous hyperæsthesia is stated to occur in tabes and other morbid conditions. Egger's conclusions have been controverted in certain particulars.

### SIDE-STERNAL LINE.

A vertical line drawn on the surface of the chest over each border of the sternum. It is used in referring to the topography of the chest. (See Thorax, Shape, etc., p. 464.)

## SKIN ERUPTIONS

Symptomatic eruptions only here considered—The varieties of skin eruptions to be observed :

- (a) Erythema, due to scarlatina, rōtheln, small-pox, typhoid fever, erysipelas, measles, sepsis, intestinal disturbance, enemata, syphilis, rheumatism, erythema nodosum, dengue, drugs, vasomotor disturbance (tache cérébrale).
- (b) Roseola : typhoid fever, syphilis, relapsing fever.
- (c) Papular and macular eruptions, due to measles, rōtheln, small-pox, leprosy, glanders, syphilis, drugs
- (d) Vesicles, due to chicken-pox, small-pox, herpes facialis, herpes zoster, sudamina, drugs.
- (e) Urticarial eruptions: digestive disturbances, enemata, septic absorption, antitoxins, drugs
- (f) Pustular eruptions in chicken-pox, small-pox, drugs.
- (g) Hæmorrhages seen in purpura, scurvy, hæmophilia, leukæmia, splenic anæmia, typhus fever, measles, small-pox, drugs.
- (h) Inflammations of the skin : gout, drugs, syphilis.
- (i) Desquamation.
- (j) Mottling.
- (k) Pigmentation.

Skin affections or eruptions are only considered here as symptoms of diseased conditions or disturbances of the body generally. Purely local skin diseases are therefore not discussed.

The eruptions or rashes which occur as symptoms of bodily disease may be observed in the various forms of erythema, roseola, macules, papules, vesicles, pustules, hæmorrhages, wheals, squamæ, dermatitis (eczema). In addition, pigmentation and desquamation may be mentioned.

(a) **Erythema.**—A red coloration of the skin, usually in large patches or areas. It may be seen in the following conditions :

1. **Scarlatina.**—The rash is often punctate ; appears on the second day of the illness. Is first seen on upper part of chest or neck and face ; the region round the mouth may escape ('circumoral pallor'). The eruption is well marked at the flexures of joints.

2. **Rōtheln (German Measles).**—Red spots, which usually remain distinct on the face, where they first appear, but coalesce on the body into erythematous patches. The rash appears on the second day of the disease.

3. **Small-pox.**—Severe attacks often have an erythema of the

abdomen and limbs (especially marked on the extensor surfaces) before the characteristic variolar eruption appears.

4. A similar premonitory rash may occur in **Typhoid Fever**.

5. **Erysipelas**.—A bright red eruption, with sharply defined edge, appearing within twenty-four hours after the beginning of the illness.

6. **Measles**.—A blotchy erythematous or macular eruption may precede the characteristic measly rash.

7. **Sepsis**.—An erythematous patchy or spotted rash may result from septic absorption. It may be found on the legs or arms, also on the face and trunk.

8. **Intestinal Disorders** may produce a similar eruption.

9. **Enema Rash**.—After a soap-and-water enema an erythematous eruption sometimes occurs. It is said only to follow the use of hard soap; if potash soap be used it does not occur. It is a rare occurrence in any case, and is more likely to happen with female patients.

10. **Syphilis**.—Inherited syphilis is often characterized by a red rash, commencing on the buttocks of the infant and spreading over the body. Acquired syphilis may show an erythematous, or more frequently a macular or papular, eruption; appears first on the abdomen, spreading over the thorax, rarely on the face or hands. The rash is symmetrically disposed, is free from pain or itch, and occurs about six weeks after the appearance of the primary sore.

11. **Rheumatism**.—Erythematous eruptions of much variety of form (erythema multiforme) may be seen on the trunk and limbs.

12. **Erythema Nodosum**.—Red, raised, painful areas may occur (often associated with rheumatism), the favourite regions being the front of the leg below the knee and the extensor surfaces of the fore arm.

13. **Dengue**.—A red rash is seen on the skin.

14. **Drug Erythema**.—Red eruption may be seen to follow the ingestion of drugs in certain cases. In many instances the eruption is an evidence of an idiosyncrasy on the part of the patient, rendering him intolerant of the drug in question. Only those instances of drug eruption which have been observed on repeated occasions need be here referred to: Antipyrin, sulphonal (often accompanied by pruritus), iodine and the iodides, iodoform, bromine and the bromides, opium and its derivatives, not infre-

quently cause erythema; belladonna may cause a rash resembling scarlatina; the rash from cubebis and copaiba is sometimes an erythema, but is more frequently a macular or measy rash; quinine may produce a rash commencing usually on the face, and followed by desquamation; chloral, arsenic, and mercury all



FIG. 61.—ERYTHEMA NODOSUM.

The eruptions is copiously developed on the legs and sparsely on both fore-arms, the latter very indistinctly shown in the photograph.

produce erythema at times, commencing, as a rule, on the face, neck, or chest; salicylate of soda, chlorate of potash, turpentine, tar, boric acid, benzoic acid, strychnine may be the cause of an erythema on various parts of the body. The injection of anti-

diphtheritic and other serums is not infrequently followed by a red blush or by a spotted rash.

15. **Tache Cérébrale** and **Tache Spinale** are the red marks of capillary dilatation, produced by the irritation of local pressure or friction, as by scoring the skin with the finger-nail. In cases of cerebral or of spinal meningitis the red marks are unduly distinct, and unusually prolonged in their duration.

A similar, but perhaps less distinctly marked, tache is to be seen in typhoid and other fevers, so that the diagnostic value of the sign is only moderate. (See Reflexes, p. 346.)

(b) **Roseola**.—The characteristic eruption of **typhoid** is the best instance of this form of eruption. It consists of small round rose-red spots, 2 to 4 millimetres in diameter, which disappear on pressure (lay a glass spatula or microscopical slide on them and they disappear). They are best and earliest seen on the abdomen, also on the lower part of the thorax, both back and front; less frequently on the upper part of the chest and on the limbs. They make their appearance about the beginning of the second week of the fever (the seventh to the tenth day), and may occur in successive crops. Each spot lasts two or three days and then fades. They may reappear with the advent of a relapse. Each spot is slightly raised, and may at times show signs of hæmorrhage—viz., it may persist on pressure, and on fading it may take on the colours seen in an ecchymosis.

The secondary eruption of **acquired syphilis** sometimes assumes the form of a small roseolar eruption, which, however, has a tinge of brown, absent from the typhoid spots.

**Relapsing fever** may show similar but rather smaller rose-red spots.

(c) **Papular and Macular Eruptions**.—Spots or blotches, raised somewhat above the level of the surrounding skin. They are usually red and congested from capillary injection, disappearing to a great extent on pressure. The eruption is seen in the following abnormal conditions:

1. **Measles**.—Clusters of red spots, often crescentic in shape and slightly raised, appearing on the third or fourth day of the illness, first on the face, then spreading downwards. They disappear on pressure, and last for four or five days. In severe cases the spots are darker, and even purplish, from extravasated blood. Slight desquamation usually follows the eruption.

Inside the mouth the eruption may also be seen. On the soft

palate it appears (often earlier than the cutaneous) as red, slightly raised spots; on the inner side of the cheeks and lips whitish or bluish white spots, surrounded by a red areola (**Koplik's** or **Filatow's spots**); they are best marked opposite the molar teeth, and may often be seen one or even two days before the appearance of the skin rash.

2. **Rötheln (German Measles).**—Red spots, disappearing on pressure. They are arranged in clusters and slightly elevated; each spot varies in size from 1 to 10 millimetres. They first appear on the face and scalp as a rule, on the first or second day of the fever. Spreading rapidly, some of the clusters coalesce, forming blotches of erythema, but those on the face remain discrete. The rash disappears in five or six days, and desquamation sometimes occurs.

3. **Small-pox.**—Shotty papules may be felt and seen about the third day of the disease. In the earliest stage their favourite seat is the forehead and wrists.

4. **Leprosy.**—This rare disease may be mentioned as causing pigmented or, on the contrary, bleached spots or macules.

5. **Glanders.**—About a week or so after infection an eruption of red papules appears, turning later into vesicles, bullæ, and pustules.

6. **Syphilis.**—See above (p. 363).

7. **Papular and Macular Eruptions due to the Ingestion of Drugs.**—Antipyria may cause a macular rash which may closely resemble measles. It is sometimes purplish in colour and of hard consistency. Arsenic may also cause a rash simulating measles. The copaiba rash is itchy, and may resemble measles both in its character and distribution. Iodine and the iodides, and, more rarely, bromine and the bromides, may cause a papular or macular eruption. Pustular eruptions are the commoner. The papular form is usually due to a long-continued use of the drugs. Salicylic, boric, and benzoic acids may occasionally give rise to papular or macular eruptions. Similar skin disturbances may follow the subcutaneous injection of diphtherial and other antitoxins.

(d) **Vesicular Eruptions.**—Blisters, which may occur singly or in groups. The following are the commoner instances of vesicular eruptions occurring in the course of general disease:

1. **Chicken-pox.**—In this affection papules begin to appear

on the second day of the disease, quickly becoming vesicles, and, later, pustules. The spots appear first on the face and trunk, and (unlike small-pox) only on the arms and hands when they are freely developed on the body. They are separate, may be found in all stages of development in the same patient, and are not accompanied by severe constitutional disturbance.

2. **Small-pox.**—The shotty papules (see p. 366) becomes vesicles about the sixth day of the disease.

3. **Herpes Facialis.**—In various feverish conditions groups of blisters may be found on the face, and chiefly near the mouth. They ultimately become pustular, and dry up with the formation of scabs. The affection in which this most constantly occurs is pneumonia. It is said that the pneumonias which are marked by profuse herpes as a rule do well. This rule is subject to many exceptions. In simple catarrh of the upper respiratory passages, as well as in bronchial catarrh, herpes facialis is often seen. It is at times, though rarely in profusion, seen in typhoid fever. In epidemic cerebro-spinal meningitis it has also been observed.

4. **Herpes Zoster.**—Groups of vesicles following the course of one or more cutaneous nerves.

5. **Sudamina** are minute blisters, found in conditions characterized by profuse sweating—*e.g.*, rheumatism, phthisis, opium-poisoning.

6. **Vesicular Eruptions due to Drugs.**—The iodides and bromides may give rise to vesicular eruptions. This is not a very common form of iodide or bromide rash, and is generally the result of long-continued use of the drug. Frequently the blisters become very large (*bullæ*). In chronic arsenical poisoning vesicles may appear on the face, hands, and neck. Salol occasionally causes a vesicular eruption. Rarely vesicles may be caused by boric acid, by salicylic acid, or by *copaiba*.

(*e*) **Urticarial Eruptions (Wheals).**—As a symptom urticaria most frequently indicates some disturbance of the digestive tract. It is also seen at times after the administration of a soap-and-water enema (see above, p. 363). **Septic absorption** from the surface or from the gas o-intestinal tract may give rise to an urticarial eruption. A similar result may follow the subcutaneous injection of diphtherial and other **antitoxins**.

**Urticarial Drug Eruptions.**—These are not uncommon, and may

at times be observed in susceptible individuals after even moderate doses of the following: antipyrin, salol, sulphonal, phenacetin, laxoin, and other synthetical products. The copaiba rash is often distinctly urticarial. Bromine, iodine, quinine, opium, salicylic acid, and their derivatives; boric acid, antimony, santonin, arsenic, chloral, and digitalis, may all occasionally be the cause of this form of eruption.

(f) **Pustular Eruptions.**—As an evidence of general disease or poisoning pustules are not very frequently met with. They occur in **chicken-pox** as a consequence of the vesicles turning turbid and purulent a couple of days after the blisters appear. In **small-pox** the vesicles become pustules about the eighth or ninth day of the disease. In **furunculosis** the condition is more local, but depends not only on the presence of pyogenic microorganisms in the skin, but also on a low opsonic index. (See Blood Examination, p. 81.)

The following instances of **pustular drug eruptions** may be mentioned: Arsenic often gives rise to an acne-like eruption. Bromine, iodine, and their derivatives cause eruptions of various forms, of which the pustular is the commonest, it occurs as spots, resembling those of acne, both in appearance and, to some extent, in distribution. The face, neck, chest, shoulders, back, and arms are often involved, the legs less frequently, though the dorsum and sole of the foot and the palm of the hand are at times invaded. Chloral and antimony may occasionally be the cause of a pustular rash. The local application of croton oil or of chrysarobin must be borne in mind as a cause of pustules.

(g) **Hæmorrhagic Eruptions.**—Blood may be extravasated in large quantity into the cellular tissue, giving rise to extensive patches of discoloration (ecchymoses), or may ooze from the capillaries, forming small spots, red or dark in colour, which do not disappear on pressure, and which change colour as they fade (petechiæ), or may form streaks of similar character (vibices); or the extravasated blood may separate the layers of the skin, and so form a 'blood-blister,' or may find its way into blisters already formed. The chief conditions in which hæmorrhages into the skin are observed are as follows:

1. **Purpura**—petechiæ, vibices, and ecchymoses generally distributed over the body, but best marked as a rule on the legs. The condition is either an idiopathic and so-called primary



disease (*purpura simplex*), in which cases it is often confined to the legs, or it may be a feature in more obviously toxæmic conditions, such as pyæmia, septicæmia, ulcerative endocarditis, gonorrhœal infection, bubonic plague, epidemic cerebro-spinal meningitis, and other grave toxic states. A severe form of idiopathic purpura is the so-called *purpura hæmorrhagica* or *morbus maculosus*



FIG. 62.—IODIDE RASH.

The patient, suffering from arterio-sclerosis and aortic incompetence, had taken less than  $\frac{1}{4}$  drachm of iodide of potash (in 5-grain doses thrice daily) when the eruption appeared.

of Werlhof, in which not only extensive cutaneous and subcutaneous bleeding takes place, but the blood escapes from the vessels in every region, both internally and externally. A form of purpura is constantly associated with articular rheumatism (*purpura* or *peliosis rheumatica*, or *Schonlein's disease*). In

addition to the purpuric eruption and the arthritis, one finds in some cases, chiefly among children, signs of gastro-intestinal disturbance (pain, vomiting, diarrhœa), the symptom-complex being known as **Henoch's purpura**.

2. **Scurvy** shows petechiæ and ecchymoses in the skin, subcutaneous tissues, and mucous membranes.

3. **Hæmophilia, leukæmia, splenic anæmia**, and other diseases of the blood and blood-forming tissues, may give rise to hæmorrhagic eruptions.

4. **Typhus Fever**.—The eruption appears about the fifth day of the disease, and consists of small red and dark red spots, which occur first on the upper regions of the thorax or on the abdomen. Thence they spread rapidly over the rest of the trunk, the limbs, including the backs of the hands, and only slightly on to the face. When it first appears, a spot is rose-red, and disappears on pressure. As it develops it becomes more difficult, and finally impossible, to obliterate it by this means.

5. **Measles**.—In severe cases the measly rash is dark and livid in colour, due to hæmorrhage into the spot—the so-called **black measles**.

6. **Small-pox**.—The characteristic eruption of the disease may, like that of measles just mentioned, be modified by the appearance of blood in the pustules, giving them a dark or dusky aspect; or the typical rash may be preceded by an erythematous (see p. 363) or hæmorrhagic eruption on the skin and mucous membrane. Such hæmorrhagic forms in both measles and small-pox indicate a severe toxic affection, and are usually of grave import in prognosis.

7. **Hæmorrhagic Drug Eruptions**.—The pustules and vesicles of the iodide rash are at times filled with extravasated blood, and petechiæ may also form. Antipyrin and sulphonal have produced at times a petechial eruption.

(7) **Inflammatory Affections of the Skin** (dermatitis and eczema of various forms) are more commonly of local than of general origin. A dry and scaly form of eczema is frequently seen in **gouty subjects**. **Arsenic** and the **iodides** may also be the cause of a similar eruption. Scaly eruptions are of frequent occurrence, but are also usually of local nature. **Syphilis**, and the occasional squamous eruption following the use of **arsenic** or **borax**, are instances of general disturbances giving rise to this type of rash.

(i) **Desquamation**, or peeling of the superficial layers of the skin, follows most inflammatory or hyperæmic conditions of the skin. In most cases it is seen as a fine branny scurf, which may be rubbed off the skin as a dry powder. This is the condition of the skin after the eruption of measles, rōtheln, erysipelas, or septicæmia has faded. In the case of scarlet fever, however, and exceptionally in that of some of the other erythematous eruptive diseases, the cuticle peels off in large masses, especially in those situations where the skin is thick, such as the palms of the hands and soles of the feet.

(j) **A Mottling of the Skin** is sometimes seen in typhus fever and in septic conditions.

(k) **Pigmentation**. See p. 286.

### SKODAIK RESONANCE.

On percussing immediately below the clavicle in a case of pleural effusion, and on the same side as the effusion, the note is often observed to be clearer and of a higher pitch than on the opposite healthy apex. This is due to a relaxation of the lung owing to the presence of fluid, which occupies a portion of the intrathoracic space, and so permits the tense lung to relax. It is found that moderate tension of an air-containing cavity produces a better tone than a condition of forced stretching (see p. 454).

**SLEEPINESS.** See **Unconsciousness**, p. 498.

### SLEEPLESSNESS (Insomnia).

Inability to sleep is a symptom common to many disorders. It may, indeed, be in some degree habitual, and in such cases depends partly on temperament and partly on occupation and habits. The man who is accustomed to sufficient exercise in the open air, and who is moderate in the use of tobacco, coffee, and tea, is not as a rule troubled with insomnia, unless his nervous organization is oversensitive, or unless one of the following affections is present: fevers of nearly all descriptions, meningitis, melancholia, and other forms of mental disturbance, intracranial syphilitic lesions, delirium tremens, gout, itching or painful affections of the skin, and pain in any region of the body.

**SLURRING SPEECH.**

A type of speech defect best marked in general paralysis of the insane, in which there is a stumbling over the syllables, and a special difficulty is experienced in dealing with the labials and dentals. It is also to be heard in a less typical form in bulbar and pseudo-bulbar paralysis. (See Speech Disorders, p. 382.)

**SMELL, Abnormalities of.**

Defects in the sense of smell are of but minor importance in diagnosis, as they generally result from local affections of the nose. Loss of the sense (**anosmia**) may exceptionally be due to lesion of the olfactory bulb or of the intracranial connections of the nerve. It may also occur as a consequence of lesion of the fifth nerve, which diminishes the secretion of the nasal mucous membrane. The resulting dryness interferes with acuteness of smell. It may be lost in some of the degenerative affections of the central nervous system (*e.g.*, locomotor ataxia), or may be a symptom of hysteria.

Excessive acuteness of the sense of smell (**hyperosmia**) is an occasional feature in neurotic and hysterical affections.

Perversions of the sense (**parosmia**) are sometimes observed in mental affections, and (rarely) after head injuries. It is not unusual for the aura of epilepsy to assume this form (see p. 49).

**SNUFFLES.**

Children suffering from nasal obstruction may be observed to have a snuffling habit of breathing. This may arise from one or other of the numerous forms of narrowing of the nasal passages. If it occur in infancy, it is suggestive of syphilis.

**SOMNOLENCE.** See Unconsciousness, p. 498.

**SORDES.**

In states of prostration and exhaustion, such as may be seen in severe typhoid or typhus fever, or in the typhoid state from any cause, the mouth is found to be in a filthy condition, which is difficult to remedy, and is a source of suffering and discomfort to the patient. The tongue is hard and dry, and thickly coated:

the teeth, gums, and lips are covered with **sordes**—*i.e.*, flakes of brownish or black colour, composed of particles of food, epithelial debris, dried mucus and saliva, and micro-organisms.

### SPADE-HAND.

An enlargement of the hands, causing them to present a thick, coarse, square shape. When the hypertrophy obviously depends on enlargement of the bones, it is likely to be an evidence of **acromegaly**; if the soft tissues are chiefly involved, the deformity may be part of the disturbances due to **myxœdema**.

**SPASM.** See **Increased Muscular Action**, p. 249.

### SPASTIC GAIT.

In walking there is a stiffness of the legs, owing to a difficulty in bending the joints. The rigidity is due to a spastic condition of the muscles, the extensors being chiefly affected. In advancing the limb the toe is scraped or brushed on the ground. The symptom is observed in affections of the lateral columns of the cord, of which the most commonly occurring are multiple sclerosis, primary and secondary lateral sclerosis, and amyotrophic lateral sclerosis. The subject is further considered in the article on Gait (p. 149).

### SPEECH, Disorders of.

Organs of speech—Cortical centre for speech movements—Motor speech centre—Sensory speech centres: auditory speech centre; visual speech centre—Motor writing centre—Course of nerve impulses giving rise to speech—Deaf-mutism—Aphasia—Methods of testing speech defects—Paraphasia—Cortical sensory aphasia—Alexia—Agraphia—Subcortical sensory aphasia—Apraxia—Cortical motor aphasia—Paragraphia—Transcortical motor aphasia—Summary of aphasia—Anarthria—Slurring speech—Scanning speech—Stammering—Lisping.

Efficiency in the power of speech requires a healthy and active condition of the muscular and other structures forming the organs of speech, and an equally normal state of the regions of the brain, spinal cord, and peripheral nerves, which have the function of evoking the action of the speech organs.

The instruments for the production of speech need not be described here in any detail. They consist, briefly, in an organ

for the production of voice, the larynx. (Disorders of the voice are considered in a separate article, at p. 555.) The organs of respiration have to be under proper control in order to elicit sound from the vocal cords at the moment it is required. Articulation is accomplished by means of the movements of the jaws, tongue, palate, lips, and cheeks.

The nervous impulses which call forth muscular contractions of the structures involved in the very complicated act of speech emanate from that portion of the Rolandic area of the cortex which lies at the lower end of the ascending frontal convolution of both sides of the brain, the **cortical centre for speech movements**; thence they proceed peripherally to their respective centres or groups of nerve cells in the pons, medulla, and anterior cornua of the cord. In discussing the subject of Reflexes it is pointed out at p. 336 that voluntary movement partakes largely of the nature of a reflex act, and that the centres in the Rolandic area are stimulated by a sense of movement which impels them to emit impulses to the centres in the anterior horns of the spinal cord, etc. This principle of nerve impulses proceeding to the periphery from the sensori-motor cortical area at the bidding of afferent stimuli holds good in the mechanism of speech production. The centres for speech movements at the lower end of the ascending frontal convolution on each side receive their orders from the **motor centre for speech**, situated in right-handed persons at the hinder part of the third left frontal convolution (Broca's convolution), and at the opposite side in left-handed subjects. (See Fig. 30, p. 206.) (P. Marie has recently brought forward serious objections to this generally accepted view as to the function of Broca's convolution. Until, however, the older theory is definitely disproved, it may be taught as stated in these pages.) The function of this centre is to receive, store, co-ordinate, and properly distribute to the appropriate portions of the centre for speech movements a multitude of messages or impressions acquired originally from the ears, eyes, and other receptive organs, but modified by their detention in various psychical centres which have been developed in the child's brain as it learns to speak. A newborn infant in sucking, crying, etc., uses the muscles which are later to be employed also in speech. In due time it attempts to repeat the sounds it hears spoken by those around. These sound impressions reach its brain, and are recorded in the **auditory speech centre**, which is thus formed in the left first temporal convolution. Connecting or association fibres join this centre with the motor

speech centre in Broca's convolution, which is gradually educated into the co-ordinating centre above referred to. Mimetic sounds thus formed by the child are at first purely mechanical, without an attempt at conception or understanding on its part. By degrees, however, the accumulated memories of sounds are formed into conceptions, associated by the child with certain objects or ideas, and result in the reproduction of intelligible or remembered sounds.

Impressions reaching the brain from the retina go to educate a cortical **visual speech centre**, situated in the left supramarginal and angular gyri, whence stimuli proceed by communicating fibres to the motor speech centre in Broca's convolution. Similarly, in time sensory impressions of all descriptions exert their influence on the motor speech centre, and, possibly, through psychological sensory centres similar to those of hearing and vision. These two are, however, the only centres of the kind at present definitely located.

Closely connected with speech, and in some respects inseparable from it, are reading and writing. Visual and auditory impressions are stored in the sensory speech centres, which, by education, result in the recognition of written symbols and the power to reproduce them in writing. There is some difference of opinion as to the existence of a **motor writing centre** separate from the motor speech centre. Recent observations point to the second left frontal convolution as the situation of such a centre (Monro), but it is held by many competent authorities that the motor speech centre and the motor writing centre are one and the same, the location of which is the third left frontal convolution.

Conscious speech is the result of drawing on the psychological speech centres, of which the auditory is the most important, for memories stored there. Thence they are transferred to the motor speech centre, and onward to the cortical centres for movement, whence they issue as motor impulses traversing the motor tract to innervate the muscles concerned in the act of speech. This course is shown diagrammatically in the table on p. 376, in which also appear the paths of sensory impulses coming from the peripheral sensory organs to the psychological centres.

In studying disorders of spoken and written speech we bear in mind that they are the result of lesions occurring in one or more of the stages through which the nerve impressions pass, or in the organs of speech themselves (mouth, larynx), or that they are the result of functional disturbance without obvious anatomical lesion.

COURSE OF THE NERVOUS IMPULSES CONCERNED IN THE PRODUCTION OF SPEECH

Lesions causing interruption in Stage 1, if they involve the sense of hearing, produce—

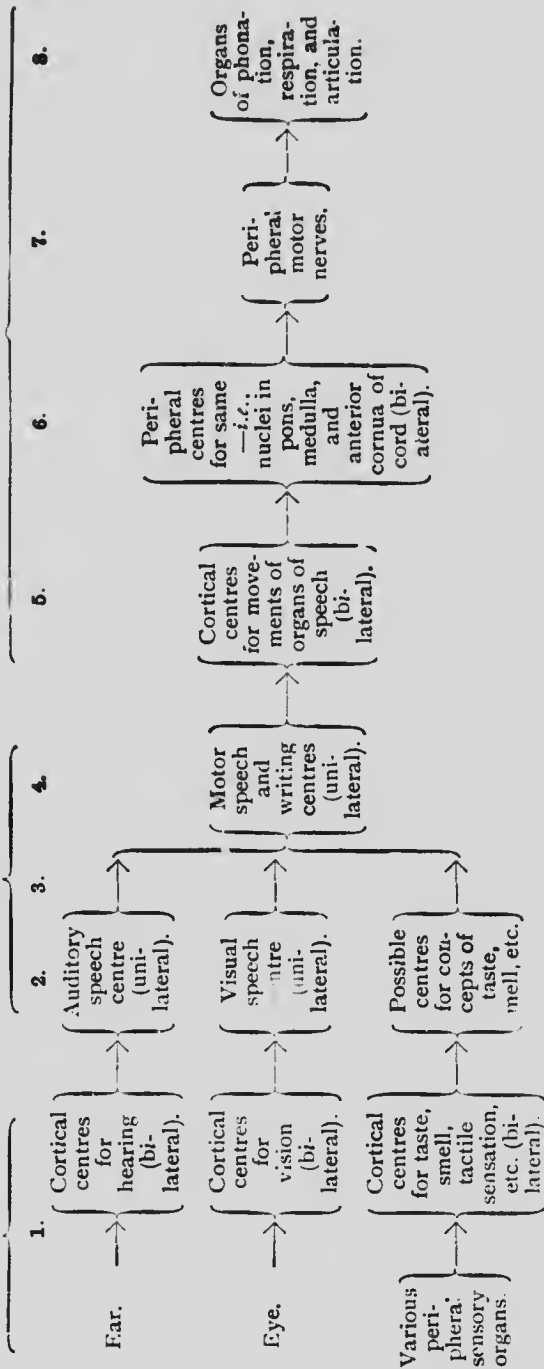
Lesions which involve Stages 2, 3, and 4 cause—

Lesions which involve Stages 5, 6, 7, and 8 give rise to—

DEAF-MUTISM.

APHASIA.

ANARTHRIA.





The stages referred to (see table) are—(1) The ears and their connections with the centres in the temporal lobes for the **perception** of sound, and to a less extent various other sensory paths to the brain; (2) the centres for **conceptions** and memories of sounds, visions, tactile and other sensory impressions; (3) the innumerable association fibres and tracts which connect these regions with each other and with the various centres of the brain, and especially with the motor speech centre in Broca's convolution; (4) this last-named centre and the fibres connecting it with (5) the cortical centres for producing muscular contraction; (6) the connections of the latter with the nuclei in the pons, medulla, and anterior cornua of the cord; (7) the peripheral motor nerves involved in the act of speech; and, finally (8), the organs of respiration, phonation, and articulation. The functional speech defects complete the list.

If the lesion affect (2), (3), or (4) the condition is known as **aphasia**. In this situation even a lesion of slight extent will greatly impair or abolish the ability to speak, read, or write, or to understand spoken or written words, while movements of the tongue, lip etc., unconnected with speaking, will be normal. Should the lesion be situated in the cortical centres for movement (5), or any portion of the mechanism peripheral to that region, the resulting speech defect is termed **anarthria**. Here the impairment of speech is likely to be less complete than in aphasia, as the innervation is bilateral, and the defect often occurs as a clipping or mutilation of syllables, or other imperfection of pronunciation. In anarthria, moreover, the tongue, lips, and palate are inefficient, not only in their speech movements, but also in any other action which they may be called on to perform.

We are now in a position to consider the various speech disorders, which may be divided into three groups: (1) Deaf-mutism, (2) aphasia, (3) anarthria.

1. **Deaf-mutism** is the result of absence of the educational and oft-repeated auditory impressions which are necessary in order that the auditory speech centre may be developed. Without this centre the individual can form no conception of the relation of sounds to objects or written words. The condition is found in those who are congenitally deaf, or who have become deaf in childhood before they had advanced far in the art of speech. Even as late as six years old the occurrence of deafness has been known to cause dumbness, owing to the child having

forgotten all it had learned of speech. Deaf-mutes may be taught to produce an imitative speech by copying the lip and mouth movements which they see in their teacher, and an intelligible, though monotonous and expressionless, speech results.

2. **Aphasia** is the result of injury to the sensory or motor speech centres, or to their association pathways connecting them with many important regions of the brain—viz., (2), (3), and (4), as referred to above.

Before proceeding to put the patient's powers of speech to the test, his sight and hearing should be investigated, so as to exclude fallacies from these causes. Also it should be ascertained if there is any paralysis or defective power in the muscles concerned in speaking and writing, and, further, one must ask if he is right-handed.

A consideration of the points mentioned above (p. 377) will usually determine whether the condition is aphasia or anarthria. In the latter event the nature of the imperfection of speech is to be studied, and, if possible, identified with one or other of the conditions mentioned below (p. 379). Should the case appear to be one of aphasia, the following are the chief points to be investigated: Can the patient (i.) speak voluntarily and without prompting? If so, does he (ii.) misplace, misuse, or mispronounce any of his words? Can he (iii.) read aloud; (iv.) repeat words after the observer; (v.) write spontaneously and correctly; (vi.) write to dictation correctly; (vii.) write from copy; (viii.) read and understand what has been written? In the last four tests his writing and comprehension of figures must be observed. Can he (ix.) understand spoken words? (x.) Is he able to recognize familiar objects and substances by means of his various senses—*i.e.*, can he make proper use of them, or name them, judging them by their appearance, smell, taste, sound, or feeling? The inability to perform this feat successfully is known as **apraxia**. (xi.) Can he tell the number of syllables in a spoken or written word? His power over the individual letters of print or writing may be tested. Thus he may be asked to write out the alphabet, to copy letters and figures, and to write them to dictation, to recognize spoken or written letters, etc.

In testing the writing of a patient whose right arm is paralysed (as it frequently is under these circumstances), he may be asked to attempt 'mirror-writing' with the left hand, or he may be able to construct words out of movable printed letters.

A great variety of aphasic defects in speech may be observed,

presenting the utmost difficulty in elucidation. A certain proportion of the cases conform fairly well to definite types of aphasia, but many cases are so mixed and modified by the diversity of the organs injured by the lesion that a definite conclusion, based on speech defects, as to the nature and situation of the lesion cannot be attained. The following are some of the principal types of aphasia from which one may obtain aid in diagnosis:

(a) The patient cannot understand spoken or written words. He cannot speak connectedly, though he may utter irrelevant or mispronounced words (**paraphasia**). He cannot read, nor can he write spontaneously or to dictation, though he may copy in the manner of one who draws unintelligible symbols.

Such a condition indicates destruction of the auditory speech centre, from which memories and conceptions, not only of spoken, but also of written, speech are drawn. In the case of those exceptional individuals whose memory of written words is mainly dependent on visual impressions, destruction of this centre would effect but little impairment of their powers of reading or writing.

The lesion causing this type of aphasia involves the more or less complete destruction of that portion of the left first temporal convolution in which the centre is situated, and may be due to cerebral hæmorrhage, embolism, abscess, tumour, depressed fracture of the skull, etc. The aphasia is termed (because of the situation and nature of the disturbance) **cortical sensory aphasia**.

(b) The patient can understand spoken language, and can express his thoughts in speech perfectly, with the exception of slight paraphasia in some cases. He is, however, completely at a loss as regards written (or printed) language. He is unable to read (**alexia**). It is not that he cannot say the words which the written symbols mean, for they mean no more to him than if they were written in Chinese, and he has no memory of having ever attached any intelligible significance to the characters. This is the condition known as **word-blindness**. He cannot write (**agraphia**) even to dictation, and it is doubtful if he can copy writing as a drawing exercise.

Destruction of the visual speech centre in the left supramarginal and angular gyri is here indicated. Ideas and memories imprinted there by visual impressions are lost, so that he is unable to recognize the form of the letters and words, and he has no recol-

lection of the form and meaning of the characters by which he might express his thoughts. His memory of the sound of words is, however, unaffected. The causes mentioned in the last type of aphasia may also operate here, and the condition is another instance of cortical sensory aphasia.

(c) He cannot understand spoken words (**word deafness**), but is able to speak spontaneously and correctly; he cannot, however, repeat words and sentences spoken by another, as they convey no more meaning to him than if they were in an unknown foreign tongue.

This is the condition produced by a lesion in the white structure of the brain immediately below the cortical auditory speech centre, interrupting the paths of afferent impulses from the cortical centre of hearing on both sides of the brain, but not damaging the auditory speech centre. The spontaneous activity of the speech-forming mechanism is not impeded, but its receptive functions are at an end. The condition is known as **subcortical sensory aphasia**.

(d) He has word blindness; he cannot read; he can write spontaneously, but cannot copy. He can only read his own writing by his muscle sense: tracing the letters informs him of their shapes, unless the afferent fibres proceeding from the muscular and cutaneous sensory nerves to the visual speech centre are damaged. In this case his muscle sense does not help him, and he is unable to recognize or properly use any object presented to him (**apraxia**). He is able to speak, both spontaneously and by repeating the words of the observer. As a rule, he has hemianopsia. (See Disorders of Vision, p. 545.)

This condition is also an example of subcortical sensory aphasia, and is due to a lesion interrupting the course of the association fibres from the primary cortical centre for vision in the occipital lobe to the visual speech centre.

(e) The patient cannot speak spontaneously, nor can he repeat. He may have a few words at his command, which he uses inappropriately and without meaning. He understands spoken words perfectly, and can understand what he reads, though he is unable to read aloud; in severe cases his understanding is impaired. He may or may not be able to write voluntarily, more commonly not; he may be unable even to write to dictation, but can usually copy, if his right arm is not paralysed.

In this condition the mental action of speech, rather than the

sensory or **perceptive** function, is disordered; it is known as **cortical motor aphasia**, and is due to a lesion involving Broca's convolution. The auditory and visual memories are unimpaired, but the organ (the motor speech centre) which co-ordinates and distributes them to the appropriate cortical centres of movement is inactive; hence the understanding is usually good, while the performance of the act of speech or writing is defective.

(f) A variety of speech defects very similar to the last described (e) is observed in lesions interrupting the impulses proceeding from Broca's convolution to the Rolandic centres for speech movements, the so-called **subcortical motor aphasia**. Voluntary speech is impossible; he cannot write because the hand is probably paralysed, but may write with the left hand, or some other portion of the body (tongue, teeth).

(g) The patient can speak voluntarily, but makes numerous mistakes in his choice of words (**paraphasia**); he may be able to write to dictation and to copy, but similarly in a more or less inaccurate fashion (**paragraphia**); he understands spoken and written words. This condition is due to an interruption in the course of the association fibres connecting Broca's convolution with the auditory and visual speech centres, and with numerous other regions of the brain. It is known as **transcortical motor aphasia**.

**Summary of Aphasia.**—(a) and (b) The patient is mentally blind and deaf so far as speech is concerned; cannot speak intelligibly; cannot read nor write. Cortical sensory aphasia.

(c) and (d) Mentally blind and deaf; he can speak and write spontaneously, but cannot repeat words nor write from dictation. Subcortical sensory aphasia.

(e) Spontaneous speech, and often writing, are impossible; there is paraphasia and he cannot repeat; he understands what he hears and reads. Cortical motor aphasia.

(f) Similar to (e), but may write if hand is not paralysed. Subcortical motor aphasia.

(g) There are paraphasia and paragraphia; he understands spoken and written words. Transcortical motor aphasia.

3. **Anarthria.**—As stated above, lesions involving the mechanism of speech, rather than its psychology, are likely to produce a less complete disturbance of speech. In the higher faculties of speech the fact that these are confined to one

side of the brain renders a complete lesion of that description more probable than one of a more mechanical nature, in which both sides of the brain are represented. The centres in the lower half of the ascending frontal convolution which preside over the movements concerned in speech, or any portion of the tract peripheral to that region, down to and including the actual organs of speech, may be the seat of disease or injury. Functional disturbances, though not strictly referable to this category, may be here included for convenience' sake.

As compared with aphasia, the chief distinction to be noted in anarthria is that there is no tendency to use the wrong words, nor is there any defective understanding necessarily involved. The muscles defective in speech production are also defective in any other function they may possess (swallowing, chewing, moving the tongue, etc.) in cases of anarthria. In this respect they differ from those affected in aphasia, which are only disabled in speech efforts, but are able to carry out any of their other duties.

Paralysis of the tongue, soft palate, lips, etc., occurs in bulbar paralysis, a degenerative process resulting in destruction of the motor nuclei in the pons and medulla. It may occur as an accompaniment of the analogous spinal affection, progressive muscular atrophy, in amyotrophic lateral sclerosis, and in the final stages of locomotor ataxia, or it may appear as an independent malady. Difficulty in talking, swallowing, masticating, are the chief signs. The speech defect is observed as mispronouncing or elision of syllables, rather than the misuse or confusion of words which marks aphasia. The labials and dentals are especially defective, such words as 'artillery' being unrecognizable. Pseudo-bulbar paralysis, the result of a lesion (usually bilateral) in the lower part of the ascending frontal convolution, or in the internal capsule near the genu, is a result of hæmorrhage or other gross lesion of the brain, quite distinct from the progressive and degenerative process causing true bulbar paralysis.

The speech defect in general paralysis of the insane is often very similar to that of bulbar paralysis. A slovenliness and a special difficulty with the dentals and labials, together with tremor of the lips and tongue ('slurring speech'), distinguish this condition from the foregoing. Alcoholic intoxication produces a defective articulation in many respects resembling that of general paralysis. It is possible, however, that both these conditions of

speech may be examples of a partial or imperfect aphasia, seeing that the lesion in general paralysis so commonly involves the cortex, and presumably the speech centres suffer.

The slow, deliberate, scanning speech, to be observed in cases of multiple sclerosis, and less typically in the rarer condition hereditary ataxia (Friedreich's ataxia) is in some measure the result of the spasticity which affects most of the actions of the body in these affections. Not only the organs of speech articulation, but also those of respiration, may be in an exalted state of muscular tone and reflex activity, which may account for the monotonous and staccato style of speech.

**Stammering or stuttering** speech results from excessive and ill-controlled nerve impulses, due to functional disturbance of the speech mechanism. In some respects the defect partakes more of the nature of aphasia than of anarthria. The other functions of the speech organs are unaffected, and the defect is not in the pronunciation of syllables, but in the co-ordination of the muscular acts, which are individually powerful, but imperfectly controlled. A somewhat similar embarrassment of speech may occasionally be seen in chorea and hysteria.

Slight defects, such as lispings or other indistinct articulation, are usually the result of **habit** or **educational deficiencies**.

Imperfect speech may be the result of disease or malformation of the mouth and fauces (see Abnormalities of Voice, p. 555)—*e.g.*, glossitis, tumour or ulcer of the tongue, defect of teeth, tonsillitis, paralysis of the soft palate (diphtheria commonly), cleft palate, hare-lip, etc.

### SPES PHTHISICA.

It is a well-established observation that phthisical patients, even in an advanced stage of the disease, are often optimistic as to their chance of recovery. So frequently has this been noticed that the name 'spes phthisica' has been given to the symptom.

### SPHYGMANOMETER (Sphygmomanometer).

An instrument designed to furnish a means of recording in terms of millimetres of mercury the tension of the arterial pulse. It is described, and the method of using it is stated, in the article on the Arterial Pulse (p. 300).

**SPHYGMOGRAPHIC TRACINGS.**

By means of the sphygmograph it is possible not only to record the condition of the radial pulse, with many advantages over the unaided finger, but also to investigate the condition of the right heart and the venous system. See the articles on the Arterial Pulse (p. 301) and the Venous Pulse (p. 312).

**SPINAL LINE.**

A vertical line drawn on the posterior surface of the thorax in the middle line, connecting the spines of the dorsal vertebræ. It is used in referring to the topography of the chest (see p. 464).

**SPIROCHÆTA PALLIDA.**

A micro-organism, probably a protozoon, shown by Schaudinn to be the cause of syphilis. It is a very delicate, spirally curved motile organism, 10 to 15  $\mu$  long, and is found in the primary, secondary, and tertiary lesions, and in the organs of congenital syphilis.

**SPLASHING SOUNDS.**

The presence of fluid and air in a large cavity can often be perceived by the production of splashing sounds. The diagnostic bearing of the sign is discussed in the article on the Examination of the Stomach (p. 393).

**SPLEEN, Enlargement of.**

The position of the spleen lying below the diaphragm and in relation to the ninth, tenth, and eleventh ribs on the left side, renders it impossible to palpate the organ, unless it be considerably enlarged. Its long axis is parallel with the eleventh rib, and the anterior boundary of the area of dulness which it produces lies immediately behind the mid-axillary line. The enlarged spleen may be recognized by its situation, by its mobility with respiration, by the fact that it usually retains its shape and the characteristic notch on its anterior border, even when much enlarged.

Hypertrophy of the spleen is to be observed in most acute inflammatory affections, and it is of especial diagnostic importance in typhoid fever; in malarial districts the enlarged spleen with fever at once suggests the presence of the malarial parasite in the



blood. Of chronic affections associated with enlarged spleen, one may bear in mind venous congestion due to heart or liver affections (portal obstruction), rickets, cirrhosis of the liver, splenic anæmia, Banti's disease, leukæmia, Hodgkin's disease, waxy disease, pernicious anæmia, and inherited syphilis. It should be remembered that among children the spleen enlarges from comparatively slight causes. (See Anæmia, p. 29.)

### SPUTUM, Examination of.

Collection of material for examination.

#### A. Macroscopical characteristics of sputum :

- I. Quantity.
- II. Odour.
- III. Separation on standing.
- IV. Colour.
- V. Composition : Mucoïd — Serous — Purulent — Mucopurulent — Purulo-mucoïd — Sanguineous — *Corpuscula oryzoïdea*—Dittrich's plugs—Perles of Lænnec — Fibrin moulds — Actinomycosis granules — Lung tissue.

#### B. Microscopical examination of sputum :

- I. Cellular elements :
  1. Epithelial cells. 2. Leucocytes. 3. Red cells.
- II. Non-cellular elements :
  1. Elastic fibres. 2. Crystals : Charcot-Leyden crystals, fatty acid crystals ; calcium oxalate ; triple phosphates ; cholesterin ; leucin ; tyrosin. 3. Curschmann's spirals. 4. Fibrin coagula. 5. Dittrich's plugs. 6. Lung tissue. 7. Echinococcus. 8. Actinomyces.
- III. Micro-organisms :
 

Tubercle bacillus — Pneumococcus — Streptococci — Staphylococci — Micrococcus tetragenus — Diphtheria bacillus — Micrococcus catarrhalis — Meningococcus — Influenza bacillus — Friedländer's bacillus — Bacillus pestis—Anthrax bacillus.

**Collection of Material for Examination.**—In the first place, care should be taken that the patient knows that the sputum to be examined should be coughed up, and not hawked from the naso-pharynx. If an uncontaminated specimen be required, it is necessary that the patient should cleanse the mouth with boiled water before expectorating. The vessel into which the expectoration is received should be clean ; for cultural examination of sputum a sterile vessel is required. In cases where the sputum is merely to be examined by staining of film preparations, the

addition of  $\frac{1}{2}$  per cent. carbolic lotion to the vessel will prevent further bacterial growth.

It is best to obtain a considerable amount of sputum where possible; in some cases that of twenty-four hours should be kept.

**A. Macroscopical Characteristics of Sputum.**—Sputum is usually examined in a shallow glass dish, such as a Petri dish on a black background.

1. **The Quantity of Sputum.**—Not very much inference can be drawn from the quantity of sputum, as it varies exceedingly in different forms of bronchial inflammation. Large quantities of pus in sputum are of importance, and indicate rupture of empyema into a bronchus if of sudden occurrence.

2. **Odour of Sputum.**—As a rule, no appreciable odour arises from fresh sputum, but in some cases it may be very offensive, as in gangrene of the lung and putrid bronchitis. Any sputum may become offensive on standing for a considerable time.

3. **Separation on Standing.**—In some cases—*e.g.*, bronchiectasis, gangrene of the lung, etc.—the sputum settles on standing into three distinct layers—an upper frothy layer, a middle serous layer, and a lower containing plugs of pus.

Any purulent sputum may settle into an upper serous and lower purulent layer.

4. **The Colour of Sputum.**—This varies and depends largely on the amount of cellular elements present; these being non-transparent, give it a yellow or greyish colour. The presence of blood also accounts for variations in colour of the sputa—from pink to that of 'prune-juice' expectoration. In other cases the colour may be due to specific bacteria—*e.g.*, *Bacillus pyocyaneus*, etc.

5. **Composition of Sputum.**—The sputum is variously composed of mucus, serum, pus, and blood. The types usually described are:

(a) *Mucoid Sputum.*—Characterized by presence mainly of mucus greyish in colour, tough in consistence, sometimes stringy.

(b) *Serous Sputum.*—Where there is a preponderance of a watery or serous element.

(c) *Purulent Sputum.*—Where the consistence is that of pus, with a tendency to separate into two layers.

(d) *Muco-Purulent Sputum.*—This is a very common type where the sputum has a more or less homogeneous appearance. In colour it is greyish yellow, in consistence tough and tenacious; it is non-transparent, but if viewed on a dark background the opaque masses of pus can be seen in the more translucent mucus.

(e) *Purulo-Mucoid Sputum*.—In this case there occur large masses of pus, often in coin or disc shape—*i.e.*, **nummular sputum**.

(f) *Sanguineous Sputum*.—Blood occurs in the sputum in various forms:

(1) **Pure Sanguineous Sputum**: Where the blood is alone, either mixed up in a froth with air and pinkish in colour, or in small lumps which have coagulated before expectoration.

2. **Muco-Sanguineous Sputum**—'Rusty Sputum': Where blood is mixed intimately with tough, viscid mucus, commonly found in pneumonia.

3. **Sero-Sanguineous Sputum**—'Prune-Juice' Expectoration: Thin watery sputum, dark brown in colour, often with numerous air bubbles seen through it. This type occurs in cases of pneumonia which are accompanied by œdema of the lung.

4. **Purulo-Sanguineous Sputum**: Here in the large nummular masses of sputum from cavities there is a dirty reddish colour, due to presence of altered blood in the pus.

Other structures may be visible to the naked eye in some sputa, such as—

*Corpuscula Oryzoidea*.—Small cheesy masses which arise from tubercular cavities.

*Dittrich's Plugs*.—Greyish masses of varying size, usually found in gangrenous sputum.

*Perles of Lannec*.—Little pellets of mucus which, on examination, often prove to be coiled-up examples of Curschmann's spirals found in cases of asthma.

*Fibrin Moulds* of small bronchi may be seen as branching structures.

*Actinomycosis Granules*.

In cases of gangrene of the lung pieces of lung tissue may be found as dark, shreddy masses.

**B. Microscopical Examination of the Sputum**.—Microscopically, the sputum is found to contain mucus and varying cellular elements, as well as the special structures already noticed as being apparent macroscopically. To recognize the mucus it is usual to add a little dilute acetic acid to the film, which renders mucus somewhat opaque and shows striations in it.

I. **The Cellular Elements** consist of—

1. **Epithelial Cells**—*viz.*, large squamous cells, which originate from the mouth epithelium; cylindrical cells, occasionally showing a ciliated border, coming from the respiratory mucous membrane;

and, lastly, desquamated alveolar epithelium, the protoplasm of which often shows degeneration in form of myelin or fat droplets. Other forms contain pigment granules, either carbon or altered blood pigment. This latter form of cell occurs in cases of chronic venous congestion of the lungs, and is known as the 'heart-failure cell.'

2. **Leucocytes** (or Pus Corpuscles).—These may be either polymorphonuclear or mononuclear. The former may be neutrophile or eosinophile. In some cases the leucocytes contain pigment or carbon similar to that described in the alveolar cell.

3. **Red Blood Cells** occur often in the sputum where there is no real hæmoptysis.

**II. Non-Cellular Elements.**—Besides these cellular elements other structures may be noted.

1. **Elastic Fibres**, often best found by heating the sputum with 10 per cent. KOH on a slide before examining. The fibres have a wavy outline, are highly refractile, have a double contour, and are often branching. Sometimes the fibres are grouped together, so as to give the outline of a lung alveolus.

2. **Crystals** of various types may be found, the commonest, perhaps, being **Charcot-Leyden crystals**, clear, octahedral crystals; crystals of fatty acids, in long needles; much more rarely, calcium oxalate, triple phosphate, cholesterin—even leucin and tyrosin.

3. **Curschmann's Spirals**, when examined microscopically after the addition of a little dilute acetic acid, show a beautifully spiral arrangement of mucus. These filaments of mucus are often wound round a central mucous filament, and in them are embedded numerous leucocytes, principally eosinophile.

4. **Fibrin Coagula** are seen to be composed of numerous parallel refractile threads, which become clear on the addition of acetic acid. A considerable number of leucocytes may also be seen in them, and sometimes Charcot-Leyden crystals embedded in these moulds of the bronchi.

5. **Dittrich's Plugs** consist mainly of detritus and bacteria.

6. **Lung Tissue** is usually most easily recognized by the refractile elastic fibres and their alveolar arrangement.

7. **Echinococcus of the Lung** may show itself by the presence of hooklets in the sputum or a portion of the laminated membrane.

8. Under the microscope the **Actinomycosis** granules can be recognized as containing the threads and clubs of the ray-fungus.

**III. Micro-organisms in the Sputum.**—The methods used to identify micro-organisms are: (1) Examination of stained films:

(2) culture from a carefully taken specimen; (3) inoculation of animals by carefully prepared specimen.

In this article only the first method will be discussed. For details as to methods (2) and (3) the reader is referred to special textbooks on the subject. Two methods of staining sputum are, in the main, alone necessary—*i.e.*, staining by the Ziehl-Neelsen method for tubercle bacilli and staining in general by Gram's method.

**Detection of Tubercle Bacilli.**—In many cases it will be sufficient to take a small fragment of pus from the suspected sputum, place it on a cover-slip, press on top of this another cover-glass, then draw the two apart; this manœuvre may be repeated three or four times, until a thin film is produced. The film is now stained in exactly the same way as described at p. 530 in the urinary examination.

**Antiformin Method for Demonstration of Tubercle Bacilli.**—The examination is greatly facilitated in many cases by the recently introduced antiformin method, and especially when the sputum is viscid and nummular.

Antiformin is a commercial solution of sodium hypochlorite (6 per cent.) with sodium hydrate (7.4 per cent.). The following are the steps of the method:

- (1) Two to 4 volumes of a 25 per cent. solution of antiformin are added to 1 volume of sputum.
- (2) This mixture is shaken for several minutes, 10 to 15 minutes being usually enough to render the mixture homogeneous.
- (3) Some distilled water is added to reduce the viscosity, and the whole centrifugalized.
- (4) The precipitate is washed with distilled water and again centrifugalized.
- (5) The deposit is then stained for tubercle bacilli.

If the mixture is allowed to sediment for some hours in a conical vessel, the deposit can be used, but is difficult to fix on the slide. Fixation of such a film can be effected by putting the dried preparation in a 2 to 3 per cent. solution of corrosive sublimate, then drying with filter-paper and heating over a flame.

The method can be used to isolate the tubercle bacillus from all ordinary media—sputum, fæces, urine, etc.—leaving the bacillus suitable for culture or inoculation experiments, as the antiformin does not kill the bacillus.

**Detection of Pneumococcus.**—These cocci occur in groups of two; are very often lancet-shaped and show a capsule. They stain by Gram's method (see Urinary Examination, p. 530), and so may be identified.

**Diphtheria Bacilli and Staphylococci** are also easily detected as Gram-positive cocci.

**Micrococcus Tetragenus** is also a Gram-positive coccus, but is arranged in tetrads, usually with a capsule.

**Diphtheria Bacillus** is Gram-positive, but is usually recognized by cultural methods.

**Micrococcus Catarrhalis**.—This is a small Gram-negative coccus found very commonly in the sputum.

**Meningococcus** occurs frequently in the sputum during an epidemic, or rather in the secretion of the naso-pharynx. It can only be recognized definitely from the *Micrococcus catarrhalis* and other Gram-negative cocci by specific agglutination tests, or cultural methods.

**Influenza Bacillus**.—This is a small rod-shaped Gram-negative bacillus, about one-third the length of a tubercle bacillus. It occurs in enormous numbers in the sputum and nasal secretion of influenza vera.

**Friedländer's Bacillus**.—This bacillus occurs as plump rod-like bodies, occurring in pairs, surrounded by a capsule; they are Gram-negative.

**Bacillus Pestis**, a small bacillus which varies in appearance, sometimes almost a coccus form or irregularly oval. It is Gram-negative.

**Anthrax Bacillus**, a very large rod-shaped bacillus occurring in sputum and wool-sorter's disease; it is Gram-positive.

Other bacteria, such as *Bacillus typhosus*, *B. coli*, *B. proteus*, *B. pyocyaneus*, etc., may be found in the sputum.

From the foregoing it will be seen that Gram's method of staining will serve to discriminate in many cases between various bacteria of the sputum—viz.:

Gram-Positive.	Gram-Negative.
Tubercle bacillus. Pneumococcus. Streptococci and staphylococci. Micrococcus tetragenus. Diphtheria bacillus. Anthrax bacillus.	Micrococcus catarrhalis. Meningococcus, Influenza bacillus. Friedländer's bacillus. Bacillus pestis.

J. E. MACILWAINE.

### SQUINT (Strabismus).

Normally the visual axes meet at a spot in the field of vision known as the fixation point. In various diseased conditions of oculo-motor innervation, and of the eye itself, the visual axes fail to meet at this point. They may meet in front of it, when

there is convergent squint. The axes may diverge, the strabismus then being divergent. The subject is considered in more detail in the article on Diminished Movement, at p. 216.

### STAMMERING.

A disorder of speech in which the attempts at articulation are of an explosive and imperfectly controlled character. It is the result of a functional disturbance of the innervation of the organs of speech. (See Speech, Disorders of, p. 383.)

### STAMPING GAIT (Ataxic Gait).

The imperfectly controlled movements of the limbs in walking, characteristic of muscular inco-ordination, and seen typically in locomotor ataxia. The subject is further considered in the article on Gait, at p. 150.

**STELLWAG'S SIGN.** See *Exophthalmos*, p. 136.

### STEPPAGE GAIT (Pseudo-Ataxic Gait).

Paralysis of the muscles in front of the tibiæ and fibulæ is most commonly (especially when bilateral) the result of peripheral neuritis. It causes the foot to drop, and in order to clear the toes in walking the knees have to be raised unusually high, giving a prancing, high-stepping mode of progression. (See Gait, p. 150.)

### STERNAL REGION.

That portion of the thoracic surface corresponding to the sternum. It is divided into three subregions—the upper sternal, above the angle of Ludwig; the mid-sternal, from the second to the fourth costal cartilage; the lower sternal, all below the fourth costal cartilage. The abnormalities to be found in this region are considered in the articles on the Shape, etc., of the Thorax (p. 464), Percussion (p. 450), Auscultation (p. 408), Pain (p. 270).

**STOMACH, Dilated.** See *Stomach, Examination of*, p. 392.

### STOMACH, Examination of.

Inspection: distension; dilatation; gastroptosis; peristalsis—Palpation: tumour; tenderness; succussion sounds and wave—Percussion—Auscultatory percussion—Gastroscopy—Gastrodiaphany—Hour-glass contraction.

Examination of the stomach contents—Method of using the stomach-tube; contra-indications—Motility of the stomach—Test meals—Free hydrochloric acid—Günzburg's reaction—Estimation of the total acidity—Estimation of the quantity of free hydrochloric acid—Hyperchlorhydria—Hypochlorhydria—Supersecretion—Organic acids.

By the various means at our disposal we investigate the shape, size, movements and position of the stomach and the character of its contents.

Some reference has already been made to the physical examination of the stomach in the articles on the abdomen (pp. 1 and 15), and it is here desirable to consider seriatim the diagnostic signs to be observed in examining the organ.

By **Inspection** one learns in many cases more than by any other means. It is often advantageous to distend the stomach with gas, which can generally be effected with only a slight amount of inconvenience and no danger. In cases of gastric ulcer or of imperfectly compensated heart disease, it may be safer to omit the distension. The simplest method is to administer one teaspoonful of sodium bicarbonate dissolved in half a tumblerful of water, followed immediately by the same amount of tartaric acid dissolved in a like quantity of water. A more accurate, but less convenient, method is to pump air into the stomach by means of a Higginson's syringe through a stomach-tube. The distended stomach produces an epigastric swelling, the lower border of which (the greater curvature) is normally two fingers' breadth or more above the level of the umbilicus. The lesser curvature of the normal stomach is behind the liver and close up to the ensiform cartilage.

A lowering of the greater curvature to or below the level of the umbilicus, the lesser curvature and the pylorus not being materially displaced, indicates a **dilated stomach**. This is due to stenosis of the pylorus in most cases, but may also result from atony of the stomach or from a bulky diet, in which large quantities of fluid or vegetables are ingested (*e.g.*, potato-fed persons and those who drink copiously).

If the greater curvature be recognized at a lower level than normal, and at the same time there be a hollowing of the epigastric region, the pyloric end of the stomach being especially low, the condition is that of **gastroptosis**, which is often associated with dislocation downwards of the intestines and kidneys. This is best observed with the patient in the erect posture, as a projec-



tion in the umbilical and hypogastric regions and a recession in the epigastric region.

An important sign to be observed by inspection is peristalsis of the stomach. When these movements are visible, one is almost invariably justified in diagnosing pyloric obstruction. It may be difficult to make sure that the movements are gastric and not intestinal. In the former case they usually, but not invariably, pass from left to right, and are, of course, to be seen over that portion of the abdominal wall which is in contact with the stomach (see p. 13).

**Palpation.**—The presence of a tumour in the stomach may be verified by palpation. If in an elderly subject, it is most probably a malignant growth, and is most commonly to be found in the pyloric region. Occasionally the cicatricial thickening of the stomach in cases of chronic ulcer may be felt. A pyloric tumour is usually freely movable by palpation, and only slightly so with respiration. In these respects it differs from an enlarged gall-bladder or a tumour of the liver. Pain or tenderness on palpation is common to many forms of gastric disturbance, but is especially characteristic of gastric ulcer. Splashing sounds may be elicited by placing the left hand flat over the epigastrium, and pressing suddenly or striking sharply with the right hand over the lower ribs on the left side. These sounds are to be observed where there are gas and fluid in the stomach. If they occur at times when the stomach ought to be empty—*i.e.*, any time over six hours after an ordinary meal—they denote dilatation of the stomach. If they are produced by extremely gentle strokes, they are probably the result of atony of the stomach. Care must be taken to discriminate the splashing sounds produced in the colon or small intestine from those of the stomach. The former may often be abolished by aperients. A somewhat similar palpation or stroke may produce a visible wave travelling across the abdomen. This may be observed when the stomach is dilated and full of fluid.

**Percussion.**—By simple percussion one may in some instances be able to distinguish the position and size of the stomach. This is possible in conditions of gastric distension, whether naturally or artificially produced. It is also practicable to locate the situation of the greater curvature by ordinary percussion in the following manner: The patient is to be examined when the stomach is empty; he is in the sitting posture, and his abdomen

is carefully percussed and the result noted; he is then directed to drink a tumblerful of hot water, and the stomach again percussed; one or two more tumblerfuls of hot water are to be drunk, and the stomach region is gently percussed. A somewhat crescent-shaped area of dulness, increasing in size as the fluid is ingested, is as a rule to be distinguished; in dilated or prolapsed stomachs this dull area reaches as low as, or lower than, the level of the umbilicus.

A combination of percussion with auscultation (**auscultatory percussion**) is sometimes found useful in the examination of the stomach. Indeed, it is in the examination of this organ that this method is especially recommended. It is described and figured in a separate article at p. 52.

Simple auscultation of the stomach is of no practical diagnostic importance except in obstruction of the cardiac orifice of the organ. In this condition the stethoscope placed over the stomach fails to detect the gurgling of entering fluid after the act of swallowing liquids. (See *Œsophagus, Examination of*, p. 265.)

By means of X-ray examination (see p. 571), the shape and size of the stomach may be investigated.

Direct inspection of the interior of the stomach (**gastroscopy**) has been practised, but without much practical advantage. The method is not suitable for clinical examination as a rule.

A more promising procedure is that known as **gastrodiaphany**. A small electric lamp is introduced into the stomach at the point of a stomach-tube. The room being dark, areas of illumination are seen on the anterior wall of the abdomen, corresponding to the cavity of the stomach. The method is beset with difficulties, but may prove useful when more experience of its results is obtained.

The condition known as hour-glass stomach results from the contractions of cicatrices following gastric ulcer. It is rarely a congenital condition. The following symptoms are characteristic of the condition (Moynihan):

1. In washing out the stomach part of the fluid is lost.
2. If the stomach is washed clean, a sudden reappearance of stomach contents may take place.
3. 'Paradoxical dilatation': when the stomach has apparently been emptied, a splashing sound may be elicited by palpation of the pyloric segment.
4. After distending the stomach a change in the position of the distension tumour may be seen in some cases.
5. Gushing, bubbling, or sizzling sound heard on distention with CO<sub>2</sub> at a point distinct from the

pylorus. 6. In some cases, when both parts are dilated, two tumours, with a notch or sulcus between, are apparent to sight or touch.

**Examination of the Stomach Contents.**—By this procedure one may acquire information as to (*a*) the motility of the stomach, (*b*) the quantity and activity of the digestive secretions of the organ, and (*c*) the presence or absence of abnormal constituents among the solids and fluids contained in the stomach. It may be practicable to secure suitable specimens of the stomach contents by vomiting on the part of the patient. More fruitful results may as a rule be obtained by withdrawing the contents of the stomach by means of the stomach-tube.

A soft rubber tube is chosen, closed at one end, near to which are one or two holes in the side of the tube. The other end is provided with a funnel. Eighteen inches from the lateral openings near the closed end of the tube a ring is indelibly marked. The patient is seated (duly protected by means of a large towel or sheet fastened round the neck), the examiner being seated opposite him. The tube has been softened in warm water, and its closed end, smeared with glycerine, is passed to the posterior wall of the pharynx. The patient is now directed to make efforts to swallow, and at the same time to breathe deeply, while the examiner continues to pass the tube backward and downward till the eighteen-inch mark reaches the incisor teeth. Until the patient has become accustomed to the operation there is considerable difficulty with retching and dyspnœa. There is, however, hardly any danger of suffocating if the tube be kept well to the back of the pharynx during the act of introducing it. As soon as about eighteen inches of the tube have passed the teeth, the funnel end of the tube should be lowered so as to lie in a vessel placed at or near the level of the floor, and the patient is directed to make expulsive efforts as of vomiting or defæcation. This will in most cases result in the expulsion of the stomach contents into the vessel. It may be necessary to pour into the upturned funnel end of the tube about a pint of warm water, and while the tube is still full of water it is again lowered, when the siphon action of the apparatus will empty the stomach. Particles of solid food may block the holes at the point of the tube, and these may often be dislodged by forcing in more water, the funnel end of the tube being raised as much as possible. The substances thus removed (if possible not too much diluted) are then to be examined chemically and microscopically.

This procedure is to be undertaken in those cases where the nature of the affection is obscure. It is contra-indicated in the following conditions: In cases of extreme weakness, in fevers, in defective compensation of heart disease, in arterio-sclerosis (owing to the danger of cerebral hæmorrhage as a result of violent vomiting), in aneurism of the aorta, in pregnancy, in cases of hæmorrhage from the stomach or lungs.

Before withdrawing the stomach contents, one must be informed as to the nature of the substances which have been lately ingested, and as to the length of time they have been in the stomach and subjected to the action of the digestive processes. In order that these points may be free from doubt, it is customary to make the examination after the administration of a **test meal** upon a fasting stomach.

(a) **The motility of the stomach** may be tested by observing the changes that have taken place in the food after a given time. If an ordinary mixed meal containing a fair proportion of proteids has been selected as a test meal (which corresponds to Leube's test meal) there will be little or no solid matter found on washing out the stomach after six or seven hours if the motility be normal. Should the movements be defective (atony or dilatation), solid masses may be removed which have been ingested, not only in the immediately preceding meal seven hours previously, but even in meals swallowed twenty-four or forty-eight hours before. A light meal consisting of one or two slices of bread, with a breakfast-cupful of weak tea (Ewald's test meal), disappears from the healthy stomach inside two hours. The appearance of solid lumps of bread in the washings of the stomach after that interval is, therefore, an indication of atony or dilatation of the organ.

A less useful method of investigating the muscular activity of the stomach is that of administering a dose (say 15 grains) of salol in a capsule immediately after a test breakfast. The salol passes unaltered from the stomach to the intestine, where it is split up into phenol and salicylic acid, and is excreted by the kidneys as salicyluric acid. This may be recognized by the appearance of a violet colour on the addition of chloride of iron to the urine, and in normal cases the reaction occurs in about an hour after swallowing the capsule of salol. There are certain defects in this method which detract from its value as a test of gastric motility.

(b) **The quantity and quality of the gastric juice** is most

conveniently estimated by determining the presence and quantity of free hydrochloric acid in the fluid obtained by filtering the stomach contents. A delicate and practical clinical qualitative test is **Günzburg's method**. The reagent is composed of phloroglucin 2 grammes, vanillin 1 gramme, absolute alcohol 30 c.c. A few drops of the reagent, with a similar quantity of filtered gastric contents, are placed in a white porcelain capsule, and evaporated to dryness with gentle heat. Free hydrochloric acid causes the appearance of a rose-red colour. During normal digestion the stomach contains free hydrochloric acid. It is usually absent in cancer of the stomach, and occasionally in atonic dyspepsia (see below, p. 398). A less delicate test is **Boas's resorcin reagent** (resorcin 5 grammes, cane-sugar 3 grammes, dilute spirit 100 c.c.), used in a similar way to Günzburg's; if free HCl is present, a reddish purple colour results.

The **total acidity** of the stomach contents is next to be estimated, bearing in mind that other factors than free hydrochloric acid contribute to the amount of acidity present. Salts of the mineral acids (chiefly acid phosphates), organic acids (see below) and their compounds, all combine in certain cases to increase the acidity of the fluid. The method adopted to estimate the acidity consists in determining the amount of caustic soda which is required to completely neutralize the acid contained in a given quantity of stomach contents. In order to ascertain the point at which neutralization takes place, an **indicator** is required. In estimating the acidity from all causes, a convenient indicator is a 1 per cent. alcoholic solution of phenolphthalein, which is colourless in acid solutions and pink in alkaline. A burette is filled with decinormal solution of sodium hydrate (4 grammes NaHO to 1 litre water), and placed in a stand; 10 c.c. of unfiltered stomach contents, diluted with about 100 c.c. of water, are well shaken up with a few drops of phenolphthalein solution in a flask, which is placed on a sheet of white paper under the burette. From the latter the decinormal soda solution is run into the flask drop by drop, and the contents repeatedly shaken. As soon as the diluted stomach contents begin to turn alkaline a pink colour is seen to permeate the fluid. The amount of soda solution expended is then read off the burette. A convenient measure of the acidity is the quantity of decinormal soda solution required to neutralize 100 c.c. of stomach contents. If the amount used in neutralizing 10 c.c. in the above experiment be, say, 7.5 c.c., 100 c.c. of

stomach contents will require 75 c.c. of decinormal solution, and the acidity of the specimen is said to be 75. During digestion the acidity is normally between 40 and 60.

The quantity of free hydrochloric acid present is similarly estimated by observing the quantity of decinormal solution used in neutralizing a given quantity (usually 10 c.c.) of gastric contents. In this case the indicator is a 0.5 per cent. alcoholic solution of dimethylamidoazobenzol, a few drops of which added to the fluid in the flask causes it to turn red, owing to the presence of free hydrochloric acid. The soda solution is gradually run in till the flask contents become neutral, when the red colour disappears and is replaced by a greenish yellow. The quantity of soda solution expended is noted. It is known that the 4 grammes of caustic soda contained in 1 litre of decinormal soda solution exactly neutralize 3.65 grammes of free hydrochloric acid; therefore 1 c.c. of decinormal soda solution neutralizes 0.00365 gramme of free hydrochloric acid. Supposing that the quantity of decinormal soda solution expended is 6 c.c., the 10 c.c. of stomach contents which are being examined must contain  $0.00365 \times 6 = 0.0219$  gramme of HCl. The percentage of HCl (the quantity contained in 100 c.c. of stomach contents) is therefore 0.219. About 0.2 per cent. is the average amount during normal digestion.

A normal quantity of hydrochloric acid may be present even in cases where the digestion is disturbed, as in some instances of gastritis or gastric ulcer. Increase in the amount of hydrochloric acid (**hyperchlorhydria**) is frequently found in gastric ulcer, in neurotic dyspepsia, and in acute or chronic gastritis. Absence or diminution of the free acid (**hypochlorhydria**) is the rule in cancer of the stomach, in pernicious anæmia (often confused with gastric cancer), in dilatation of the stomach from chronic catarrh or from atony, and sometimes from cicatricial contraction of the pylorus. It may also occur in nervous dyspepsia, in anæmia, and in fevers. An excess (**supersecretion**) of the gastric fluids generally is found in catarrh of the stomach, in the gastric crises of locomotor ataxia, in gastric neuroses, and sometimes in gastric ulcer.

(c) **Abnormal constituents** may be found in the contents of the stomach. Of these the most important are organic acids, bile, blood, and fæcal matter.

Organic acids may be readily demonstrated by **Uffelmann's test**. To a test-tubeful of a 1 per cent. solution of carbolic acid add one drop of liquor ferri perchloridi. Dilute the solution till it becomes

an amethyst-blue colour. On the addition of a fluid containing organic acids (lactic acid commonly) the blue colour is changed to yellow. Free lactic acid is often found in conditions where hydrochloric acid is deficient or absent, and especially in cancer of the stomach. It is present (often in company with acetic or butyric acid) when fermentative changes are proceeding in the stomach.

The other abnormal constituents of the stomach contents and their clinical significance are referred to in the article on Vomiting, at p. 557.

**STRIDOR** (*L. stridere*, to make a creaking sound).

A harsh, noisy type of respiration, produced by an obstruction to the entrance of the air into the upper air passages, and heard chiefly in the inspiratory portion of the act. The conditions giving rise to the symptom and the accompanying features are discussed in the article on Dyspnoea (p. 131).

**STUPOR.** See **Unconsciousness**, p. 449.

### **STUTTERING.**

A functional disturbance of speech, in which the motor nerve impulses are of an explosive and ill-governed character. (See Disorders of Speech, p. 381.)

**SUBCLAVIAN SYSTOLIC MURMUR.** See **Arteries, Examination of**, p. 43.

### **SUBCREPITANT RÂLES.**

Adventitious crackling sounds found in bronchitis, croupous and catarrhal pneumonia, œdema or hæmorrhage of the lung, phthisis, or hypostatic congestion. They are coarser in quality than the fine crepitant râles, and probably signify the presence of a more copious or fluid condition of the secretion than that which contributes to the occurrence of the latter (see p. 419).

**SUBNORMAL TEMPERATURE.** See **Temperature**, p. 405.

### **SUBSULTUS TENDINUM.**

In conditions of serious prostration the patient's muscles may be stimulated to irregular contractions, causing a jerking or twitching of the limb or region involved. The symptom is often an accompaniment of delirium.

### SUCCUSSION-SOUNDS.

Splashing may be heard, and at times even felt, on vigorously shaking a patient whose pleural cavity contains both air and fluid. It may be audible at a distance or to the patient himself, but is best appreciated by the stethoscope. A similar sound may be elicited in the stomach or colon, even in a healthy condition of those organs, but more commonly in dilatation.

### SUPPRESSION OF URINE (Anuria).

A marked diminution in the quantity of urine excreted or its total cessation are conditions of extreme danger.

The causes of the diminution or stoppage of the urinary excretion are :

(1) Acute Bright's disease or acute congestion, active or passive ; acute exacerbations of chronic nephritis.

(2) Injury to the renal tissue from hydronephrosis, pyonephrosis, abscess.

(3) Copious loss of fluids, as in diarrhœa, dysentery, severe hæmorrhage, vomiting, cholera.

(4) Reflex suppression, following injuries or operations, especially on the urinary organs.

(5) Hysteria sometimes exhibits genuine suppression of urine. One must be cautious in accepting the diagnosis of hysterical anuria, lest the condition be really retention, or even deception as to the amount of urine passed.

(6) Obstruction to the outflow from the kidneys. This may be the result of impaction of both ureters with stones ; impaction of one ureter, the other kidney being either seriously diseased or absent ; twisted ureters in floating kidney (very unlikely to occur simultaneously in both kidneys) ; blocking of ureters by cancer or cicatricial contractions.

In addition to the absence, partial or complete, of urine in the bladder, the patient soon shows signs of uræmia, and unless the excretion is re-established death ensues in twelve to fourteen days. In those cases of anuria resulting from obstruction to the outflow (6), the symptoms are for some days unobtrusive, and the cases are termed by Roberts **latent suppression**. The small quantity of urine passed may be clear and light in colour, and of a low specific gravity, containing little or no urea or albumin, in which respects it differs from the urine passed in the other forms of suppression, for as a rule a very small quantity of bloody urine of high density is passed in the latter cases.



**SUPRACLAVICULAR REGIONS.**

On each side of the chest the small portion of the pulmonary region lying above the clavicle is known by the above term. Abnormalities to be found in that region are referred to in the articles on Thoracic Shape, etc. (p. 464); Movements, Percussion (p. 450); Auscultation (p. 408); and in that on Pain (p. 270).

**SWEATING, Abnormalities of.**

The amount of sweat may be increased, diminished, or abolished in various morbid conditions; it may be partial in its distribution, or its character may be altered.

**Excessive sweating (hyperidrosis**, or when exceptionally copious it is known as **colliquative sweating**). This occurs in certain acute fevers, especially in rheumatic fever, septicæmia, abscess, and tuberculosis. In the last-named affection the sweating occurs during sleep, and is especially likely to come on in the small hours of the morning. Debility and collapse cause sweating, and the use of diaphoretic drugs has to be borne in mind—*e.g.*, nitre, acetate of ammonium, pilocarpine, ipecacuanha. It may also be seen in exophthalmic goitre. A rare disease occurring in epidemics known as military fever or sweating sickness is characterized by the appearance of sudamina and profuse perspiration.

**Absence of sweating (anidrosis)** is seen in Bright's disease, diabetes (sometimes there is profuse sweating in this disease), profuse diarrhœa, cholera, dysentery, and, indeed, most conditions in which there is a copious loss of fluid by other channels than the skin; in myxœdema a harsh, dry skin is found.

**Localized sweating** is to be observed. In cervical caries and in thoracic aneurism damage to the sympathetic may result in unilateral sweating (**hemidrosis**); a similar effect is sometimes produced by neuralgia, by migraine, or by tumours and inflammations in the neck. Sweating of the head is characteristic of rickets.

The **character** of the sweat may alter: it may contain **urea** in cases of Bright's disease, **bile** in severe cases of jaundice, blood (**hæmatidrosis**) or other colouring matter (**chromidrosis**) on rare occasions. It may have a peculiarly offensive odour (**bromidrosis** or **osmidrosis**), due to bacterial activity.

**SYNCOPE.** See Unconsciousness, p. 502.

**TABETIC FOOT.**

An extreme degree of destruction of the tarsal and metatarsal bones occurring as a result of disturbed trophic control. The deformity, produced partly by absorption of the bones and partly by dislocation of the metatarsus under or over the tarsus, suggests the appearance of the toes being articulated directly on to the tarsus. The condition is quite different and distinct from the disorganized foot the ultimate result of a perforating ulcer and septic disease of the bones. The clinical significance of the condition is considered in the article on Trophic Disturbances (p. 495).

**TACHE CÉRÉBRALE (Tache Spinale, Dermographism, Trousseau's Spots).**

On lightly irritating the skin, as by scoring it gently with the finger-nail, the portion of skin touched becomes in certain conditions bright red, and may be raised in a wheal. (See Reflexes, p. 347.)

**TACHYCARDIA (Gr. *ταχύς*, swift; *καρδία*, the heart).**

Abnormal frequency in the pulse-rate from any cause is often spoken of as tachycardia, though some authors restrict the term to a paroxysmal affection, in which periodical attacks of frequent pulse occur. The nature of the symptom and the conditions in which it occurs are considered in the article on the Arterial Pulse (p. 305).

**TALIPES.** See **Contracture**, p. 105.

**TASTE, Abnormalities of.**

Deficiency in acuteness of the sense of taste, or its complete abolition (**ageusia**), is observed commonly as a result of affections of the tongue or mouth, and of conditions which impair the sense of smell. It is less frequently due to disease of the trigeminal or glosso-pharyngeal nerves, or their central connections. Facial paralysis is often accompanied by partial and one-sided ageusia, owing to the implication of the chorda tympani.

Perversions of taste sense (**parageusia**) are sometimes the form in which the epileptic aura occurs. They are not infrequent in

cases of mental disease and in hysteria. More commonly they result from local irritation in the mouth, or from digestive disturbance and the administration of drugs (*e.g.*, iodine or iodide of potassium).

### **TEETH, Hutchinson's (Peg-top Teeth, Screwdriver Teeth).**

Of the later effects of congenital syphilis a deformity of the permanent teeth, especially of the upper central incisors, is among the most characteristic. They are usually shorter than normal; they taper from the gum to the edge, in the centre of which is a single notch. They are often placed well apart from each other, and may be crookedly or irregularly seated in the jaw. Care must be taken not to mistake the furrowed and irregular teeth due to malnutrition or illness, occurring during the period of growth of these teeth, for those above described. If the upper central incisors, and possibly other incisors, show the features above enumerated, and if the patient also suffer from keratitis and middle-ear disease, congenital syphilis may be confidently diagnosed.

### **TEMPERATURE OF THE BODY.**

Slight variations from the normal temperature of  $98.4^{\circ}$  F. are quite consistent with health. From  $98^{\circ}$  to  $99^{\circ}$  F. may be taken as normal. The morning temperature is lower than that of the afternoon in most healthy subjects by a degree or less.

The methods of taking the temperature are, briefly: in the rectum (the most accurate, but not the most convenient, method); under the tongue (the most generally suitable method); in the groin (the best method for young children); in the axilla (the most usual and the most inaccurate method). On an average the axilla temperature is one degree lower than that of the mouth or rectum.

Much difference is to be noted in the readiness with which the temperature rises in individuals. Some show a considerable elevation from causes which in others would produce but little pyrexia. In children the temperature rises with comparatively slight provocation.

A temperature raised above normal (pyrexia) is the chief and most constant feature in the condition spoken of as **fever**, so much so that this term is commonly used as a synonym for pyrexia. Correctly speaking, the term 'fever' includes not only pyrexia, but

also malaise, weakness, disturbance of the digestion, with loss of appetite, thirst, diminution of the quantity of urine, emaciation, rapid breathing, and often temporary mental disturbance.

With few exceptions, acute inflammatory affections are accompanied by elevation of temperature. The range of the temperature, its duration, its mode of onset, and the manner of defervescence, are in many instances characteristic of the disease of which it is an important sign.

(a) The **invasion** or **onset** of the fever may be abrupt or gradual. In the former it is often accompanied by a rigor, which in children is frequently represented by a convulsion. Sudden onset occurs in croupous pneumonia, scarlet fever, erysipelas, disorders of gastro-intestinal origin in children, tonsillitis, and malaria. Influenza is often of this type. A gradual onset is commoner, and is seen in most of the acute feverish conditions—*e.g.*, typhoid fever, measles, bronchitis, broncho-pneumonia, rheumatism, etc.

(b) The **height of the fever**, or **fastigium**, exhibits much variety. It may be (1) **continued**—*i.e.*, there is no material fall in the temperature till the end of the fever approaches; typhoid fever, pneumonia, acute tuberculosis, are examples. (2) **Remittent** temperature falls a couple of degrees or so each day, but does not touch normal. This type is found in the third week of typhoid, in phthisis, in suppuration, in pyelitis, in septic absorption. (3) **Intermittent** fever shows periods of freedom from pyrexia. The temperature falls at some time of the day to or below normal. Malaria is the best instance of this type, which is also seen in hectic, suppuration, often in sepsis, and, in the rarer conditions, ulcerative endocarditis and Hodgkin's disease. If the intermission lasts more than a day, the temperature is then spoken of as (4) **relapsing**. This variety may be seen in relapsing fever, typhoid fever, and influenza. Malaria is often of this type.

(c) The **termination** of the pyrexia may be by—(1) **Crisis**—*i.e.*, an abrupt fall down to, or nearly to, normal inside twenty-four to thirty-six hours. This occurs in lobar pneumonia, measles, chicken-pox, malaria, tonsillitis, relapsing fever. A sudden improvement in the patient's condition (pulse-rate and respiration), sweating, an increase in the quantity of urine secreted, and decrease in the feelings of discomfort associated with the fever, usually accompany the critical fall of temperature. A **pseudo-crisis** is sometimes seen in pneumonia: the temperature falls suddenly to a considerable extent before the time for the real

crisis has arrived; it then mounts again to the height it had occupied before the false crisis. A sudden fall of the temperature to subnormal depth may indicate hæmorrhage or collapse (see below). (2) **Lysis** is the gradual defervescence which characterizes the majority of fevers. Rheumatism, typhoid fever, lobular pneumonia, scarlet fever, pleurisy, and septic infections are among the commonest examples of this mode of termination. A termination by lysis in affections which usually end by crisis probably indicates the supervention of a complication.

**Hyperpyrexia** is an elevation of temperature exceeding  $106^{\circ}$  F., and may occur in the course of some of the fevers, notably in acute rheumatism, typhoid, malaria, and sunstroke. It is more commonly seen as the termination in fatal cases of cerebral hæmorrhage, injuries of the brain and cervical portion of the spinal cord, typhoid fever, yellow fever, scarlet fever, tetanus, etc.

**Subnormal Temperature.**—The body heat remains below the normal level ( $98^{\circ}$  F.) in certain chronic affections—*e.g.*, valvular disease of the heart, and chronic lung diseases which give rise to cyanosis; chronic wasting disease associated with imperfect nutrition—*e.g.*, cancer of the œsophagus or stomach; and in some mental affections, especially melancholia. A sudden fall from fever height to below normal is one of the signs of collapse and of internal hæmorrhage. One must therefore be careful to distinguish this from a healthy crisis. In the latter condition the fall of temperature is accompanied by signs of improvement in the general condition. The pulse improves, the patient feels better, and may fall into a natural sleep. When the temperature drops in consequence of collapse, the pulse is more frequent, becomes small, of low tension, and often irregular; at the same time the respirations usually increase in frequency, and may be sighing, irregular, or show other signs of dyspnoea. It is in croupous pneumonia that such an unfortunate occurrence might most readily be mistaken for a beneficial fall of temperature. Collapse from heart failure or from the intensity of the disease and extent of lung involved may be the cause of such a fall in the temperature. Other examples of this unfavourable fall of temperature are bleeding from typhoid, gastric, or duodenal ulcers, from an extra-uterine pregnancy, or from the lungs. It also occurs in perforation of the viscera, as in typhoid fever, gastric ulcer, or duodenal ulcer. After a hæmorrhage the temperature often remains subnormal for some time, but in case of perforation of

the abdominal organs it soon rises again to fever heat, owing to the supervention of peritonitis.

When the fever has passed off in any acute affection, whether it have terminated by crisis or by lysis, the temperature often remains subnormal for a time, indicating the lowered metabolism which may be expected to follow the excessive activity of the pyrexial period.

### TENESMUS (Gr. *τείνω*, to strain).

The term is commonly applied to straining efforts to empty the bowel, as may occur in irritative conditions of the intestine. Its occurrence indicates in most cases that the large intestine is the seat of the irritation, and it is typically found in dysentery. Less frequently the term is applied to similar straining efforts in urination (vesical tenesmus).

**TEST MEAL.** See *Stomach, Examination of*, p. 396.

### THIRST.

The sensation of thirst is generally referred to the throat, and is usually accompanied by a dryness of the soft palate. By moistening the palate thirst may to some extent be relieved, but the actual cause of the sensation is diminution of fluid in the lymph spaces generally. This results from the loss of fluid from the blood, or the insufficient supply of fluid. The specific gravity of the blood tends thus to be raised, and in order to maintain it at its normal density, fluid is withdrawn from the lymph spaces. The soft palate naturally shares in the dryness of the tissues, and is thus an index of the condition of the fluids in the body.

Thirst is, then, a marked symptom in conditions characterized by loss of fluid from the body. **A rise in temperature** is almost invariably accompanied by thirst (typhoid fever is an occasional exception). Here an unusual amount of water passes out from the lungs in consequence of the rapidity of respiration, and much fluid may be lost by sweating. **Profuse diarrhœa**, especially the copious fluid motions of cholera, give rise to thirst. **Sudden severe hæmorrhages** have a similar effect. **Persistent and copious vomiting** and irritated states of the stomach are often the cause of severe thirst. **Profuse sweating** from any cause—*e.g.*, muscular exercise, heat, fevers—is a familiar excitant of thirst. Perhaps the most important cause of thirst is the **passage of large quantities**

of urine (polyuria). This is a feature in diabetes mellitus and insipidus, and here thirst (combined, as a rule, with an increased appetite for food and loss of weight) is often the first symptom to draw attention to the condition. Polyuria is also observed in chronic atrophic nephritis (small white kidney), in chronic renal sclerosis (small red kidney), and in amyloid disease of the kidney, in which conditions thirst may be somewhat increased, but not to the extent that occurs in diabetes.

Thirst produced by **insufficient supply of fluid** is of less interest to the diagnostician, as it rarely occurs in medical affections.

## THORAX, Auscultation-Sounds of.

### Part I.—Sounds resulting from Respiration.

*Breath-Sounds.*—Intensified: puerile; broncho-vesicular; transitional; bronchial. Weakened: from impaired production; from impaired conduction—Cog-wheel breathing—Tubular breathing—Cavernous breathing—Amphoric breathing—Metamorphosed breath-sounds—Laennec's veiled puff—Stridor—Summary of breath-sounds.

*Voice-Sounds.*—Vocal resonance; bronchophony; diminished vocal resonance; pectoriloquy; whispering echo; ægophony; Bacelli's sign—Summary of voice-sounds.

*Adventitious Sounds.*—Rhonchi—Râles: crepitant; subcrepitant, mucous; gurgling; consonating; metallic; cavernous—Friction-sound—Bell-sound—Metallic tinkling—Succussion-sound—Summary of respiratory adventitious sounds.

### Part II. Sounds produced by the Circulatory Organs.

*The Normal Heart-Sounds.*—Accentuation of both sounds—Accentuation of the second sound; of the first sound—Weakening of the sounds; of the first sound; of the second sound—Altered rhythm of the sounds—Pendulum rhythm—Embryocardia—Reduplication of the heart-sounds—Gallop rhythm—Altered quality of the sounds: musical; metallic; rough—Summary of modifications of the normal heart-sounds.

*Adventitious Sounds of Circulatory Origin.*—*Murmurs*, their causes and significance; their position; their time; their conduction; their character—Vascular murmurs—Venous hum—*Exocardial sounds*: pericardial friction; pericardial splashing; pleuro-pericardial friction; cardio-pulmonary and other sounds—Summary of adventitious sounds of circulatory origin.

The use of the stethoscope is almost entirely restricted to the examination of the thoracic contents. The general principles of auscultation are considered at p. 50. By this means the following subjects may be investigated:

*Part I. Sounds resulting from Respiration.*

1. The sounds produced by the act of respiration under normal circumstances, and their modification in various diseased conditions.

2. The voice-sounds as heard over the healthy chest and as modified by disease.

3. The various new or adventitious sounds produced in connection with disease of the lungs—viz., râles, rhonchi, friction-sounds, etc.

*Part II. Sounds resulting from the Heart's Action.*

1. Modifications of the normal heart-sounds.

2. Adventitious sounds of circulatory origin, including (A) endocardial and vascular murmurs, and (B) exocardial sounds—viz.: (a) pericardial friction-sound; (b) pericardial splashing-sound; (c) pleuro-pericardial friction-sound; and (d) cardio-pulmonary sounds.

**Part I. Sounds resulting from Respiration.**

1. **Breath-Sounds.**—The act of breathing causes sounds which vary in quality and intensity according to the region under examination. Near the trachea and larger bronchi it is a harsh, to-and-fro sound, the tracheal or bronchial breathing. This is observed over the upper part of the chest in front, especially near the sternum, and is better marked on the right than on the left side, owing to the larger diameter and more horizontal position of the right bronchus, as compared with the left. Posteriorly bronchial breathing is heard in the neighbourhood of the upper three or four dorsal vertebræ, near the position of the root of the lung. This sound is synchronous with the inspiratory and expiratory acts, and is simply the sound produced in the glottis, or sometimes that produced in the mouth, conducted peripherally through the air channels, and thence to the surface. (See Bronchial Breathing, p. 95.) The further from the larynx one places the stethoscope the less distinct is the sound, as a larger quantity of imperfectly conducting lung parenchyma is interposed, until, on listening over the sides or bases of the lungs, one hears the characteristic **vesicular breath-sounds**.

This sound is easily recognized as a sighing, whispering rustle, coinciding almost entirely with the act of inspiration. As a rule,



the inspiratory sound is three or four times longer than the expiratory, though the act of expiration occupies a rather longer time than that of inspiration. Moreover, the short expiratory portion is softer and somewhat lower pitched than the inspiratory. In some instances a faint, soft sound persists during the whole duration of expiration.

The origin of this sound is a point upon which observers are not agreed. The two chief views that are held on the method of its production are—(a) that it is the glottidean breath-sound modified and obscured by the imperfectly conducting spongy lung tissue, and (b) that the sound is produced in the lung at the spot under examination by the passage of air to and from the air cells. The majority of observers favour the former theory, as it agrees better with clinical experience. There is no doubt that the normal lung tissue is a bad conductor of sound, for the increased intensity of the voice or heart sounds is striking when the normal lung tissue is replaced by the homogeneous, highly-conducting inflammatory consolidation. Moreover, it is stated that a piece of animal lung laid over the trachea transforms the harsh tracheal sound into one resembling vesicular breathing. It may also be remembered that the amount of air current reaching the air cells must be very trifling, a great part of the interchange of gases in the alveoli being effected by diffusion, so that the process is probably almost noiseless. Powerful arguments have been adduced against the glottidean origin of the breath-sounds, and in favour of its production in the alveoli and smallest bronchioles, and there seems good reason to believe that both modes of origin have a share in the production of the sound.

**Increased Breath-Sounds.**—An increase in the intensity of the breath-sounds may occur as a louder vesicular breathing than normal, or **bronchial breathing** may be heard at places where vesicular breathing should normally be observed. It may be due to an increased production of the sound, or to an improved conduction of the vibrations. From the former cause unduly loud breath-sounds are heard over a lung acting excessively. This occurs when one lung sustains most of the respiration, owing to disease of the other lung, the so-called **puerile breathing**. It is also heard in forced breathing, in dyspnoea, in affections of the larynx which cause narrowing of the rima glottidis, in obstruction of the trachea or larger bronchi from pressure, foreign bodies, etc., and in cases where some obstruction in the upper air passages

produces noisy mouth or nose breathing. Increase of the breath-sounds from improved conduction is observed when a portion of the spongy lung tissue is replaced by a more homogeneous or consolidated condition, as is seen in pneumonic or tubercular infiltrations of the organ, in tumours, in compression, and to a less degree in relaxation of the lung, provided the larger bronchi remain patent. It also occurs when the chest-walls are excessively thin. The increased breath-sound generally takes the form of bronchial breathing rather than louder vesicular breathing.

It is possible that the increase of breath-sounds in consolidation of the lung may have other causes beside an improved conduction. There may be a local production of sound in the affected region of the lung. In cases of compact consolidation of the lung the breath-sounds are usually not only loud and bronchial, but have an added blowing quality, with a metallic, whistling character which is easily recognized, and is known as **tubular breathing**. The intensity of this sound to a great extent depends upon the superior conducting qualities of the consolidated lung, which transmit the glottidean breath-sounds very effectively. Nevertheless, it is probable that the blowing, 'tubular' character (as if one were blowing through, or across the open end of, a tube) is acquired in the lung tissue under examination (see below, p. 413, and Fig. 63).

**Broncho-Vesicular Breathing.**—Combined vesicular and bronchial breathing is sometimes to be heard, in which inspiration is either vesicular or a combination of both forms, while the expiratory part is usually bronchial. This sound is simply a form of increased intensity of breath-sound, and may generally be regarded as the first stage in the production of bronchial breathing, as it may be observed in all stages from vesicular to pronounced bronchial breathing. Hence it is known as **transitional, indeterminate, or mixed breathing**. In the least definite forms of mixed breathing all that can be found abnormal in the sound is a prolongation of the expiratory portion of the vesicular breathing, without the distinct blowing character of the expiratory element observed in well-marked broncho-vesicular breathing.

Prolonged expiration of the vesicular or bronchial type is, then, of diagnostic importance as an evidence of increased conduction or production of breath-sounds. If heard at the apex of the left lung it is suggestive of tuberculous infiltration. At the right apex it is of less serious import, as owing to the anatomical relations

of the bronchi mentioned above, the breath-sounds are louder to the right of the sternum than to the left.

Bronchial breathing, if heard toward the base of the lung, is often a sign of pneumonia, less commonly of tuberculous disease. In this region of the chest, and especially at the sides and back, a soft, distant, purely bronchial sound may indicate pleural effusion. This is ascribed to relaxation of the lung, rendered possible by the partial occupation of the thorax by fluid. The lung in this relaxed condition is a better conductor of sound than in the normal state. If the breathing be tubular, a complete consolidation with patent bronchi is probable. If the breath-sounds be not only bronchial, but also **cavernous** (see below, p. 412), a cavity may be diagnosed if the evidence obtained by percussion corroborates.

The disappearance of bronchial breathing before resolution of a consolidation may indicate the plugging of a bronchus, which may be relieved by coughing. A similar disappearance of bronchial and cavernous breathing may result from the filling up of a cavity with secretion, the removal of which by coughing may restore the sounds to their former character.

**Decreased Breath-Sounds.**—Diminution in intensity of the breath-sounds is the result of either deficient production or imperfect conduction of the audible vibrations. Lessened expansion of the chest diminishes the breath-sounds; a flattened or badly-developed chest, debility, pneumothorax, and emphysema reduce the respiratory movements, though in the last-named affection bronchial catarrh may cause loud breath-sounds. Obstruction of the air passages, if it should not give rise to noisy respiration, will diminish the activity of respiration and so weaken the sound; but here this effect may be due also to imperfect conduction, as transmission through the blocked air channels is impeded. The weakened sounds may be confined to one side or one region of the chest if the obstruction is situated in a bronchus. Pleurisy or peritonitis will restrict the respiratory movements, owing to the pain, and the same result follows intercostal rheumatism, neuralgia or fractured ribs.

Diminished breath-sounds from impaired conduction of the vibrations is observed when the air passages are completely blocked, as in **massive pneumonia**, and occasionally the same result occurs in tubercular infiltration of the lung if the bronchus should be occluded. A commoner condition of this description is the

interposition of some imperfectly conducting material between the bronchi and the surface of the thorax, such as pleural effusion, serous or purulent, thickening of the pleura, tumour, and thick chest-walls.

In addition to deficient or excessive intensity of the breath-sounds, one finds various abnormalities in the quality or character of the sounds.

**Cog-wheel breathing** is a jerky, intermittent, vesicular breath-sound, heard as a rule only in inspiration. It may be an accompaniment of increased or of diminished breath-sounds, and may be heard generally over the chest or localized in one region. In the former case it is due to irregular muscular action, and may be caused by fatigue, debility, or paresis; if localized, the intermittent character of the breathing may be the result of obstruction in the bronchioles to the passage of air, and is found in bronchial catarrh, in early and in late phthisis.

**Tubular Breathing.**—A high-pitched, clear quality of breath-sound is commonly heard during inspiration and expiration over the consolidation of lobar pneumonia; in this case the altered sound is due to a combination of causes, namely, improved conduction of the laryngeal breath-sounds through the solid and more homogeneous lung. There is also a local production of sound, for the breath-sound heard over the affected lung may be even louder than that heard over the larynx. Fig. 64 gives an explanation of the local production of tubular breathing, which many observers adopt. In the unobstructed bronchus *a*, surrounded by consolidated lung, the air is at rest; while in *b*, which traverses active lung, a current flows freely across the open mouth of *a*, thus perhaps giving rise to an imperfect whistling sound, remotely resembling that produced by blowing across the opening of a key. Another local origin for the increased breath-sounds is an added resonance in some cases, given to the glottidean sound by the elasticity of the consolidated lung. This cannot be an important factor in the sound, as consolidated lung, while a better conductor of sound than normal lung tissue, is an inferior resounding medium, as is shown by its behaviour under percussion (see p. 279).

**Cavernous and Amphoric Breathing.**—A low-pitched bronchial breathing, to which is added a reverberating quality, may be observed when listening over a large air cavity. A curious metallic tone is given to the sound, similar to that acquired by sounds

emitted in a hollow enclosed space, and hence it is known as **cavernous breathing**. The metallic quality is due to overtones developed in the cavity by reflections from the walls, which must be fairly smooth, and the cavity must, it is said, measure at least 2 inches in its longest diameter. A somewhat similar sound, and one produced most probably in a very similar manner, is the reverberating breath-sound termed **amphoric breathing**, because of its resemblance to the sound produced by blowing across the mouth of a narrow-mouthed vase or wide-mouthed bottle. Cavernous and amphoric breathing may be distinguished from each other by their tone, but for diagnostic purposes it is un-

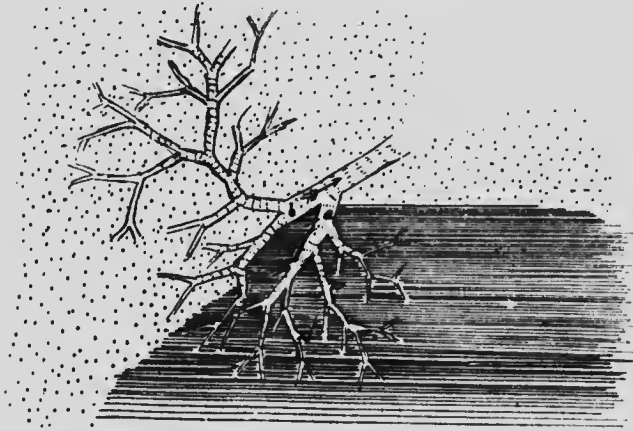


FIG. 63.—TUBULAR BREATHING.

The shaded area represents consolidated lung. In the bronchus, *a*, and its branches, surrounded as they are by immobile lung tissue, the air is at rest. From *b*, a bronchus surrounded by active lung, a current of air passes across the mouth of *a*, thereby causing a local production of sound.

necessary to do so, as they both are strong evidence in favour of a cavity in the lung, of severe bronchiectasis, or of pneumothorax in free communication with a bronchus.

**Metamorphosed Breath-Sounds.**—During the act of respiration a change in the character of the sound may be observed; inspiration may begin as a harsh or bronchial murmur, becoming softer as it proceeds, and ending in an amphoric sound. A variation in the pitch of a bronchial sound may be noted. Both the conditions just mentioned occur as a result of pulmonary cavities; after inspiration has proceeded a certain length the cavity may become sufficiently distended, either to produce an amphoric quality in

the sound or to alter its pitch. Movements in the bronchial secretion may cause other varieties in the breath-sound, thus Lænnec's **veiled puff** ('souffle voilé') is a sudden change in the intensity or quality of the sound due to the temporary removal of a plug or curtain of mucus from a partially blocked bronchus.

**Stridor.**—A noisy or discordantly musical breath-sound, due to an obstruction of some description in the bronchi, trachea, or larynx, is termed **stridor**, and may often be perceived without the aid of the stethoscope. It may be due to laryngitis, laryngismus, pertussis, diphtheria, foreign body, asthma, mediastinal tumour, thoracic aneurism, etc. The stridor may be expiratory, or more commonly inspiratory, and is accompanied by exaggeration of the respiratory movements, and by retraction of the intercostal spaces. It is further referred to in the article on *Dyspnœa* (p. 125).

**Summary of Breath-Sounds.**—Bronchial breathing heard in regions where normally vesicular should be found indicates either increased production of the glottidean sounds (as in puerile breathing, *dyspnœa*, narrowing of the air passages at the glottis, trachea, or bronchi, and in noisy mouth-breathing, etc.), or else improved conduction of the same sounds (pneumonic or tubercular consolidation, tumours, compression of the lung). It may also depend on the local production of a blowing sound (tubular breathing in cases of consolidated lung).

Weakened breath-sounds are the result of either imperfect production of the normal respiratory sound—*e.g.*, defective expansion of the chest, due to faulty development or to the presence of pneumothorax, emphysema, or painful affections of the respiratory apparatus, obstruction of the air passages; or of diminished conduction of the tissues (pleural effusion, thickened pleuræ, tumours, thick chest-walls, massive pneumonia).

Cavernous and amphoric breathing point to a large, fairly smooth-walled air-containing cavity. Stridor is the result of an obstruction in the bronchi, trachea, or larynx.

Variations in the intensity of a sound during the act of breathing (metamorphosed breathing, cog-wheel breathing) usually signify some obstruction in the air passages—*e.g.*, catarrh of the mucous membrane—or impaired and debilitated movements.

2. **Voice-Sounds** (speech, infantile cries, or cough) are transmitted from the larynx in all directions. Those appreciated by the stethoscope's aid follow the same path along the air columns of the trachea and bronchi, through the parenchyma of the lung

and through the chest-wall, as has been already referred to in speaking of breath-sounds. In discussing the movements of the chest-wall (p. 477) the palpable vibrations known as vocal fremitus are described. The same vibrations, when observed by the ear, give rise to the voice-sounds, or **vocal resonance**. The sound has a droning, buzzing quality, in which the articulation of words and syllables cannot be distinguished, but the sound may be loud and drumlike in character. Over those regions of the chest in which loud or bronchial breathing is normally heard the vocal resonance is loud, and gives one the impression of being generated at the surface directly under the stethoscope. Noteworthy variations in intensity of the voice-sound depend almost entirely upon changes in conduction of vibrations by the thoracic organs and tissues, as the decrease or increase of vocal resonance due to weakened or strengthened voice production is of but trifling importance to the diagnostician.

**Increased Vocal Resonance.**—An increased loudness of the sound, which is apparently close to the observer's ear, indicates in most cases consolidation of the lung. The augmented sound is termed **bronchophony**, and, if well marked, suggests that the consolidation is dense and is in close relation to large open bronchi. If indefinite, the consolidation may be imperfect, or it may be deeply placed in the chest, with intervening healthy lung tissue. It is therefore in pneumonia, phthisis, compression or relaxation of the lung, provided the bronchi are patent, that bronchophony is observed.

**Decreased Vocal Resonance.**—The voice-sound may be diminished in intensity by any condition which impairs the conductivity of the thoracic contents. In pleural effusion the sound is weaker than normal, and seems to come from a source at some distance from the surface. A thickened pleura, unusually thick chest-walls, an obstructed bronchus, pneumothorax with considerable intrapleural tension, and emphysema, all cause diminution of the vocal resonance.

The sound of the voice may be modified not only in quantity, but also in quality, by disease. It may become more or less distinct; it may have reverberating or other added qualities.

**Pectoriloquy.**—If the sounds are abnormally clear and articulate, and are apparently spoken directly into the ear, they are known as **pectoriloquy**, and are observed in conditions in which the conduction is unusually perfect. It is chiefly on listening over a

pulmonary cavity which communicates freely with a bronchus that one discovers this sign. It is, however, also found in cases of pneumothorax similarly in direct communication with the larynx, and of consolidation of the lung, connecting a large bronchus or a cavity with the surface. Pectoriloquy is not necessarily louder than normal vocal resonance; it is frequently weaker. It may also have a metallic or amphoric quality.

**Whispered Pectoriloquy.**—If the patient is directed to whisper instead of speak while the chest is being auscultated, the articulation may be even better observed in the conditions just mentioned. Here the sounds are not really voice-sounds, but breath-sounds, to which are added various reverberations from the buccal and naso-pharyngeal cavities.

**Whispering Echo.**—In large pulmonary cavities one hears at times during phonation, in addition to the bronchophony, a sort of whispering echo, the result of reverberation from the walls of the cavity.

**Egophony.**—A curious nasal or whining quality of voice is to be heard over the situation of a pleural effusion, especially, but not exclusively, at its upper limits. It may also be observed in exceptional cases over pulmonary consolidation without fluid in the pleural cavity. This sound, known as **ægophony**, from its resemblance to the bleating of a goat, is probably due to interception by the fluid of the fundamental tones of the voice and the persistence of discordant overtones and harmonics. It is also attributed by some to compression of the bronchi interfering with the normal conduction and resonance taking place in those air columns.

**Baccelli's Sign.**—In serous effusion the whispered voice can usually be heard distinctly enough, but it has been pointed out by Baccelli that a purulent pleural effusion prevents a whisper reaching the stethoscope.

**Summary of Voice - Sounds.**—Vibrations audible at the surface of the chest, and originating in the vocal cords, give rise to vocal resonance. This may be increased (bronchophony) by improved conductivity of the tissues, and is found in pneumonia, phthisis, compression or relaxation of the lung. The contrary condition, decrease of vocal resonance, results from impaired conductivity of the thoracic contents, and occurs with pleural effusion, thickened pleura, fatty chest-walls, obstructed bronchi, pneumothorax, emphysema.



Pectoriloquy is chiefly a sign of pulmonary cavity, and may occur as a result of consolidation of the lung or of pneumothorax. Whispered pectoriloquy is an even more delicate index of the conducting power of the lung.

Ægophony is heard over a pleural effusion, chiefly at its upper limits. It may also occur from pulmonary consolidation.

3. **Adventitious Sounds.**—In addition to the breath and voice sounds and their modifications, one may observe, on auscultating the chest, certain fresh or **adventitious sounds**. Most of these can at once be ascribed to affections of the respiratory or of the circulatory systems, judging by their quality, rhythm, situation, etc. Those concerned in respiratory affections will be first considered.

(a) **Rhonchi.**—Among the somewhat embarrassing richness in variety of sounds obviously respiratory in origin, a characteristic group known as **rhonchi** can be readily distinguished. A definitely musical sound, repeated with each inspiration or expiration, of all varieties of pitch, is heard either singly, in solo performance, or many notes in chorus (compared to a 'nest of kittens'). Those tones of low pitch are spoken of as **sonorous rhonchi**, while the higher-pitched notes are termed **sibilant rhonchi**. In describing the sounds various familiar comparisons are commonly made. Thus we may speak of them as snoring rhonchi, as cooing, whistling, grunting, groaning, whining rhonchi, according to the suggestion evoked by the character of the sound. When the sounds are audible to the unaided ear, as they frequently are, the term **wheezing** is used. Rhonchi may be heard over any portion of the pulmonary region, and originate in the bronchi from localized narrowing of their calibre. This may occur from thickening of the mucous membrane, from the deposition in the tube of viscid and non-fluid mucus, or from spasm of the muscular coat of the bronchioles. Sonorous rhonchi are produced in the larger bronchi, and are often spoken of as **large rhonchi**, while sibilant rhonchi, originating in the smaller air tubes, are also known as **small rhonchi**. When heard over large areas of the chest, they are a sign of bronchitis, asthma, or more rarely of phthisis. In the first condition there may, perhaps, be also audible sounds suggesting the presence of a more fluid secretion in the tubes (*râles*; see below), and there may be a more or less copious muco-purulent expectoration. In asthma the sounds are mostly sibilant, chiefly heard in the prolonged expiratory act, and are not productive of

much cough or expectoration. Localized rhonchi, especially if heard best at the apex of a lobe, and if accompanied by crackling sound, but even if they are the only adventitious sounds present, suggest an early or catarrhal stage of phthisis.

(b) **Râles.**—An immense group of sounds is next to be recognized, which have in common a crackling, rattling, or indistinctly bubbling character, conveying to the ear the impression of the passage of air in a current or in bubbles through fluid which must vary considerably in different cases as to quantity, density, viscosity, etc., or they may suggest the separation of contiguous sticky surfaces. These sounds have received from different observers a host of descriptive names, but are most conveniently indicated by the generally accepted French term *râle*, meaning a rattling or crackling sound. Qualifying adjectives can, then, be added without limit to designate the numerous varieties of *râles* heard in respiratory affections.

In the first place, it seems advisable to avoid the use of the expressions 'dry *râle*' and 'moist *râle*.' All *râles* originate in moist surroundings—in some, no doubt, the moisture is more pronounced than in others. The term 'dry *râle*' is used by some writers to indicate rhonchi, while others apply it to a clear, non-musical, crackling *râle*, suggestive of scanty fluid.

Another term that has been used in different senses is **crepitation** (L. *crepito*, to crackle). It corresponds etymologically to *râle*, and is often employed as a synonym for the latter. At present it is commonly used to indicate a very finely-divided *râle*, which might be more consistently and explicitly, if less concisely, designated **crepitant *râle***.

The following is a brief description of the various *râles* to be heard in the chest. Four groups may be formed, in accordance with the apparent fineness or coarseness of the sound.

(i.) **Crepitant *Râles*** are, as just stated, very fine crackling sounds heard almost exclusively during inspiration. They have a crisp quality, and may be compared to the sound produced by rolling a small lock of hair between the fingers near the ear, or by tearing paper. They may be heard in the rare instances that one has the opportunity of examining the lung in the earliest stage of croupous pneumonia, while engorgement has taken place and before consolidation has been established (*crepitatio indux*). A similar, though often coarser, *râle* may be heard in the earliest period of resolution of pneumonic consolidation (*crepitatio redux*).

They are also heard in many cases of catarrhal pneumonia, in hæmorrhagic infarction, and in early œdema of the lung.

The sound is probably produced by the act of inspiration drawing apart the walls of air cells and of the smallest bronchioles, which, owing to the above-named affections, are in contact and somewhat adhesive. By some observers (Osler, Lindsay) it is suggested that the sound is not an intrapulmonary râle, but is an ill-defined pleural friction-sound. It occurs in croupous pneumonia where pleurisy is the rule, and is usually accompanied by pain. Against this view are the convincing facts that a similar, if not identical, sound is heard in catarrhal pneumonia and in pulmonary œdema, in which condition pleurisy is exceptional, and that in these conditions, as well as in croupous pneumonia, the sound is usually heard only during inspiration. Further, it is stated that crepitant râles may be heard in healthy lungs, especially at the apices on deep inspiration, when the patient has habitually under-inflated his lungs by shallow breathing. The sound may often be distinguished from fine pleural friction by the disappearance after coughing. (See p. 420, below, in reference to the indiscriminate use of the terms 'consonating râles' and 'crepitant râles.')

(ii.) *Subcrepitant Râles*.—Crackling sounds, not quite so fine or minute as the foregoing, are known as **subcrepitant râles**. They may be clear and distinct, an evidence of good conducting quality of lung, such as is found in consolidation from any cause, or they may be dull, indistinct, and blurred, from imperfect conduction, which may result from the interposition of spongy lung tissue, pleural effusion, thickened pleural membrane, etc. They are produced in the small bronchi, and indicate a more copious or fluid condition of the secretion than that which causes the crepitant râles. They are, therefore, found in bronchitis, croupous and catarrhal pneumonia, pulmonary œdema, hæmorrhage, phthisis, or hypostatic congestion.

(iii.) *Mucous Râles*.—A larger and coarser class of râle is that known as the **mucous râles**. They originate in the larger bronchi or in pulmonary cavities, and are suggestive of a copious liquid secretion. They may be observed in bronchitis, bronchiectasis, and phthisis.

(iv.) *Gurgling Râles*.—Still larger and coarser bubbling or **gurgling râles** are caused by the passage of air through a collection of fluid in a phthisical cavity or in the dilated bronchi of

bronchiectasis, and by the accumulation of mucus and fluids in the respiratory passages when death is near.

The first of these groups, as already stated, is only heard during inspiration as a rule, while groups (ii.), (iii.), and (iv.) occur both during expiration and inspiration. In many cases râles are only produced when the patient draws a deep breath, or it may be necessary to make him cough. On the other hand, the râles sometimes disappear after coughing. This is especially likely to occur in cases of mild bronchial catarrh, but may be an evidence of early phthisis.

(v.) *Consonating Râles*.—Other qualities, besides the apparent size of the elements forming the râle, are utilized in order to classify the sounds. A clear, crackling, bright, resonating character is given to those sounds known as **consonating râles**, which are often observed in and around the solidified lung of pneumonia or phthisis. This quality is the result of improved conduction and resonance of the affected tissue. On the other hand, the absence of this quality is observed in the bubbling râles of bronchitis, where there is no intervening resounding or well-conducting medium.

The term 'crepitant râle' is employed by some authors to indicate the same sound as is here referred to as a consonating râle. It seems desirable that the former term should be limited to the finer variety of râles referred to at p. 418. This nomenclature restricts the term 'consonating' to the resonant, clearly-conducted quality of these râles, the word 'crepitant' being understood to indicate a *fine* rather than a *bright* sound. No doubt the fine râles of pneumonia often have this bright sparkling quality, in which case they should be termed consonating crepitant râles, just as one speaks of the coarser, sharply-conducted râles heard in phthisical consolidation and softening as consonating mucous râles.

*Metallic Râles*.—An extreme degree of this consonating quality is termed metallic or ringing râles, noted in certain cases of consolidation round large open bronchi, and in cases of cavity.

*Cavernous Râles*.—If the reverberating quality be still more marked, a cavernous or amphoric râle is the result. The amount of reverberation will depend on the size of the cavity and the condition of its walls. The râles will probably be of the mucous or gurgling types, but it is common to find râles of any of the four groups above mentioned, to which a cavernous, metallic, or

consonating character has been added in consequence of the presence of consolidation, of cavities, of dilated bronchi, etc. The recognition of these different qualities is by no means always easy, but careful practice and observation will abolish most of the difficulty and uncertainty.

(c) **Friction-Sound.**—A rubbing or grating sound may be heard over the pulmonary region, most distinct during inspiration, as a rule, but also heard with expiration. It is caused by the rubbing of the visceral and parietal pleuræ upon each other. Normally, of course, this movement produces no sound; but if the membranes have become roughened or dried by disease, their friction may be enough to set up audible vibrations. The sounds produced present great variety in quality and intensity, owing to the differences in roughness, consistency, and mobility of the surfaces affected in different cases. They may range from a faint brushing sound through intermediate gradations to a harsh, grating, scraping, or creaking noise. The fainter varieties may easily be mistaken for crepitant râles, the latter being asserted by some authorities (see above, p. 419) to be nothing but friction-sound. A fairly accurate idea of the commoner type of friction-sound is gained by completely covering one ear with the palm of the hand and gently rubbing the back of that hand with a finger of the other hand (Sahli). The rub is usually interrupted, not exactly synchronous with act of inspiration or expiration, and may closely resemble cog-wheel breathing. It may be sufficiently creaking or musical to suggest rhonchi, and has often been taken for indistinct bronchial breathing or for subcrepitant and mucous râles. Attention to the following description by Lindsay of the characters of pleural friction will minimize errors in diagnosis: 'It is rubbing or scraping (exceptionally crackling or crepitant) in character. It is "superficial." It is heard both during inspiration and expiration. It is increased in intensity by a deep inspiration. It is not affected by coughing, either as regards its intensity or its area of distribution. It is often accompanied by friction fremitus and by localized pain. It is usually confined to a small portion of the chest, but exceptionally may be heard over a wide area. Its most usual seats are the inferior antero-lateral and posterior regions of the chest. It is entirely annulled when the breath is held, unless the slip of lung over the pericardium is involved. In this latter case it may be excited or modified by the movements of the heart. It is often intensified by pressure of the stethoscope.

Examination of the chest in the neighbourhood of the doubtful sound may reveal unambiguous friction.'

When the friction-sound is restricted to the pulmonary region (see Percussion-Sounds, p. 451), it indicates pleurisy, including that accompanying pneumonia, or phthisis, and tumour. It is also said to be observed in cholera, owing to the excessive dryness of the pleura. When heard in the præcordial region it usually denotes pericarditis (see p. 446), but may be evidence of pleurisy affecting the narrow bevelled edge of lung which overlies the heart. In this case the rub might be heard synchronous with both the heart-beat and with respiration, and is termed the **pleuropericardial friction**.

If heard at the apex of a lobe, and especially of the upper lobe, phthisis is probable. The commoner situations are the infra-axillary, mammary, and infrascapular regions, where it denotes simple or tubercular pleurisy, or pneumonia.

The disappearance of the rub may be due to the separation of the pleural surfaces by effusion, to adhesion of the surfaces, or to resolution of the pleural inflammation. The reappearance of the rub, especially when it is found in the upper portions of the area of dulness, implies the removal by absorption of fluid at that region; the persisting dulness, if present, will probably be due to thickened pleural membrane.

There are a few adventitious sounds to be heard over the pulmonary region, which are unconnected with respiratory affections. A creaking sound in the scapular and suprascapular regions may be caused by movements of the scapula upon the thorax, or by grating in the shoulder-joint. These sounds may be an evidence of rheumatic affections, or of joint lesions from other causes. The inuscular sound produced by contraction of the trapezius, pectoral, or other muscles may become evident. Noises arising on the skin from carelessly applying the stethoscope, from a hairy surface, or from scraping on the skin or on the stethoscope by clothing, fingers, etc., may simulate friction-sounds. Care in examination and observation of the effect upon these sounds of respiration will in most cases obviate error.

(d) **Bell-Sound**.—A sound which has been referred to in describing the percussion-sounds (p. 461) is known as the bell, anvil, or coin sound, or bruit d'airain. It is produced by placing on the surface of the chest a coin, which is to be struck with another coin (like a hammer and anvil); while this is being performed by an

assistant, the stethoscope is placed upon an adjoining portion of the chest surface. If the pleural cavity below the coin contain air (pneumothorax), a clear, ringing, bell-like note is heard. In other cases a mere indistinct metallic sound results.

(e) **Metallic Tinkling.**—On listening over a hydropneumothorax one hears at intervals a faint but clear musical note, termed **metallic tinkling**, resembling the sound of a drop of water falling in a cistern or other reverberating air chamber. It is generally believed to be due to a similar cause—namely, to the falling of a drop of fluid from the chest-wall or lung into the serous or purulent exudation of a pneumothorax. A somewhat similar sound is produced in pulmonary cavities by the reverberations added to a mucous r le (amphoric r les).

(f) **Succussion - Sound.**—Succussion, or shaking the patient, gives rise to a splashing sound, which may be best appreciated by listening with the stethoscope, but it may be distinctly audible to the unaided ear, and the patient may be able to perceive it; in addition, it may sometimes be palpable. It is sign of fluid and air in the pleural cavity. A similar phenomenon is often observed in the stomach and colon, not only in dilatation, but also in health.

**Summary of Adventitious Sounds.**—Respiratory adventitious sounds include—

(a) Rhonchi: Sonorous or large; Sibilant or small.  
 (b) R les: (i.) Crepitant R les, the finest crackling sound. (ii.) Subcrepi. R les, a slightly coarser type of r le. (iii.) Mucous R les, large, coarse, rattling sounds. (iv.) Gurgling R les, the coarsest bubbling or gurgling sounds. Consonating R les, Metallic R les, Cavernous R les: Any of the above-named r les may be so altered in character by the presence of resonance or improved conductivity of sound, as to be placed in one of these groups.

- (c) Friction-Sounds: Produced in pleural cavity.
- (d) Bell-Sound: Produced in pleural cavity.
- (e) Metallic Tinkling: Produced in pleural cavity.
- (f) Succussion-Sound: Produced in pleural cavity.

## Part II. Sounds produced by the Organs of Circulation.

We have now to study those sounds, perceived by listening over the thorax, which can be recognized as the production of the circulatory system. Changes may be noted in the normal heart-

sounds as to their intensity, character, or rhythm; and abnormal or adventitious sounds may be heard, which may originate in the heart, the pericardium, or in the bloodvessels.

The posture of the patient has an effect upon the sounds thus produced. It is therefore advisable in many cases to auscultate when the patient is in both the upright and the recumbent position.

1. **The Normal Heart-Sounds.**—The first sound of the heart produced by, and synchronous with, the systole of the ventricles, is best heard at the apex-beat and its immediate neighbourhood. The sound, being mainly the result of vibrations set up in the mitral and tricuspid valves, is readily conducted through the ventricular walls to the chest-wall, with which they come in contact at that spot. At the apex the second sound is also audible, but is fainter than the first. This is due to the fact that the second sound is produced mainly in the great vessels by the sudden increase of tension of the semilunar valves, which occurs as soon as the intraventricular tension falls. The sound, therefore, is best heard where the great vessels are nearest to the surface—that is, over the region adjoining the second costal cartilages. The intensity of a sound diminishes rapidly as one recedes from its source; hence the second sound is only heard faintly at the apex, and for the same reason the first sound is less distinct at the base of the heart. The characteristic rhythm of the beat is therefore a trochee at the apex (lúb-dup), and an iambic at the base (lub-dúp).

Alterations in the intensity, character, and rhythm of the heart-sounds, omitting for the moment the question of murmurs or other abnormal sounds, deserve the utmost attention from the diagnostician.

**Strengthened Sounds.**—An increase in the strength of the sounds may be noted. If both sounds are augmented, and more widely heard than usual, there is either an excessive activity in the production of the sounds, or they are more efficiently conducted to the ear than normally. Examples of the former cause are seen in emotional excitement, in Graves' disease, in bodily exertion, in cardiac hypertrophy of Bright's disease, and sometimes in that of valvular heart disease. The second condition is exemplified by retraction of the lung from the præcordial area, by pulmonary consolidation, by the presence of resonating air cavities, such as pulmonary cavities, pneumothorax, or distended stomach.



Increase in the intensity of the individual sounds is of more interest than that of both. The second sound may be unduly accentuated. This results from hypertrophy of one or both ventricles, provided the semilunar valves are intact. A common example of this condition is hypertrophy of the right ventricle in consequence of incompetence or stenosis of the mitral orifice. Here the pulmonary circulation is engorged, and if the circulation be nevertheless efficiently maintained—that is, if **compensation** be effected by increased power of the right ventricle—the tension in the pulmonary artery will be raised, and the semilunar valves guarding it will close with greater force and sound. Under these circumstances an augmented second sound, loudest near the second left costal cartilage, is a favourable sign, as it is an evidence of effective compensation. If, on the other hand, a case of mitral disease do not present, or if it lose, the increased second sound, an unfavourable view must be taken of the patient's condition (see below, p. 426). Any other condition interfering with the pulmonary circulation—*e.g.*, emphysema, consolidation—will in due time and under favourable circumstances cause hypertrophy of the right ventricle, and accentuated second sound. Raised tension in the aorta and hypertrophy of the left ventricle will equally give rise to an increased second sound. In this case the place of maximum intensity is the aortic area around the right second costal cartilage. Bright's disease, renal sclerosis, arterio-sclerosis, and aortic aneurism, are the commonest causes of the intensified aortic second sound.

An accentuated **first** sound is less frequently heard alone. The most notable instance is the loud, sudden systolic sound heard at the apex in cases of mitral stenosis. This sound usually follows immediately upon a preceding murmur, but may be the only auscultatory evidence of obstruction of the mitral orifice. In cases where the murmur occurs earlier in the diastole, is indistinct, and where the heart's rhythm is disturbed, this augmented first sound may be mistaken for an accentuated second sound, the character of which it may closely resemble. Well compensated mitral stenosis may present accentuation of both sounds—of the second from compensatory hypertrophy of the right ventricle, and of the first from causes which are not quite obvious. As to the cause of the loud thumping first sound in mitral stenosis, the explanation most commonly adopted is Broadbent's view that it results from the sudden contraction of the imperfectly filled left

ventricle. It is also said to arise from the forcible and abrupt closure of the tricuspid valve, owing to the hypertrophy of the right ventricle.

**Weakened Sounds.**—The heart-sounds are diminished in intensity by all conditions which decrease the power of ventricular contraction, or which reduce tension in the aorta and pulmonary artery. They may be weakened in debility following acute disease, hæmorrhage, collapse, in failure of the heart from overexertion or degeneration of its muscle fibres, in central or peripheral nervous disease affecting the vagus, etc. Imperfect conduction of the sounds diminishes their intensity, as, for example, in pulmonary emphysema, pericardial or even pleural effusion, excessive fat or œdema of the chest-wall.

Weakness of the first sound is in some cases the direct result of diminished force of ventricular contraction. From the moment the ventricles begin to contract, the auriculo-ventricular valves, the walls of the ventricles, and the commencement of the aorta and pulmonary artery, are all thrown into a state of tension, and all contribute a share to the vibrations which constitute the first sound. This tension may be reduced by weakness of the ventricular contraction, as in the conditions referred to in the last paragraph. Diminution in the first sound in mitral stenosis is, therefore, a sign of unfavourable import, as it signifies failure of the ventricles to contract with the force which they previously possessed, and if at the same time the second sound is weakened, failure of compensation is certain (see next paragraph). On the other hand, diminution or loss of the first sound is not so ominous a sign in cases of mitral incompetence. Here the damage to the valve cusps and to the chordæ tendinæ weakens or abolishes their share in the production of the first sound, while the ventricles may still be contracting with due force. Moreover, a loud murmur produced by mitral regurgitation, occurring as it does synchronously with the first sound, may cause the latter to appear diminished, or may even mask it entirely.

When the second sound is weaker than normal, it may be due to lowered tension in the pulmonic circulation or in the aorta. If in the course of any affection causing increased tension in the pulmonary artery (pneumonia, bronchitis, emphysema, mitral disease), the accentuated second sound is found to grow weaker or to disappear, failure of the right ventricle may be diagnosed. In mitral stenosis in particular this is an important sign. Here

the right ventricle alone is hypertrophied, the left being sometimes even smaller than normal, owing to the reduced amount of blood the latter has to transmit. Hence the second sound in mitral stenosis is mainly pulmonic in origin, and failure of the right ventricle leaves both pulmonic and systemic circulation low in tension. With mitral incompetence the conditions are somewhat different. Here not only the right, but also the left, ventricle is hypertrophied, and the aortic second sound may be fairly distinct, in spite of regurgitation through the mitral orifice. Failure of the right side may still leave the left ventricle capable of maintaining a fair tension in the aorta, especially as reflux through the mitral orifice is impeded by the engorged state of the left auricle, with the result that the aortic second sound may be audible, while the pulmonic sound is lost.

Diminution in the intensity of the second sound may be caused by aortic low tension. This may be the result of conditions which weaken the ventricular contractions, and so also weaken the first sound (see above). Cases of high arterial tension without concurrent high pulmonic tension (nephritis, arterio-sclerosis, etc.) may during unfavourable periods in their course present a weakened second sound, instead of the usual augmented one. This denotes dilatation and failure of the overtaxed left ventricle. Stenosis of the aortic orifice is another and rare cause of low arterial tension and feeble second sound. Incompetence of the aortic valves is also a cause of lowered tension in the aorta during and following the time of the second sound. The imperfect closure of the aortic cusps, which constitutes the lesion, is an additional reason for weakening of the second sound.

If the tricuspid valve has become incompetent, either from endocarditis, or—as is more commonly the case—from dilatation of the right ventricle, the pulmonary tension is reduced, and the pulmonic second sound weakened.

Changes in the duration of the sounds should be distinguished from alterations in their intensity. As a rule, the sounds produced by hypertrophied ventricles are more prolonged than those caused by dilated ventricles.

**Disturbed Rhythm of the Sounds.**—Irregularities in the rhythm of the sounds, as distinguished from irregularities of the **heart-beat**, are here considered. The whole subject of arrhythmia is discussed in a separate article (see p. 37).

Accentuation of the second sound (see p. 425) may invert the

normal rhythm at the apex. Instead of being a trochee (lúb-dup), the sounds may there form a spondee, being equal in intensity (lúb-dúp), or they may even present the rhythm of an iambic (lub-dúp). Similarly, accentuation of the first sound may reverse the rhythm as observed at the base of the heart.

**Pendulum Rhythm.**—An alteration in the relative duration of the pauses separating the two sounds sometimes results in loss of the accent normally placed upon one or other sound; the result is an even series of sounds, such as might be produced by a pendulum swinging truly, the normal rhythm being that of a badly-hung pendulum. This may be observed in a strongly-acting heart at times with a high arterial tension (*e.g.*, in nephritis), and is probably due to prolongation of the closure-time of the heart, whereby the second sound is delayed.

**Embryocardia.**—A similar rhythm, but at a more frequent rate, is termed embryocardia, or foetal-heart rhythm, and is usually a sign of grave import. It is found in conditions of heart exhaustion, as in fevers; in diphtheritic or other paralyzes affecting the heart, and in the terminal stages of heart disease.

**Reduplication of the Heart-Sounds.**—Instead of the two normal sounds, not uncommonly one hears three, and even possibly four, with each heart-beat, the latter being, however, a rare occurrence. Either the first or the second sound may be double, and in many cases the two elements of the doubled sound follow each other so closely that it is not difficult to determine which sound is reduplicated. In other instances the three sounds may be separated by intervals so nearly alike in duration that the identity of the sounds is more uncertain.

A doubling of the **second sound** is probably caused by want of synchronism in the closure of the semilunar valves. This is usually attributed to difference in the intravascular tension of the aorta and pulmonary artery. To this theory one may object that there is normally a vast difference in the tension of the two vessels, in spite of which their valves usually close simultaneously. Sahli points out that the valves close immediately the blood ceases to flow from the ventricles, irrespective of the degree of arterial pressure, and that the second sound is not produced at that exact moment, but a little later, when the relaxation of the ventricle permits the arterial tension to put the valve in a state of tension. The sudden tightening of the valve cusps, and not their closure, would, according to this view, cause

the sound. This is a highly important difference, as it transfers the time, but not the force, of the occurrence of the second sound from the influence of the arteries to that of the ventricles. Any condition, then, which delays the diastolic fall of pressure in the ventricle will delay that portion of the second sound contributed by the valve at the outlet of the ventricle in question. Any condition, on the contrary, which hastens the ventricular relaxation will cause an earlier appearance of the respective portion of the second sound. This is in accordance with clinical experience. One of the commonest causes of doubled second sound is mitral disease. In mitral stenosis the diastolic flow of blood into the left ventricle is less active than that through the uncontracted tricuspid orifice into the right ventricle; hence the diastolic fall of pressure occurs more promptly in the left than in the right ventricle, with the result that the aortic cusps vibrate before the pulmonary. In mitral regurgitation, on the other hand, the distended left auricle will rapidly fill the left ventricle, and so delay the fall of pressure in the latter, with the result that the tension of the aortic valve occurs later than that of the pulmonary.

Further, in normal hearts during rapid respiration a doubled second sound may often be heard at the end of inspiration. This may be explained by the fact that the blood is detained by this means in the pulmonary vessels, with consequent delay in filling the left ventricle; its diastolic fall of pressure is thus hastened, and the aortic valve is heard before the pulmonary. In mitral stenosis, and during inspiration, therefore, with raised tension in the pulmonary circulation, the **aortic** valve, according to this view, is the first to vibrate, which is contrary to the opinion commonly held; while in mitral incompetence, with similar high pulmonary tension, the pulmonary valve is the first to contribute its share of the second sound. One ought, therefore, on listening over the aortic cartilage, to hear the **first** element of the reduplicated second sound more distinctly in cases of mitral stenosis, and the **second** element of this sound more distinctly in mitral incompetence; and this, according to Sahli, is what one does find. It cannot be affirmed that there is any important diagnostic significance in this interesting question as to the causation of the doubled second sound beyond the inference that the presence of this sign usually indicates mitral disease.

Apparent reduplication of the second sound may be in reality

the addition of an imperfectly-developed murmur. This may be the early diastolic murmur of mitral stenosis (see p. 439), when it is heard best at the apex, or it may be due to aortic regurgitation.

Reduplication of the **first sound** is said to occur at times from want of coincidence in the contraction of the ventricles. It is also said to occur by the production of an additional sound in the aorta, owing to vibrations set up in its walls by the expulsion wave. Its usual cause is probably the introduction of an indistinct valvular murmur. The triple rhythm heard in cases of mitral stenosis presents three sounds, usually of equal intensity, but frequently with the accent upon the second sound. They are best heard at the apex, and are in most cases the two normal heart-sounds preceded by an indefinite presystolic murmur. Instead of being presystolic, the murmur may be early diastolic, when, as mentioned in the last paragraph, it may simulate a doubled second sound, heard best at the apex.

**Gallop Rhythm.**—A triple rhythm differing from the last-mentioned in being heard both at the base and at the apex, and in having, as a rule, its third element accentuated, is known as the gallop or cantering rhythm. It probably corresponds in its time to the triple rhythm of mitral stenosis just referred to, the first of the three elements being an abnormal sound, or possibly murmur, due to passive tension of the weakened ventricular wall, or to auricular contraction. While the method of its production is uncertain, its occurrence is a feature in conditions of failing myocardium. It is commonly observed with the debilitated heart of broken-down compensation, of fatty degeneration, or of advanced Bright's disease. It may also occur with the excited cardiac action of excessive exercise, of emotional conditions, and of exophthalmic goitre.

**Altered Timbre of Sounds.**—Occasionally an unusual **timbre** or quality of the heart-sounds may be noticed. A musical sound is usually to be regarded as a murmur, but at times a tympanitic or resonant quality may be added to the sound by some altered condition of the valves which does not amount to a lesion. A heart acting forcibly in consequence of exertion or of emotions has often a metallic or ringing quality, especially if the chest-walls are thin. The presence of air in the pericardium (pneumo-pericardium), the proximity of pulmonary cavities, of pneumothorax, of a distended stomach, or of pulmonary consolidation, may add a

resonance or reverberation to the heart-sound. A ringing quality in the second sound is commonly observed in arterio-sclerosis.

In many cases a roughness or impurity of a sound is noted, and it is a matter of extreme difficulty and importance to decide if the character of the sound implies a lesion of an orifice of the heart. Sometimes a murmur can be developed in such cases by directing the patient to perform certain exercises, such as raising the body from the sitting to the standing position several times rapidly. In this case the impure sound is probably an ill-defined murmur, and an evidence of a valvular lesion. There are, however, not infrequently instances of impurity of one or other of the sounds which do not apparently depend upon any valvular lesion, but probably are due to some unusual degree of rigidity or tension of the cardiac tissues.

#### **Summary of Modifications of the Normal Heart-Sounds.**

—The first sound is normally best heard at the apex of the heart, the second at the base.

Increased intensity of both sounds is observed in excessive cardiac activity; in certain cases of Bright's disease and of valvular disease of the heart; in pulmonary consolidation and excavation, pneumothorax, etc.

Increase of the second sound occurs in mitral disease compensated by hypertrophy of the right ventricle; in emphysema, pulmonary consolidation, Bright's disease, arterio-sclerosis, aortic aneurism.

Increase of the first sound is well marked in mitral stenosis.

Decreased intensity of both sounds occurs in debility, acute disease, hæmorrhage, collapse, degeneration of heart muscle, nervous diseases, emphysema, pericardial or pleural effusion, etc.

Weakness of the first sound is the result of the conditions first enumerated. Its chief diagnostic value is in cases of overtaxed right or left ventricle.

Weakness of the second sound is also noted in failure of either ventricle. It is found in aortic incompetence, aortic stenosis, and in tricuspid incompetence.

Prolongation of the first sound indicates hypertrophied ventricles; a shorter sound denotes dilatation.

The rhythm of the heart-sounds may be altered: embryocardia indicates heart exhaustion; pendulum rhythm often denotes high arterial tension.

Reduplication of the sounds is caused by want of synchronism

of the semilunar valves as to the onset of their tension, and by the introduction of additional heart-sounds or of unrecognized murmurs.

Doubled second sound usually denotes mitral disease.

Doubled first sound is usually an unrecognized murmur.

The gallop or cantering rhythm generally denotes a failing heart.

A metallic quality of the sounds may be noted when the heart is acting forcibly, and in arterio-sclerosis.

Reverberation or resonance may be added to the heart-sounds by adjacent cavities or consolidations.

A roughness or impurity of the sounds may be an incipient or indefinite murmur, or may be due to some altered condition of the cardiac tissues without valvular lesion.

2. **Adventitious Circulatory Sounds.**—Abnormal or adventitious sounds originating in the organs of circulation in the thorax may be divided into two groups—viz.: (A) Endocardial and vascular murmurs, and (B) exocardial sounds.

The distinction between the two groups is not always easy, and demands careful consideration.

**Endocardial Murmurs.**—The word *murmur* is usually applied only to those sounds which are generated by a stream of blood passing into and out of the chambers of the heart or through the larger bloodvessels. Gee defines a murmur as 'any fundamental change in the character of a heart-sound, or any superadded sound heard over the heart region, pericardial sounds excepted.'

By some authorities the term 'murmur' is made to include the sounds produced by friction in the pericardial sac, but the restriction of the use of the word to the meaning just stated is more convenient.

To describe the acoustic qualities of a murmur, as compared with those of the normal heart-sounds, is an unfruitful task. Instead of, or in addition to, the familiar *lub-dup* a variety of sounds may be observed, which are described as blowing, rasping, whiffing, aspirate, etc. This is not the place to study the characters of the various sounds. They can only be learnt at the bedside, and we must confine our attention now to the significance of the sounds once they are recognized.

The blood current passing through the heart does not normally produce a murmur; there are occasions on which some unusual conditions, such as an excessively rapid current, or uncommon



elasticity of the heart or vessels' walls, may give rise to a murmur, while there is no definite lesion of the organ. Murmurs which do not depend upon anatomical changes in the heart are termed **functional, hæmic, or accidental**, the last being the most appropriate name, and it is not definitely known from what causes they arise. The following points are in favour of a murmur being accidental: Anæmia or fever may be present; there may be a **venous hum** (see p. 444). Other evidence of cardiac affection may be wanting—*e.g.*, history of rheumatism or other sufficient cause may be absent, no enlargement of the heart nor accentuation of the second sound, no changes in the pulse, nor signs of impaired circulation in the various organs and tissues of the body. The murmur is most commonly heard best over the pulmonary orifice. It is systolic in time; it is usually soft and blowing in character; it disappears if the patient's general condition improves.

The first requirement for the production of a murmur is a sufficient rapidity of blood current. Indeed, it is possible, judging from the experimental production of murmurs in smooth-walled tubes of glass, rubber, or other material, that excessive rapidity of current through normal channels may give rise to murmurs (Weber). More commonly the sound is the result of vibrations set up either by friction of the fluid upon some abnormal roughness of the inner surface of the blood channel, or by the formation of a **fluid vein** (see p. 147). The latter condition is the usual origin of cardiac and vascular murmurs, and consists of a series of movements and vibrations in the fluid consequent upon the propulsion of the fluid stream from a channel, the sectional area of which is less than that of the chamber or channel into which the stream is flowing; in other words, a sudden widening of the blood channel will cause vibrations in the fluid, and may be forcible enough to be transmitted through the tissues to the stethoscope. A similar result is sometimes produced by a sudden narrowing of the channel. It will, therefore, be seen that the morbid changes in the interior of the heart produced by endocarditis, atheroma, septic conditions, violence, etc., are in many cases those appropriate to the production of a fluid vein. The injury will affect especially one or more of the orifices, with the result that the opening becomes contracted (**stenosis**), or it may become dilated, or the cusps of the protecting valves may not come properly into apposition, so that leakage of the fluid occurs through an orifice which should be closed at that particular moment. The escape of

blood thus through a valve against the direction of the normal current is known as **regurgitation**, while the defective condition of the valve is termed **incompetence**. In either case fluid is being ejected through a smaller opening into a larger chamber already full of blood, and if it passes through with sufficient rapidity a murmur will result.

Lesions of the orifices causing murmurs arise in the vast majority of cases, primarily, at any rate, from endocarditis, and from degenerative processes (atheroma) commencing in the systemic arteries. It is in the left ventricle almost exclusively that these affections locate their destructive influence. A considerable number of cases are the result of secondary degenerative changes in the heart's muscle, with dilatation of the chambers and of their apertures. In this process the right heart takes a large share.

A murmur being detected, we have to inquire if it is a sign of disease in the heart, or if, on the contrary, it is merely an accidental murmur. If the former, we endeavour to form an opinion as to which portion of the endocardium is affected, and in what manner the lesion has damaged the efficiency of the organ. In order to come to a correct conclusion on these points, one must not rely too much upon the study of the murmur. This must only be taken in conjunction with the information obtained by every means of investigation (see p. 99).

In studying a præcordial murmur one must determine (*a*) at which spot on the surface of the chest it is most distinctly heard, and (*b*) with which of the periods of the cardiac cycle it corresponds—that is, what relation it bears to the work being done by the heart at the moment. These two considerations are by far the most important, and in some cases are enough to indicate the nature and situation of the lesion which causes them. In addition, one should always note (*c*) the direction in which the murmur is most readily followed as one moves the stethoscope away from the point where the murmur is most intense, and (*d*) the character or quality of the murmur.

(*a*) **Point of Maximum Intensity.**—In many instances a murmur, especially if it be a weak or indefinite one, can only be heard over one small area of the chest surface, while in others it may be loud and insistent enough to be heard over the greater part of the thorax. It is, however, even in the latter condition, nearly always possible to mark a spot upon the chest where the sound is more

intense and distinct than elsewhere. This point of maximum intensity does not correspond exactly to the anatomical position of the orifice from which the sound emanates, but is determined by the varying conditions of the underlying structures as to their sound-conducting capacity.

Murmurs best heard in the immediate neighborhood of the apex-beat are nearly always produced at the mitral orifice. If due to regurgitation they may be loudest just outside the limit of apical pulsation, while the murmur of mitral obstruction is usually a little nearer the sternum.

If the point of maximum intensity be found to lie at the lower end of the sternum, or close to either side of its lower part, the lesion is probably at the tricuspid valve. A lesion at this orifice is almost always regurgitation, and is synchronous with the apex-beat (see p. 472). About the same position a murmur caused by regurgitation through the aortic orifice is frequently heard at its best, but being diastolic in time it is not easily mistaken for the tricuspid lesion. About the middle of the sternum or down its left side is the region in which this murmur is most commonly loudest.

Murmurs heard best in the aortic area—that is, in the immediate vicinity of the second right costal cartilage—originate at the aortic valve or in the aorta. In only a certain proportion of cases, however, the murmur due to aortic regurgitation is best heard at this place, for it is often most distinct in the position mentioned in the last paragraph.

At the pulmonary area, the corresponding region to the left of the sternum, murmurs generated at the pulmonary orifice and in the pulmonary artery are best heard. It is maintained by some observers that murmurs produced by mitral regurgitation are often best heard in the pulmonary area. On this point there is considerable difference of opinion.

In many cases there seem to be more than one situation more favourable than the rest of the thoracic surface for perceiving the murmur. For example, a murmur synchronous with the heart's impulse may be distinctly heard at the apex, and on gradually moving the stethoscope by short intervals towards the sternum the sound diminishes in intensity for a certain distance. Then, as the sternum is approached, the murmur grows louder again, and is quite distinct at the lower end of the sternum. This is an instance of two distinct murmurs due to separate lesions, the

apical one being the result of mitral regurgitation, while that heard behind the sternum denotes tricuspid regurgitation. The identification of these lesions is, of course, dependent upon corroboration from other sources—*e.g.*, the character of the pulse, the size and shape of the heart, pulsation in the veins, enlargement of the liver, etc.

Various combined lesions are commonly observed, regurgitation and stenosis being often present at the same orifice, or several orifices may be affected in different ways

(*b*) **Time of the Murmur.**—The physiological cycle of the heart-beat may for clinical purposes be divided into four periods: (i.) **Systole**, or period during which the ventricles are contracting and the first sound is occurring; (ii.) **post-systole** or **early diastole**, a short period immediately following the systole, synchronous with the second sound; (iii.) **mid-diastole**, or period of rest; and (iv.) **presystole**, the period immediately preceding the ventricular systole; during this period the auricles are contracting. The last three of these periods together form the diastole of the ventricles.

In order to ascertain in which of these periods a murmur occurs, one judges its position in the cycle either by referring to the normal sounds, if they are audible, or by noting the relation of the murmur to the apex-beat, if it be visible or palpable, or, if not, by palpating the carotid artery. The radial pulse is, of course, no indication of the ventricular systole, as the wave causing pulsation requires an appreciable time to reach the wrist.

**Systolic Murmurs.**—In many cases a systolic murmur may be found in patients who show no signs of organic valvular disease. The exact significance of these accidental or functional murmurs is not clearly understood, and they may generally be recognized by the signs and conditions mentioned on p. 433.

Excluding accidental murmurs, those which are systolic in time must obviously be caused by the blood stream leaving the ventricles by one or more of the four available apertures. If the lesion be at the aortic or pulmonary orifices, or in those vessels, it is an **onward** or **obstructive** murmur, being caused by the stream proceeding onward in its normal direction; if at the mitral or tricuspid orifices, the murmur must be due to the escape of blood in a direction contrary to that of the normal stream, and is therefore termed a **regurgitant** or **reflux** murmur.

Systolic murmurs, heard best at the apex, form a large propor

tion of the abnormal cardiac sounds. The leakage through the mitral valve, to which they are due, is the result of endocarditis in the larger number of instances. This affection selects the left ventricle almost exclusively, and the mitral valve and its attachments by preference, though the aortic orifice frequently suffers as well. In many cases incompetence of the mitral valve is due to quite a different cause—namely, dilatation of the orifice as part of a dilatation of the left ventricle. Here the cusps may be unable to properly meet and retain the blood during the ventricular systole, and a systolic murmur results. Anæmia is, perhaps, the commonest cause of dilatation of the left ventricle, giving rise as it does to fatty degeneration of muscular tissue. Fevers (including rheumatism) debility, high arterial tension (as in Bright's disease), excessive exercise, are among the principal causes of dilated left ventricle.

A systolic murmur, best heard at the lower end of the sternum (tricuspid area), is due to tricuspid regurgitation, often the result of pulmonary engorgement, or it may form a part of a general degenerative dilatation of the heart.

In the pulmonary area a systolic murmur is commonly 'accidental,' and is a usual sign in chlorosis and other forms of anæmia. Its origin is not definitely known.

The presence of a systolic murmur in the aortic area does not of necessity denote a valvular lesion. If it be of a rough, rasping character, and be plainly audible in the vessels of the neck, if the second sound at the aortic area be faint or absent, if the heart be hypertrophied, while the pulse is small and of moderate tension, and if a systolic thrill be felt at the aortic area, one is justified in diagnosing the rare condition of aortic stenosis. It is more frequently due to a roughening of the inner surface of the aortic orifice or of the aorta, and it may be an evidence of a dilated aorta. It is also said by some observers to be in many cases simply an accidental murmur, similar in nature to that heard over the pulmonary orifice in anæmia and other conditions as mentioned above.

In rare instances a systolic murmur heard best at the base of the heart may be due to congenital defects, such as a patent foramen ovale or a contracted pulmonary orifice. Pressure of enlarged glands or of a tumour upon the large vessels in the mediastinum may also be responsible for a similar murmur.

A murmur is occasionally heard at the apex, which is really

systolic, but, occurring as it does in the last portion of the systole, is often spoken of as **postsystolic**. A more accurate term is **late systolic**, while the term **prediastolic** is preferred by Sahli and others. It is heard in certain cases of mitral incompetence in which the valve is efficient in the earlier portion of the ventricular contraction, but fails toward the end of systole. This may be explained by an anomaly in the chordæ tendinæ or papillary muscles, or by dilatation of the engorged left auricle, producing relative incompetence of the valve toward the end of the ventricular systole when the auricle had had time to be filled by the engorged pulmonary veins.

The prediastolic murmur should be carefully distinguished from those occurring in the postsystolic or earliest period of diastole. The latter may be preferably termed **early diastolic** murmurs, and are caused by the entrance of a current of blood into the ventricle, which at that moment commences its relaxation. Normally the passage of blood from the auricles through the mitral and tricuspid valves is silent, for the current is too languid in the early diastole, and the aperture is too wide to produce audible vibrations. If, however, one of these orifices be constricted—and it is almost invariably the mitral which is so affected—both these conditions requisite for a murmur are supplied. The stenosis of the aperture raises the tension in the left auricle to such a degree that in many cases (where compensatory hypertrophy of the right ventricle has occurred), the moment the left ventricle has ceased to contract, a stream of blood is forcibly propelled from the engorged left auricle into the ventricle. The stream may be sufficiently rapid, and the orifice may be contracted enough to produce a fluid vein of sufficient force to become audible. The result is the **early diastolic** or **postsystolic** murmur of mitral stenosis. The presence of this murmur, then, is an indication (like the accentuated second sound, p. 425) that compensatory hypertrophy of the right ventricle is established to the extent of maintaining a high tension in the pulmonary circulation. As soon as the tension in the left auricle is relieved by the escape of a portion of its contents into the ventricle, the current slows down and the murmur weakens or disappears. The exact moment at which the sound disappears depends upon the degree of tension established and the promptness of its relief. In some cases it is only heard in the earliest part of the diastole, while in others it may persist into the mid-diastolic period, with only the shortest possible pause, or no complete cessation before the onset of the

presystolic murmur which is so commonly heard under these circumstances (see below). Careful auscultation of cases of mitral stenosis will prove that the early and mid-diastolic murmur is much more frequently present in this lesion than is generally supposed.

A postsystolic murmur originating at the aortic orifice must mean the reflux of blood back to the ventricle through an incompetent valve. Here the regurgitation is usually a fairly prolonged process, and continues into the mid-diastolic, or even into the presystolic, periods. Unlike the postsystolic current through the mitral orifice, causing the murmur described in the preceding paragraph, the reflux current through the incompetent aortic valve does not materially reduce the tension to which the stream is attributable; hence the rapidity of the reflux current persists, with but little diminution, as long as the intraventricular tension is low—that is, until the left auricle has emptied itself into the ventricle. For this reason the murmur arising from aortic regurgitation is usually prolonged throughout the greater part of the diastole.

In addition to the diastolic murmur above mentioned, aortic regurgitation with dilatation of the left ventricle sometimes gives rise to a murmur in the mid-diastolic or late diastolic periods, which may be distinguished from either of the above by its position at the apex of the heart, by the absence of accentuation of the first or second sound, by the presence of signs of aortic regurgitation and of dilatation of the left ventricle. This is known as **Flint's murmur**, and is probably occasioned by a reflex current passing from the aorta through an incompetent valve into the dilated ventricle, where it strikes the anterior cusp of the mitral valve. The cusp is thus displaced from the position it usually occupies during diastole in contact with the ventricular wall, and is set in vibratory movement by the stream from the incompetent aortic or possibly from the auriculo-ventricular orifice.

A diastolic murmur originating at the pulmonary orifice is a rare occurrence. The presence of pathogenic organisms in the blood may give rise to septic or ulcerative endocarditis, which not uncommonly attacks the right side of the heart. The murmur occupies the same period in the cardiac cycle as that produced by aortic regurgitation.

Murmurs heard in the period of **presystole** coincide with the contraction of the auricles. This normally belongs to the silent

portion of the cardiac cycle, and is produced under abnormal circumstances by the entrance of blood into the ventricles. The early and mid-diastolic murmur of aortic incompetence may be prolonged into this period, and Flint's murmur may also occupy this portion of the cycle, but the characteristic presystolic murmur is that of mitral obstruction. It is a short, sudden murmur, synchronous with the rapid contraction of the auricle, and is doubtless the result of the forcible ejection by the hypertrophied and dilated left auricle of its contents through the narrowed mitral orifice, thus forming a 'fluid vein.' The murmur is not invariably present in cases of mitral stenosis. In this, as in other valvular lesions, the audibility of the vibrations set up by the blood-current depends on several circumstances, the chief of which are rapidity of the current, difference in the relative size of the orifice and of the chamber into which it opens, and the elasticity and tension of the tissues. In some cases of mitral obstruction the murmur may be absent, while a thrill can be felt. Here the vibrations are probably sufficiently ample to be palpable, but too infrequent to be audible. It will now be seen that the early diastolic murmur sometimes observed in this affection (see above, p. 438) differs in at least one important point as to the mechanism of its production from the presystolic murmur. The latter originates in the emptying of the auricle by its **physiological** contraction, while the early diastolic murmur is the result of a **physical** condition—namely, escape of blood from the auricle owing to intra-auricular high tension. It therefore indicates an active and hypertrophied left auricle.

A rare condition, causing a presystolic murmur, is tricuspid stenosis. This may be a congenital lesion, or may be part of a widespread affection of the endocardium. It is best heard at the lower end of the sternum, and is accompanied by cyanosis, œdema, and distension without pulsation of the jugular veins. (See Liver Pulse, p. 189.)

(c) **Transmission of the Murmur.**—In many cases the murmur is better conducted along certain lines than others on the surface of the chest. This is ascertained in the following manner: The point of maximum intensity is taken as a centre, and the stethoscope is moved by short steps along lines radiating in all directions from the centre. The line along which the murmur is best heard is noted, and is known as the **line of transmission** or **conduction**. The direction of this line in the respective lesions is



determined by variations in the conducting capacity of the organs and tissues interposed between the origin of the sound and the stethoscope, and by the set of the blood stream. It depends, therefore, not only upon conduction, but also upon **convection** of sound.

An apical systolic murmur transmitted toward the left axilla denotes mitral incompetence. If the transmission in this direction is so marked as to render the murmur distinct in the neighbourhood of the inferior angle of the left scapula, this lesion may be confidently diagnosed.

A systolic murmur best heard well inside the apex-beat, and conducted across the sternum, is characteristic of tricuspid regurgitation.

The murmur occurring in the period of presystole and due to mitral stenosis is but slightly conducted as a rule. Its direction is rather towards the sternum. The early diastolic murmur of this affection is still more localized. Similar deficiency in conduction is to be noted in the other apical diastolic murmur mentioned above—namely, Flint's murmur.

The diastolic murmur resulting from aortic regurgitation is transmitted best downward toward the ensiform cartilage.

A systolic murmur transmitted from the aortic (second right) cartilage upward into the vessels of the neck is due to a lesion of the aortic orifice, either roughening of the interior of the orifice or (rarely) stenosis. It may also be the result of dilatation of the aorta, which in many cases is in so slight a degree as hardly to merit the name of aneurism. This murmur is often extensively transmitted in all directions.

(d) **Character of the Murmur.**—The value of modifications in the quality or loudness of a murmur as an aid to diagnosis is inconsiderable, as they depend on so many varieties of form and structure of the lesions. It is not possible to draw accurate conclusions as to the nature of the damage suffered by the valve from the timbre or quality of the murmur, which may present a blowing, whistling, musical, rough, or other character, according to the nature of the lesion and the speed of the current. The only point that can be definitely affirmed is that a musical, metallic, or scraping quality is not heard in accidental murmurs. Loudness or faintness does not constitute any measure of the amount of injury present. A weak murmur may indicate a slight lesion which, with the ordinary speed of the blood current, is only just

enough to set up audible vibrations. On the other hand, its feebleness may be due to a languid current, resulting from rest in bed, debility, cardiac failure, etc.; or the current may be strong enough, but the defect in the (incompetent) valve may be so great that the reflux current has little more cause to produce audible vibrations than the current which passes silently in the proper direction through the incompetent valve. A weak murmur with a feeble circulation, therefore, may denote a serious lesion, while a weak murmur with a strongly-acting heart indicates either a trifling lesion, in which case the radial pulse is of fair tension, or a serious and advanced incompetence; in the latter condition the pulse at the wrist is weak, compressible, or collapsing. If the murmur be loud it may be assumed that the blood current through the affected orifice is at any rate energetic, but the relation between the intensity of the sound and the amount of damage in the valve is less constant. Given a certain strength of current, the greater the stenosis up to a certain point, the louder will be the murmur; the murmur of incompetence, on the contrary, will usually be stronger in slight or moderate degrees of leakage than that of a disorganized and seriously inefficient valve.

It is a common experience that cases with a loud murmur may progress favourably, and perfect compensation may be maintained in such cases for many years. This is especially the case with mitral regurgitation, and indicates that the lesion is not sufficient to seriously disturb the heart's function. The disappearance of a murmur while the patient is obviously doing badly, and its re-appearance as recovery ensues, may be readily understood by a consideration of the foregoing paragraph.

The presystolic murmur of mitral stenosis is usually **ingravescent**—that is, it grows louder as it progresses; in this it differs from the other heart-murmurs (except the rare tricuspid stenosis murmur), which almost invariably grow progressively weaker. It is harsh and rough in tone, in which respect it resembles the murmur of aortic stenosis. On the other hand, the murmurs resulting from regurgitation through any of the three orifices usually affected (mitral, aortic, and tricuspid), are usually soft and blowing. A similar blowing character is usually observed in accidental murmurs and in those resulting from aneurismal dilatation of arteries.

Certain changes in the character of a murmur may be dependent upon circumstances unconnected with the circulatory organs. An

increased loudness may be the result of consolidation of the lung, a pulmonary cavity, or other source of improved conduction of the murmur. Similar conditions may add a hollow, reverberating, or resonating quality to the murmur.

The influence of respiration upon murmurs should be noted. If the stethoscope be placed near the edge of the lung—that is, on the area of superficial cardiac dulness and close to its boundaries—a deep inspiration will cause the lung to still further overlap the heart, and interpose its border between the pericardial sac and the stethoscope; the result is a weakening of the murmur (or of the normal heart-sounds). In addition to this mechanical interference with conduction of the sound, the production of the murmur may be influenced by respiration. Deep inspiration favours the passage of blood into and out of the right heart, and the blood pressure in the systemic circulation falls. This change is best marked during rapid breathing, with the result that the current is somewhat slower on the left side, and murmurs which happen to be originated in the left ventricle may become a little weaker during inspiration; during expiration the contrary condition prevails. If breathing be deep and slow, the inspiratory augmentation of the left ventricular sounds only occurs during the first part of inspiration. During its later periods the pulmonary and aortic tensions are more equalized by the passage of a larger quantity of blood from the dilated pulmonary vessels into the left heart. While the changes thus produced in intensity and character of murmurs are slight and may easily be overlooked, they may in difficult cases be of assistance in distinguishing murmurs arising in the left heart from those of the right ventricle. The flow of blood from the great veins into the heart may be retarded by raising the intrathoracic pressure still more than occurs during ordinary expiration. This may easily be accomplished by Valsalva's experiment, which is accomplished by making efforts of forcible expiration while the glottis is kept firmly closed. By this means endocardial murmurs become fainter or may even disappear, owing to the sluggishness of the current through the heart. One may thus in certain cases distinguish doubtful endocardial murmurs from pericardial friction-sounds, which are often increased in intensity by this procedure.

**Vascular Murmurs.**—Murmurs may be heard over the large vessels of the thorax. Those over the aorta and pulmonary artery are referred to at p. 435, and it may be added here that the

accidental murmurs mentioned on pp. 433 and 439 are considered by some observers to be of vascular origin, while others regard them as evidence of valvular imperfection.

On placing the stethoscope lightly over the **subclavian artery**, either above or below the clavicle, a murmur, usually loud and rough, may frequently be heard. It occurs in a small proportion of cases in perfectly healthy persons, and is generally heard during inspiration, when it is probably produced by a bending or kinking of the vessel as the apex of the lung ascends while the lung is expanding. In many cases it is probably due to pleural adhesions pressing upon or twisting the artery, and may then be regarded as a sign of phthisis. A fictitious systolic murmur may be produced by pressure of the stethoscope on this, as on any other large artery. In addition to this loud murmur, the two heart-sounds may often be faintly though distinctly heard on listening over the vessel. The auscultatory sounds of the arteries are discussed in the article on the Examination of the Arteries (p. 42).

**Venous Hum.**—A vascular murmur of considerable importance, which is practically a thoracic sound, is that heard at times in the veins of the lower portion of the neck. It is best heard on the right side as a rule, with the patient in the upright position and the head turned slightly away from the side under examination. The stethoscope should be placed just above the inner end of the clavicle, in light contact with the skin and avoiding pressure. The sound is known by various names—viz., venous hum, bruit de diable, humming-top murmur, Nun's murmur. It is frequently quite loud, and snoring or roaring in quality, or it may present a blowing, humming, or musical tone, continuous as a rule, with slight rhythmical variations of intensity, being increased during inspiration and in diastole, when the venous current is swiftest. In some cases the venous hum is so feeble that it can only be heard at the times just mentioned. The sound may occasionally be observed in health, and especially in children, but it is usually found in cachectic conditions characterized by anæmia, of which chlorosis is the commonest example. It may also be heard in many cases of exophthalmic goitre.

The origin of this sound is in the jugular veins, but the exact mechanism of its cause is uncertain. Like the accidental murmur heard over the pulmonary artery in anæmia, it has been attributed to the formation of a fluid vein, owing to a hypothetical decrease

in the quantity of blood in anæmia. It was supposed that such a diminution of the fluid contents of the internal jugular vein (or, in the case of the pulmonary murmur, of the right ventricle) would result in the contraction of those channels, while the lowest portion of the jugular vein, owing to its attachments to the cervical fascia, and the pulmonary artery, owing to its inferior contractility as compared with the right ventricle, remain relatively larger in size. The disproportion between the sectional area of the respective channels might, if the current were rapid enough, produce the fluid vein. Unfortunately for this theory, the total quantity of blood in chlorosis (which very commonly presents this sign) is not diminished, but is considerably increased, as Haldane and Lorrain Smith have shown, and the bruit is often heard in cases where there is no reason to suspect any departure from the normal quantity or quality of blood. There seems, however, to be a definite relation between the intensity of the sound and the rapidity of the current, as shown by the increased loudness in the upright position, during inspiration and during diastole. In some anæmic conditions an increased quantity of the blood, or a diminished viscosity of the fluid, may produce a swifter current than normal. In others the sound may be due to some local alteration in the size of the vessels, from changes in the venous valves, pressure of enlarged glands, or other cause.

The presence of this sign is occasionally of diagnostic value. When it accompanies an apical systolic murmur, it is a strong point in favour of the presence of relative incompetence of the mitral orifice, due to myocardial rather than endocardial lesion. If, as will probably be the case, the patient be anæmic, the lesion is likely to prove a temporary degenerative change in the heart muscle, and not a permanent deformity of the valve, which results from endocarditis. A similar temporary degeneration may occur in the wall of the right ventricle from anæmia, producing dilatation of the conus arteriosus and of the base of the pulmonary artery (shown by pulsation in the second left interspace). The accidental or hæmic murmur heard at that region in anæmia may perhaps be thus accounted for.

**Exocardial Sounds.**—In addition to the foregoing sounds arising from the interior of the heart and great thoracic blood-vessels, certain sounds may be heard synchronous with the heart-beat, and obviously dependent upon the heart's action, but exocardial in origin. These are: (a) Pericardial friction-sound,

(b) pericardial splashing-sounds; (c) pleuro-pericardial friction-sound; and (d) cardio-pulmonary and other sounds.

(a) **Pericardial Friction-Sounds.**—As a result of inflammation of the pericardial lining membrane, a roughening of the surfaces occurs, which gives rise to a variety of friction-sounds, similar to those consequent upon pleurisy. They vary considerably in quality and intensity, presenting all degrees of fineness and coarseness. They may be of a scraping or scratching quality; they may be crackling; they may be loud or soft; and in many cases, especially when soft and crackling, they may be easily mistaken for endocardial murmurs. As a rule, however, they are rough and harsh or rasping in character, with a sawing or to-and-fro rhythm, and give the observer the impression of arising from the surface under the stethoscope. The sound is usually heard over a very limited area of the chest, but if very loud it may be extensively heard. It is commoner at the base than at the apex of the heart, but may be heard (and often felt) over any part of the præcordial area. Its **time** does not correspond with any particular period of the cardiac cycle, not limiting itself to the moment that the blood current is passing through one or other of the orifices. It may come on quickly and disappear without warning. It may persist in spite of the presence of a fairly large pericardial effusion, as the fluid tends to collect at the sides of the pericardial sac, leaving the anterior portion of the heart free to come in contact with parietal pericardium, or the heart may, while immersed in fluid, rub on the surface of the sac over the diaphragm. The sound may become louder by pressure of the stethoscope, by rising from the recumbent to the upright position, and by raising the intrathoracic pressure, as in Valsalva's experiment (see above, p. 443). (See the tables on p. 447.)

(b) **Pericardial Splashing.**—A splashing-sound synchronous with the heart-beat may indicate the presence of both air and liquid in the pericardial sac—a rare occurrence. Similar sounds may occasionally be heard when the heart's movements cause agitation in adjacent cavities containing air and liquid—viz., pyopneumothorax, large pulmonary cavities, distended stomach.

(c) **Pleuro-Pericardial Friction.**—A friction-sound coinciding with the heart-rhythm is at times the result of extrapericardial rubbing. It is heard best at the margin of or outside the area of superficial cardiac dulness, where pericardial friction is rarely found. It is more definitely influenced by respiratory changes

TABLE COMPARING THE PERICARDIAL AND ENDOCARDIAL SOUNDS

Symptoms.	Pericardial Sounds.	Endocardial Sounds.
<b>Character</b> .. ..	Rough, scraping	Softer; even obstruction murmurs are less harsh than pericardial friction
<b>Time</b> .. .. .	Does not correspond to any of the periods of the cardiac cycle, and varies from time to time	Is invariable, and corresponds to some fixed period
<b>Effect of pressure of stethoscope</b>	Intensifies the rub	No effect
<b>Effect of patient bending forward during examination</b>	Intensifies the rub	A greater degree of movement necessary to alter the sound; usually no effect
<b>Valsalva's phenomenon</b>	Sound is either unchanged or intensified	Murmur is weakened
<b>Transmission of faint sounds</b>	Very slight	Better transmitted
<b>Position of the sounds</b>	Frequently changes	The point of maximum intensity is usually invariable

TABLE COMPARING THE PLEURO-PERICARDIAL SOUND WITH THE PERICARDIAL SOUND

Pleuro-Pericardial Friction-Sound.	Pericardial Friction-Sound.
Best heard at outermost limit of the area of superficial cardiac dullness, or over lung in neighbourhood of that area.	Best heard over the heart where it is not covered by lung.
It has distinct cardiac and respiratory phases.	Does not alter in relation to the respiratory act.
Holding the breath may stop it.	It is not diminished by holding the breath. On the contrary, if the breath is held and expiratory effort be made with the glottis closed (Valsalva's experiment), the sound is usually intensified.
The period in which the sound is heard may indicate the situation of the lesion (see Fig. 64, p. 449).	The sound does not correspond to any particular respiratory or cardiac period.

than the pericardial sound. The roughened surfaces which cause the sound by their attrition are commonly the pleura covering the pericardial sac and that of the thin portion of lung overlying the heart. During inspiration these surfaces are brought into more forcible contact, and the friction-sound is consequently at its loudest. In certain cases the rub is most distinctly heard during expiration. Here the affected surfaces are the parietal and pericardial pleuræ, which come into contact best when the lung is retracted during expiration (See Fig. 64, and table on p. 447.)

(d) **Cardio-Pulmonary and Other Sounds.**—Sounds resembling pleuro-pericardial rubs may arise from the audible expulsion of air from an emphysematous œdematous or congested margin of lung overlapping the heart (these have usually a softer sound, resembling somewhat an endocardial murmur); from the presence of surgical emphysema in the mediastinum, resulting from ruptured alveoli, tracheotomy, and other wounds (here the sound resembles rather the râles of pulmonary affections); from diaphragmatic pleurisy, or from subdiaphragmatic inflammation. A consideration of the general symptoms and condition will usually distinguish these different affections.

#### **Summary of Adventitious Sounds of Circulatory Origin.**

—A. **Endocardial Murmurs** are—(a) accidental, (b) organic.

The murmur is studied as to its (1) point of maximum intensity, (2) time, (3) transmission, and (4) character.

(1) Apical murmurs mitral in origin; lower end of sternum, tricuspid or aortic; second right intercostal cartilage, aortic; second left intercostal cartilage, pulmonary. (2) The time may be systolic or diastolic, the latter being divided into post-systolic or early diastolic, mid-diastolic, and presystolic or late diastolic periods. Systolic murmurs are caused by the passage of blood out of the ventricles through one of the four possible apertures: if at the mitral or tricuspid, the current is reversed; if at the aortic or pulmonary, the flow is onward. Accidental murmurs are almost invariably systolic. Diastolic murmurs result from the entrance of blood into the ventricles: if at the mitral or tricuspid orifice, obstruction (in the case of Flint's murmur, see p. 439); if at the aortic or pulmonary, regurgitation. (3) Murmurs transmitted from the apex toward the left axilla are probably due to mitral incompetence; toward the sternum, to mitral stenosis; from the tricuspid area toward the right, to tricuspid incompetence;



from the upper toward the lower part of the sternum, to aortic incompetence; from the aortic area toward the neck, to aortic dilatation, roughening, or stenosis. (4) Rough, harsh murmurs are as a rule stenotic, while soft, blowing murmurs are commonly regurgitant in origin. A faint murmur with feebly acting heart may indicate a serious lesion; a faint murmur with a strongly acting heart and good pulse may denote a trivial lesion, or with a bad pulse it may denote a serious and advanced incompetence.

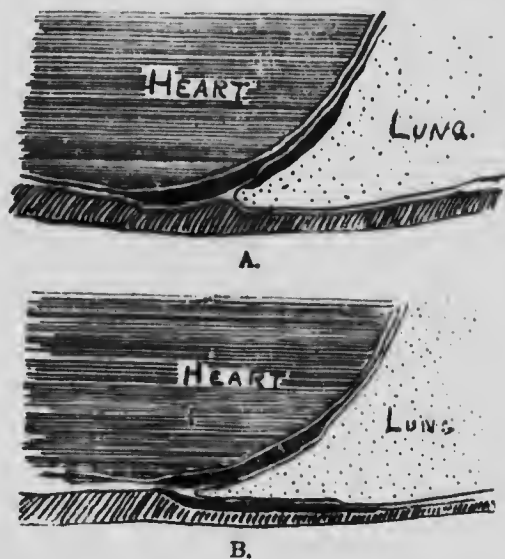


FIG. 64.—PLEURO-PERICARDIAL FRICTION. DIAGRAMS REPRESENTING THE CONDITIONS CAUSING THE FRICTION-SOUNDS WHICH ARE SYNCHRONOUS WITH THE HEART-BEAT, AND ALTERED IN INTENSITY DURING RESPIRATION.

The dark patches represent roughened serous surfaces. A, The sound is intensified on inspiration. B, The sound is diminished or abolished on inspiration.

A loud murmur indicates rapidity of the blood stream. In stenosis the greater the obstruction the louder the murmur; in incompetence a loud murmur is often less serious than a weak one. An ingravescent murmur is usually due to mitral stenosis.

**Vascular** murmurs may be heard in the thorax over the aorta, the pulmonary artery, and the subclavian artery, both in health and in disease. They are systolic in time. In the neck the venous hum of anæmia, exophthalmic goitre, and other conditions, is to be noted.

**B. Exocardial Sounds.**—Exocardial sounds are all abnormal. The chief are—(a) Pericardial friction, due to pericarditis, and rarely to new growths or blood-clots; (b) pericardial splashing-sound, from the agitation of air and fluid in the pericardial sac; (c) pleuro-pericardial friction-sound, elicited by the rubbing of roughened pleural surfaces adjacent to the pericardial sac, and moved by the heart's contractions; and (d) cardio-pulmonary sounds arising in the lung as a result of the heart-beat.

### THORAX, Percussion-Sounds of.

Resonance—The pulmonary region—Modifications of the percussion-sound in the different areas of the thorax—Traube's semi-lunar space—Objects of percussion in disease of the thoracic organs.

Increased resonance in emphysema, relaxation of the lung (Skodaic resonance), pulmonary cavities, pneumothorax.

Decreased resonance in inflammatory infiltration of the lung, infarction, congestion, œdema, collapse of the lung, cirrhosis, tumours, compression of the lung, diminished size of the lung, interposition of airless material between the lung and the surface (pleural effusion—Ellis's line), enlargement of the heart, pericardial effusion, thick chest-wall.

Amphoric resonance—Wintrich's sign—Williams' tracheal resonance—Bell-sound—Gerhardt's phenomenon—Biermer's sign—Friedreich's sign—Cracked-pot sound.

The general principles involved and the physics of percussion are considered in a separate article, at p. 279.

**Resonance.**—The various degrees of musical quality in a sound evoked by a blow delivered on the surface of the body depend upon the regularity, intensity, and rhythm of the vibrations produced by the stroke. These vibrations become reinforced, and to some extent harmonious by the presence of a resounding medium in the immediate vicinity, which takes up, amplifies, and regulates the vibrations originally started by the percussion-stroke. Air under moderate pressure in an enclosed space forms an excellent resounding medium, and the atmospheric pressure is very suitable. The number of air-containing cavities present, their size, the thickness and elasticity of their walls, the size and shape of their apertures (if they are in communication with the external air), and the character of the adjoining tissues, all combine to modify the note as to its pitch, loudness, duration, and tone. If the sound possesses these qualities in greater or less degree, it is said to be **resonant**. Other substances than air may act as

resounding media, but in an inferior degree; thus a certain added resonance may be due to the elasticity of bone, cartilage, or even of solid viscera, but for diagnostic purposes we may disregard resonance produced by these tissues.

The clearest and most musical form of resonance is that named tympanitic, which is heard on percussing over a large mass of air—*e.g.*, the stomach, pneumothorax, large pulmonary cavities, and in some cases of emphysema. The note is pitched low, and there is a reverberating or prolonged character in the sound, which distinguishes it from other percussion-sounds.

**Pulmonary Region.**—Over that portion of the chest with which the healthy lung is in contact, known as the pulmonary region, the percussion-tone is less drum-like, but has a subtympanic quality of resonance, which is modified at different spots by the quantity of air-containing lung which lies underneath, and by the density or elasticity of adjacent structures. The pulmonary region extends from about  $1\frac{1}{2}$  inches above the clavicle downwards in front to the sixth rib in the nipple line, to the eighth or ninth rib in the mid-axillary line, and to the tenth or eleventh rib in the scapular line behind. On the left side the lung usually extends downward a little farther than on the right, and the vertical anterior border of the left lung separates from that of the right at the junction of the fourth costal cartilage with the sternum, receding to the apex-beat, where it joins the horizontal anterior border, the space between the lower portions of the lungs being occupied by the pericardial sac and its contents. These boundaries refer to the condition of the chest in quiet breathing; forcible breathing alters the outlines of the lungs considerably. In the mid-axillary line the resonance during full inspiration may extend 2 inches lower than during forced expiration.

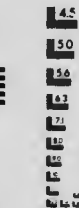
In percussing the pulmonary region of the healthy chest, we notice certain varieties of resonance in the different localities or regions of the thorax. (For an enumeration of these regions see p. 465.)

In the supra- and infraclavicular regions the subtympanic resonance is fairly well exemplified. The resonance increases somewhat towards the middle line, owing to the proximity of the trachea and larger bronchi, and diminishes in the outer parts of these regions, in consequence of the thick coating of muscle. The mammary regions are resonant above the nipples; between the nipple and the sixth rib on the right side the percussion-tone is



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somewhat duller on forcible or **deep** percussion than the areas immediately above—that is, there is **relative dulness**. This is said by some observers to be due to the deadening effect upon the note caused by the solid liver rising behind the bevelled edge of the lung, but it is more probably due simply to the fact that a forcible stroke delivered above the level of the vault of the diaphragm will bring into vibration a larger bulk of lung tissue, and will, therefore, produce a better resonance than when the thinner mass of lung below that level is caused to vibrate (see Fig. 43, p. 283). Below the sixth rib in the mammary region there is usually no lung to resound, and therefore there is no resonance, but **absolute dulness**. In order to define the edge of the lung, it is necessary to percuss very gently, as a heavy blow will cause a certain amount of lateral vibration of the chest-wall, which will bring into sympathetic vibration portions of lung tissue at some distance from the spot struck, so adding resonance to the sound elicited by striking over an airless region.

On the left side the resonance of the mammary region is encroached upon by the heart; the area between the left side of the sternum, from the fourth to the sixth costal cartilage out to the apex-beat, corresponding to the portion of pericardial sac uncovered with lung, and referred to elsewhere (p. 98), is the area of superficial cardiac dulness. On light percussion it is found to be absolutely dull, and is surrounded on all sides except below by an area of relative dulness, extending upwards and outwards one or two fingers' breadth beyond the margin of absolute dulness; to the right the relative dulness extends one finger's breadth beyond the sternum. Below, the absolute dulness is continuous with, and indistinguishable from, that due to the left lobe of the liver. The outer portion of the left mammary region receives in its lower part a tympanitic percussion-note, owing to the proximity of the stomach. This is also the case with the lower part of the left infra-axillary region. The middle portions of each mammary region may lose much of their resonance, owing to presence of the pectoral muscles, fat, or the mammary glands. The axillary and upper portion of the infra-axillary regions produce a typical pulmonary subtympaenic resonance; below the sixth rib on either side the note is modified: on the left, as just stated, it becomes more tympanitic; on the right there is relative dulness on deep percussion, owing to the thinning of the lung as it fills in the space between the vaulted diaphragm and the side of the

thorax. The sternal regions have a resonance which is somewhat tympanitic in the upper portion; and even below, where it lies directly over the heart, it usually is distinctly resonant. This is partly an osseous tone, produced by vibrations in the fairly elastic bone, but is chiefly due to the presence of air-containing lung under portions of sternum adjacent to that spot which was struck, the continuity and elasticity of the bone favouring laterally directed vibrations. The supra- and infraspinous regions are deficient in resonance, owing to the muscle and bone overlying the chest-wall. The infrascapular are the most resonant regions of the back, but are less so than the anterior areas, while the interscapular regions are slightly duller than the infrascapular.

**Traube's Semilunar Space.**—A clear tympanitic note is elicited over the lowest portion of the left side of the thorax in front, owing to the subjacent stomach. This region is known as Traube's semilunar space, and is chiefly of diagnostic interest as an aid in the diagnosis of pleural effusion. It is bounded above by the line of dullness due to the left lobe of the liver and the heart, and by the subtympanic resonance of the left lung; posteriorly it is bounded by the splenic dullness, and inferiorly by the curved line of the left costal margin. This region is more encroached upon by the dullness of left pleural effusion than by that of pulmonary consolidation, owing to the ease with which the pleural reflection may be extended downward by the fluid; its diminution in extent is also notable in enlargements of the liver, heart, and spleen.

In disease of the thoracic organs and of the chest-walls we employ percussion in order (*a*) to define the boundaries of the lungs; (*b*) to ascertain if the lungs contain the normal amount of air; (*c*) to inquire if any abnormal amount of material or tissue, which damps or imperfectly transmits vibrations, be interposed between the air cavities and the surface, such as pleural effusion, thickened pleura, excessive deposit of fat in the walls; (*d*) to ascertain if intrathoracic air be present in any other situation than the normal respiratory passages—*e.g.*, in the pleural cavities or in phthisical cavities; (*e*) to determine the size and position of the heart and great bloodvessels in the thorax, and to determine the presence of tumours, abscesses, etc., in the thorax and in its walls.

With the above objects in view, the chest is systematically

percussed, and any departure from the normal sound to be expected at the spot under examination is duly noted. The changes in the percussion-sound may be: (1) Increase of resonance; (2) decrease of resonance; (3) change in the quality or tone of the sound.

1. **Increase of Resonance** is the result of abnormally ample and forcible vibrations, and practically means an abnormally large quantity of air under the surface percussed. It is found in the following conditions:

(a) **Emphysema of the Lung** causes a hyper-resonant note, presenting in many cases a reverberating quality resembling that elicited by striking an empty wooden or cardboard box, and hence often described as the **box note**. When well marked it is heard on percussing gently over any part of the chest. The pulmonary region is increased in extent, resonance extending further above the clavicle and below the usual inferior level of the lung than usual, and the præcordial area of dulness is diminished or abolished.

(b) **Relaxation of the Lung** increases the resonance of the note. It is found that moderate tension of an air-containing cavity produces a better tone than a condition of forced stretching. In certain cases the capacity of the thorax is reduced; its negative atmospheric pressure is thereby diminished, and the elasticity of the lung is so enabled to relax and retract the organ into a smaller bulk. Examples of this condition are seen in pleural effusion. On percussing the lung immediately above the level of the fluid the tone is found to be clearer, though often of a higher pitch than normal, and is termed **Skodaic resonance**. Any abdominal affection which raises the diaphragm will relax the lung—*e.g.*, abdominal tumours, ascites, meteorism, etc. Enlargements of the heart and intrathoracic tumours may produce a similar result, and it may be possible to detect the relaxation due to commencing obstruction, atelectasis, or to pulmonary œdema. In all these conditions in which the chest's capacity is encroached upon the lung may be still further crowded out of its normal position, and compression of its tissue ensue instead of relaxation. This will tend to consolidate the lung and to **diminish** its resonance.

(c) **Pulmonary Cavities**, if large, superficial, and recently emptied by expectoration, may cause an increase of resonance, as well as the other modified percussion-sounds mentioned below (cracked-



pot sound, metallic resonance; see p. 461). Cavities in the lung are usually due to phthisis, but may be formed by abscesses of other origin. They may also be the result of bronchiectasis.

(d) **Pneumothorax** produces a marked increase in strength and clearness of the percussion-sound, which, however, is often wanting in the tympanitic quality, owing to the high intrathoracic atmospheric pressure so often resulting from a valve-like communication between the pleural cavity and the bronchus. It is in this condition that the 'coin-sound' may be heard (see p. 461).

On the right side the pulmonary region may apparently extend down to the costal margin. In other words, the liver dulness may be abolished. This is usually the result of abdominal disease (see . 10), but it may be caused by emphysema or by pneumothorax.

**2. Diminished Resonance.**—A dulled percussion-sound over thoracic areas which are normally clear indicates—(i.) a diminished quantity of air in the lung; (ii.) a diminished quantity of lung; (iii.) the presence of an unusual quantity of airless material between the lung and the surface.

(i.) The quantity of air in the lung may be diminished by (a) **infiltration of the lung** with the products of inflammation. This may be pneumonic, in which the dulness is most commonly found in the lower lobes, and in consequence is chiefly observed in the infrascapular and infra-axillary regions. Croupous pneumonia may, however, involve any portion of the organ, either from the commencement of the attack or subsequent to the bases being affected. The small masses of infiltration occurring in lobular pneumonia cannot, as a rule, be detected by percussion, unless they are so thickly distributed as to be continuous over a considerable area. Indeed, it may be said that an airless patch of less than 1 inch in diameter can probably not be relied upon to modify the percussion resonance, owing to the elasticity of the ribs, which impart vibrations to the surrounding healthy lung. Infiltration due to tubercular inflammation selects by preference an apex, either of a lung or of a lobe. It is thus commonly discovered above or below a clavicle, and not uncommonly in an interscapular region. It must not be dismissed as improbably tubercular if the lesion should affect the lower part of a lobe. Here the history and course of the affection must largely influence the diagnosis. Even where pulmonary cavities are present there

may be loss of resonance, as the walls of the cavity may be so closely in apposition as to preclude the possibility of a tympanitic note, and the surrounding tissues are certain to be infiltrated with inflammatory products, excluding the air from the vesicles and even from the smaller bronchioles. The presence of air in the larger tubes gives a slight tinge of resonance in many cases of lung consolidated by inflammation, which is conspicuously absent from well-marked pleural effusion.

(b) **Infarction** also deprives the lung of air, and if sufficiently large will cause loss of resonance over the affected region, which is commonly in the lower parts of the lung.

(c) **Congestion of the Lungs**.—The condition known as **splenization** may be produced in prolonged illness and debilitated conditions. It is mainly, but not entirely, a congestion resulting from the effects of gravitation, and is therefore termed **hypostatic congestion**. The air cells are to some extent infiltrated with blood cells and fluid, and a moderate loss of resonance occurs. The **mechanical congestion** due to chronic obstruction to the venous return from the lungs—*e.g.*, in mitral disease—giving rise in many cases to **brown induration**, does not usually affect appreciably the percussion-note.

(d) **Edema of the Lungs** may cause a dulling of the percussion-sound over the oedematous part (usually the bases behind), while the adjoining regions over normal lung may give out a hyper-resonant note, owing to relaxation of the lung (see above, p. 454).

(e) **Collapse of the Lung (atelectasis)** may be congenital, when general symptoms of impaired respiration are more prominent than modification of the percussion-sounds. It may be due to bronchitis or broncho-pneumonia, a somewhat deficient resonance being often observable. Obstruction to the entrance of air by occlusion of the larger passages, as by constriction of a bronchus by tumour or aneurism, by the presence of adenoids or enlarged tonsils.

(f) **Cirrhosis of the Lung** abolishes the resonance of the affected region. It has to be distinguished from phthisis, from pleurisy, and from malignant disease. The disease is rare, and may follow bronchitis, broncho-pneumonia (rarely croupous pneumonia), dry pleurisy. A modification of this affection more frequently seen is the chronic indurative change in the lung following bronchitis, caused and perpetuated by the prolonged inhalation of irritating

particles, a condition known as **pneumonokoniosis**, varieties of which have received the names anthracosis (coal-dust), chalicosis or silicosis (particles of stone), siderosis (steel).

(g) **Tumours of the Lung** diminish the lung resonance.

(h) Lastly, the lung may be rendered airless and toneless by **compression**, which is most commonly the result of pleural effusion. It may also depend on enlargement of the heart, mediastinal growths, and thoracic deformities.

(ii.) A decrease in the actual quantity of lung tissue is a frequent cause of loss of resonance. Several of the conditions just mentioned exemplify this. Fibrotic contraction, compression, and collapse are practically tantamount to a diminished quantity of lung tissue. Necrotic changes, tubercular and gangrenous, are actually instances of this defect.

(iii.) The interposition of airless material between the lung and the surface is a common cause of diminished resonance. An effusion of fluid into the pleural cavity is the form it usually takes. The quality of the sound is an intense dulness. When the fluid is in large quantity, there is a complete absence of resonance, differing in that respect from the dulness of consolidation of the lung where the bronchi are frequently patent, and give a slight degree of resonance to the note. As one would expect, gravitation of the fluid to the most dependent part causes the dulness to appear first in the lower portions of the pulmonary region. This usually occurs in the *infrascapular* region, but may be earliest observed in the *infra-axillary* region. In many cases of pleural effusion, and especially in those due to pleurisy, the upper boundary of the dulness does not accurately trace a horizontal line.

**Ellis's Line.**—It was shown by Ellis, and is commonly observed, that even in those cases which have maintained the upright position the fluid rises highest in the scapular or mid-axillary lines, falling as it passes forward, and to a less extent as it approaches the median line behind. This may be due to the force of gravity, with, in addition, the presence of adhesions, which may offer a resistance to the progress of the fluid in its exudation. In addition, the condition of the lung must be an important factor. The fluid will naturally make room for itself along the lines of least resistance, and the resistance will be less where the retracting power of the lung is greatest—that is, the **thick and voluminous** portions of the organ at the back and sides

of the lung. These portions, therefore, are earliest displaced from the parietes, and here the layer of fluid is thickest, while the lower anterior parts and that near the middle line behind are less bulky, less elastic, and do not so readily recede before the accumulating exudation. If the quantity of fluid is very considerable, this shape of the dull area cannot be demonstrated;



FIG. 65.—LEFT PLEURITIC EFFUSION.

The shaded area is the region of diminished resonance due to the effusion, the heart, and the liver. A, the position of the apex-beat. 'Traube's semilunar space' is encroached on by the effusion, and is reduced to a mere strip above the left costal margin.

the whole of one side of the chest may be quite dull, with the exception, perhaps, of the infraclavicular or of the interscapular regions. In hydrothorax, where the fluid is not the result of inflammation, and where, consequently, the pleural surfaces are not adherent, Ellis's line is not so often observed, the upper margin of the dull area being horizontal. On changing the

posture of the patient the upper margin of the dull area does not materially change in pleuritic effusion, but does so as a rule, though slowly, in hydrothorax. The latter condition, being usually bilateral, does not cause any displacement of the heart. If, however, the patient habitually assumes a lateral posture, the fluid may be confined to one side of the chest. A small quantity of non-inflammatory fluid may be impossible to detect if the patient be in the upright position, but by placing him horizontally on one side, a line of dulness may be perceived in the upper half of the chest, parallel to and adjoining the vertebral column.

If change of posture causes an immediate and marked alteration of the area of dulness, pneumothorax combined with fluid exudation is strongly suggested. In this condition the fluid is very mobile, and at once takes the most dependent position, provided the previous inflammation which gave rise to the condition have not resulted in strong adhesions.

If the non-resonant area due to pleural effusion be quite immovable, in spite of considerable changes of posture, adhesions are probably present, shutting off the fluid from the remainder of the pleural cavity. This may naturally occur in any inflammatory effusion, and is especially likely to result from empyema.

A triangular area of dulness was described by Grocco as occurring close to the spinal column on the healthy side in cases of pleuritic effusion. The diagnostic value of this sign is questionable (see p. 154).

The dulness due to an effusion of blood in the pleural cavity (*hæmothorax*) resembles that of hydrothorax. The blood does not usually coagulate for several days, and unless fixed by adhesions it is quite movable.

The lung may be separated from the chest-wall and pushed aside by enlargement or displacement of the heart or by mediastinal tumours. The former condition results in an increase in one or more directions of the præcordial dulness. Hypertrophy of the walls of the heart without dilatation of the chambers can produce but little increase in the dull area, as post-mortem an increase of more than  $\frac{1}{4}$  inch is rarely found unless the heart is also dilated. A distinct enlargement of the heart without dilatation may, however, be demonstrated by percussion in nephritis or in renal sclerosis, when increased arterial tension is revealed by an accentuated second heart-sound and by a hard radial pulse.

Extension of the præcordial dulness (both relative and absolute dulness) to the left usually indicates enlargement of the ventricles. If directly outwards, and, at the same time, if it invade the right side of the thorax, it is due to dilated right ventricle. If it extend downward as well as to the left it is mainly the result of left ventricular enlargement. Increase upward of the dulness, without much lateral extension of the præcordial dulness, usually indicates enlargement of the auricles or of the aorta and pulmonary artery. It may also be caused by solid tumours of the mediastinum. In such cases evidence of obstruction of the large veins and of the air-tubes may be found.



FIG. 66.—INCREASED AREA OF CARDIAC DULNESS.

A case of mitral stenosis and incompetence. The shaded area is the absolute dulness due to the heart and liver. P, P, P, areas of pulsation. T, situation of thrill; II, III, IV, on the second, third, and fourth ribs.

The præcordial dulness may be observed to assume a somewhat pyramidal shape, the blunt apex at the middle or upper part of the sternum and the broad base extending horizontally from the right of the sternum to the apex-beat. This is best seen when the patient is raised into the sitting posture, and is characteristic of pericardial effusion, without much adhesion of the visceral and parietal layers. The area of dulness differs from that of a dilated heart, chiefly in the fact that the dulness to the right of the

sternum tends towards the right as it is followed downwards, the pericardial sac distending most at its diaphragmatic attachment. Therefore the cardio-hepatic angle, or junction of the right border of the area of superficial cardiac dulness with the liver dulness, which is normally about a right angle or less, becomes increased to an obtuse angle by the appearance of dulness in the right fifth interspace, near the sternum (**Rotch's Sign**).

An unusually thick chest-wall causes the pulmonary percussion-sound to be deadened. This may be due to excessive subcutaneous fat, or to uncommonly well-developed muscles. Œdema of the chest may exceptionally produce a similar result.

3. **Changes in Quality or Tone.**—Changes in the quality or character of the percussion-sounds are noted at times.

**Amphoric or metallic resonance** is produced by percussing over a smooth-walled, fairly large cavity, which may be closed or may have a small aperture. It is said (Wintrich) that the cavity must measure at least 2 inches in its greatest diameter in order to produce this quality of sound. It may be imitated by percussing the moderately distended cheek, the mouth being preferably open, and may at times be evoked by percussing over the distended stomach or intestines. In disease it is chiefly in pneumothorax that this sign is observed, but it is also heard over pulmonary cavities.

**Wintrich's Sign.**—It was pointed out by Wintrich that on percussing over a pulmonary cavity communicating with a bronchus the resulting tympanitic or amphoric note is raised in pitch if the patient opens his mouth, and the pitch is lowered when the mouth is closed. The same phenomena may, however, be observed at times in percussing the apex of the lung in consolidation of that region. Here the tympanitic tracheal sound is well transmitted by the improved conduction of consolidated lung, and the pitch of the note is similarly altered by opening or closing the mouth. The change in pitch in the former case is known as **Wintrich's sign**, while the change in apical consolidation is termed **W. Williams' tracheal resonance**. An amphoric or reverberating and somewhat prolonged quality may be observed in both these percussion-sounds.

**Bell-Sound.**—In this connection may be mentioned a sound which is observed on auscultation with the aid of percussion, and which is known as **tinny, bell, or coin sound, or bruit d'airain**. It is heard by placing a **stethoscope** over a pleural cavity containing air (pneumothorax) while an assistant places a coin on

another region of the surface overlying the air cavity; this coin he uses as a pleximeter, striking it with another (plexor) coin. The resulting sound is a clear bell-like note, heard only while the stethoscope is placed over the air cavity.

**Gerhardt's Phenomenon.**—A change in the pitch of a tympanitic note or of amphoric resonance, coincident with change in the patient's posture, is termed Gerhardt's phenomenon (see Fig. 25, p. 152), and depends on the presence of air and fluid secretion in a pulmonary cavity. Change of posture causes the fluid to change its position in the cavity, so altering the shape of the cavity, with consequent change in the pitch or quality of the note. A similar change in the percussion-sound of pneumothorax is known as **Biermer's sign** (see Fig. 17, p. 57). It indicates the presence of fluid in addition to air in the pleural cavity.

**Friedreich's Sign.**—During inspiration the normal pulmonary percussion-sound is slightly higher in pitch than during expiration. Friedreich has shown that the differences in pitch during respiration are better marked in the amphoric resonance over a cavity.

**Cracked-pot Sound.**—Instead of the normal pulmonary resonance, a **chink** or indefinite metallic quality may be heard, such as may be produced by striking the closed hand filled with coins which have barely room to move, or by clasping the hands so that a little air is imprisoned between the palms, then smartly striking the back of the hand upon the knee, so as to drive the air from between them. This is known as the **cracked-pot sound**, or **bruit de pot fêlé**, and may often be noted on percussing the chest of an infant, especially while it cries. In adults with healthy lungs it may also be heard at times, provided the thorax is yielding, by forcible percussion near the trachea while he is speaking. This quality is added to the normal resonance by the forcible ejection of air through the glottis, narrowed for the purpose of phonation. In disease of the lungs the sound is caused by a similar escape of air through a narrow chink, but in this case it is at the opening from a cavity into a bronchus, instead of at the glottis, that the sound originates. It is, therefore, highly suggestive of a phthisical cavity of moderate or large dimensions.

The changes in the pitch of percussion-sounds mentioned above in connection with amphoric resonance apply equally to similar changes in the pitch of the cracked-pot sound. Thus



Gerhardt's sign, Wintrich's sign, and Friedreich's sign may be also demonstrated in the cracked-pot sound. It is of course, obvious that variations in the pitch of imperfect musical notes can only be perceived by trained and musical ears.

**Summary.**—That portion of the thorax with which the lung is in contact is termed 'the pulmonary region,' and presents varying degrees of resonance on percussion. A tympanitic area on the left side of the chest at its lower part is known as Traube's semilunar space, which may be encroached upon by various abnormal non-resonant conditions, notably by left pleural effusion, also by enlargements of the heart and pericardium, of the liver, of the spleen, etc.

Exaggerated resonance may be observed in (a) emphysema; (b) relaxation of the lung (*e.g.*, Skodaic resonance, or the relaxation resulting from abdominal or thoracic tumours); (c) pulmonary cavities (phthisis as a rule, also abscess and bronchiectasis); (d) pneumothorax.

Diminished resonance is due to (a) diminution in the quantity of air in the lung (inflammatory infiltration, either pneumonic or tubercular, infarction, congestion, œdema, collapse, cirrhosis, tumours, and compression); (b) diminution in the quantity of lung tissue itself (tubercular and gangrenous necrosis, also some of the conditions just enumerated, such as collapse, cirrhosis, etc.); (c) the interposition of airless material between the lung and the surface (serous, purulent, or hæmorrhagic pleural effusions; thickened pleural membrane; enlargements of the heart; tumours; excessive subcutaneous fat).

Changes in the quality of the resonant note occur in diseased conditions of the lung. (a) Amphoric or metallic resonance indicates a fairly large smooth-walled cavity, and is usually a sign of pneumothorax, but may also be observed in cases of pulmonary cavity; (b) Wintrich's sign is also found in the latter condition, while (c) Williams' tracheal resonance suggests consolidation of a pulmonary apex; (d) the bell or coin sound may be elicited in most cases of pneumothorax; (e) Gerhardt's, (f) Biermer's, and (g) Friedreich's signs depend for their production upon the presence of an air-containing cavity, and are of small diagnostic value; (h) the cracked-pot sound is an important indication of a cavity in the lung, but it may be heard at times in healthy chests, the walls of which are unusually yielding.

**THORAX: its Shape, Size, and Movements.**

Topography of the chest—The areas or regions into which the chest is divided, and the lines and landmarks by which it is mapped—The pulmonary region.

The typical shape of the chest—Mensuration—The cyrtometer—Subtypical chests: rickety chest; pigeon-breast; transversely grooved chest; flat chest; alar chest; funnel chest; emphysematous chest; scoliotic chest; kyphotic chest; unilateral and local deformities due to disease.

Movements of the chest-wall—Diminished respiratory movements—Increased respiratory movements—Litten's sign—Increased circulatory movements.

Displacement of apex-beat—Force, extent, and character of the apex-beat—Other præcordial pulsations—Epigastric pulsation.

Pulsating liver—Irritable aorta—Retraction of the chest-wall—Adherent pericardium—Broadbent's sign—Kussmaul's sign—Pulsus paradoxus.

Vocal fremitus—Friction fremitus—Thrill—Bronchial fremitus.

**Topography of the Thorax.**—The surface of the chest is, for facility of reference, mapped into regions, the bony and muscular prominences being utilized as far as possible for the purpose, with, in addition, certain artificial lines drawn vertically (see Fig. 1, p. 3). These are: (1) mid-sternal line, the median line in front; (2) side-sternal line, over each border of the sternum; (3) parasternal line, midway between the side-sternal line and (4) the mammillary or nipple line, drawn vertically through the nipple, or through the middle point of the clavicle; (5) anterior axillary line, drawn vertically through the spot where the anterior axillary fold joins the thorax, the arm being held out horizontally; (6) mid-axillary line, drawn vertically midway between the anterior axillary line and (7) the posterior axillary line, which is drawn vertically through the junction of the posterior axillary fold with the thorax; (8) scapular line, running vertically through the inferior angle of the scapula; (9) spinal line, the median line behind.

The situation of observed conditions may be described with sufficient accuracy for most purposes by referring them to one or other of the regions about to be enumerated. When more accurate indication is required, the clavicles, ribs, costal cartilages, nipples, spines of the scapulæ, and vertebral spines (of which the seventh cervical is usually easily recognized) form suitable landmarks.

The thoracic regions above referred to are briefly :

On each side in front: (1) supraclavicular; (2) clavicular; (3) infraclavicular, down to level of third costal cartilages; (4) mammary, down to level of the sixth rib in the nipple line; (5) hypochondriac, from sixth rib down to costal margins; (6) epigastric, a median region lying between the two hypochondriacs (5 and 6 refer chiefly to abdominal organs).

In the middle line: (7) upper sternal, above the angle of Ludwig; (8) mid-sternal, from second to fourth costal cartilage; and (9) lower sternal, below 8.

At the sides are: (10) axillary, above the level of the junction of the third costal cartilages with the sternum; and (11) infra-axillary, below 10, and bounded by the anterior and posterior axillary lines.

Behind: (12) supraspinous, corresponding to the supraspinous fossa of the scapula, with, in addition, the apex of the thorax behind; (13) infraspinous, below the level of the spines of the scapulæ, and as low as the inferior angle; (14) infra-scapular, from 13 down to level of twelfth rib; and (15) inter-scapular, on each side between the scapula and the median line.

Those regions of the thorax which are directly over lung tissue form the so-called **pulmonary region**. This extends from about  $1\frac{1}{2}$  inches above the clavicles down to the sixth rib in the nipple line, to the eighth rib in the mid-axillary line, and to the tenth rib in the scapular line. On the left side the pulmonary region is encroached upon by the heart. (For further particulars, see Thorax, Percussion-Sounds, p. 451.)

**Typical Chest.**—The normal chest varies to a considerable extent in different individuals. If divested of the bones and muscles of the shoulder girdle it presents a conical shape, with the apex upwards. In life, however, the circumference of the upper levels of the thorax is as great as, or greater than, the lower, owing to the presence of the muscles, etc., which attach the arm to the trunk. The chest should be practically symmetrical, though the right side is frequently slightly more capacious than the left, and there may be a very slight curving of the vertebral column with the convexity toward the right. The sternum should be straight in profile, with commonly a slight projection at the junction of the manubrium with the gladiolus sterni (angle of Ludwig), where the second costal cartilages join the sternum. The uppermost ribs join the sternum at a right

angle, each succeeding rib being more oblique, until at the junction of the lowest costal cartilages with the sternum the angle is about 45 degrees. Thus the angle formed by the two costal margins (the epigastric or subcostal angle) is about a right angle. The scapulæ should lie flat upon the back of the thorax, the shoulders should be almost horizontal, and the clavicles and spines of the scapulæ should not be too prominent.

The circumference of the chest is usually ascertained by a tape-line placed horizontally at the level of the nipples, the measurements during forced inspiration and forced expiration being compared. The respective results give an index of the thoracic expansibility. From  $1\frac{1}{2}$  to 5 inches of expansion may be seen in healthy adult chests, an average being about 3 inches. The average circumference of the male adult chest at rest is 34 inches.

The antero-posterior diameter of the chest, measured by callipers, is in adults about three-quarters the length of the transverse at the level of the nipples. In children the two diameters are more nearly equal.

An outline of a horizontal section of the chest often conveys useful information. It is easily obtained by moulding two strips of lead to the surface of the thorax, hinged by a piece of rubber tubing. The hinge is placed on the spines of the vertebræ, usually at the level of the nipples, one of the lead strips passing forward on each side to meet or cross at the sternum. This simple instrument, named a **cyrtometer**, is then opened and removed from the chest, laid on a sheet of paper in the same position as it had occupied on the chest, and its outline drawn on the paper. By this means an accurate diagram of the shape of the chest is secured.

**Subtypical Chests.**—Instead of the typical chest above described, one often meets with many subtypical forms, which may be quite consistent with health, but often indicate either past ill-health, faulty development, or tendency to disease in the future. Others, again, may denote actually existing disease.

**a. Bilateral Deformities of the Chest due to Diminished Intrathoracic Pressure.**—During the act of inspiration the atmospheric pressure is lowered. Any morbid condition, therefore, which prolongs the duration of the inspiratory portion of respiration will conduce to the production of certain deformities if the bony chest-wall is in any way deficient in rigidity. This latter

condition is found in childhood, even when the child is free from disease, and markedly when it is the subject of rickets. Obstruction in the respiratory passages from adenoids, enlarged tonsils, catarrh of the nasal, faucial, and bronchial passages, is the commonest cause of diminished intrathoracic atmospheric pressure; also, perhaps, a prolonged attack of whooping-cough. In rickets the extremely flexible chest-wall is indented even in the absence of obstructed respiration. Several subtypes of thorax result from these defects.

(1) *The Rickety Chest*.—A vertical depression is seen on each side of the front aspect of the chest, the ribs having fallen inward at their weakest part, the junctions of the ribs with the costal cartilages. Moreover, these joints are swollen and beaded, forming the **rachitic rosary**.

(2) *The Pigeon-Breast*.—Instead of the somewhat square-fronted chest of the last form, this variety presents a narrow, keel-shaped anterior surface, sloping sharply backward towards the angles of the ribs. It is usually due to similar conditions to the rickety chest, obstructed respiration taking a larger share in the process.

(3) *Harrison's Sulcus*, or the *transversely grooved chest*, is usually seen in combination with the rickety chest and the pigeon-breast. It often occurs in chests that are otherwise well formed, in which case it may not indicate any material departure from health in the past. It consists in a groove commencing at the lower end of the sternum, passing outward and slightly downward to the infra-axillary region. In some cases it occurs as a simple eversion of the costal margins, causing the lower opening of the thorax to be more expanded than normal. The origin of this condition is a yielding of the chest-walls from causes similar to those referred to above, the lower thoracic margins being supported by abdominal viscera which may have been unduly swollen and engorged, a condition which is not uncommon in rickety or delicate children.

**b. Deformities resulting from Imperfect Development.**—These denote a want of vigour and strength, and, owing to the defective capacity and movements of the chest, they often indicate a predisposition to phthisis:

(1) *The Flat Chest*.—The anterior portions of the ribs and the costal cartilages are flat, instead of being convex. If the flattening is very marked, or even sunken on one or both sides,

it may be produced by quite a different cause—namely, the retraction of a phthisical lung.

(2) *The Alar or Pterygoid Chest*.—An increased obliquity of the ribs is the chief defect underlying the abnormalities of this well-defined subtype. The result is sloping shoulders, long neck, projecting scapulæ (hence the name), chest unusually long, narrow, and shallow. It is also known as the phthisical or paralytic chest, and not only indicates a tendency to phthisis, but, if well marked, it strongly suggests the actual presence of the disease.

(3) *The Funnel Chest (Trichterbrust)*.—A depression of the lower end of the sternum, which may extend deeply, and may give rise to disturbances of circulation and of respiration. It is usually congenital, but may arise in early childhood from obstructed respiration. A similar hollowing of the lowest part of the sternum, or of the xiphoid cartilage, occurs with cobblers, caused by pressure of the last.

**c. Deformities due to Disease :**

(1) *The Emphysematous Chest (Barrel Chest)*.—The thorax appears to be in a condition of full inspiration: the antero-posterior diameter is increased, the sternum is more prominent, the spine is kyphotic, and the chest is rounder and more barrel-shaped than normal; the ribs are more horizontal, the intercostal spaces usually more sunken, and the epigastric angle more obtuse. It is due to long-continued coughing and dyspnoea.

(2) *Spinal Curvature Chests: the Scoliotic, the Kyphotic, and the Scolio-Kyphotic Chests*.—Owing to abnormal curves and twists of the vertebral column, the chest becomes distorted. (See the next paragraph, and Scoliosis, p. 355; Kyphosis, p. 174.)

(3) *Unilateral and Local Deformities due to Disease*.—(i.) Enlargement. The presence of a large pleural effusion or of pneumothorax enlarges the affected side, obliterates the intercostal spaces, raises the shoulder, and causes the spine to curve with the convexity toward the effusion. The last two deformities are due, according to Saëli, to a shifting of the centre of gravity. The lower thorax may be increased in size in consequence of enlargement of the abdominal organs—*e.g.*, the liver and spleen. Præcordial bulging follows enlargement of the heart and pericardium, especially in children. A local swelling may be seen over thoracic aneurism or tumour (see Figs. 67 and 68). (ii.) Contraction. The absorption of a pleuritic effusion, especially if it

had been copious and had lasted for a considerable time, often causes a considerable retraction of the affected side, with, in well-marked cases, lowering of the shoulder and concavity of the spine toward the side of the lesion. Chronic sclerotic and indurative changes in the lung (chronic phthisis, chronic pneumonia) and collapse of the lung produce similar retraction and deformity of the chest. Retraction of the upper regions of the thorax, as seen in phthisis, is revealed by an undue prominence of the clavicle, caused by an excessive sinking of the supra- and infraclavicular fossæ.



FIG. 67.—ANEURISM OF THE ASCENDING PORTION OF THE ARCH OF THE AORTA.

**Movements of Chest-Wall.**—The movements in health are mainly those of respiration. In addition, a movement of a small area in the fifth left intercostal space synchronous with the heart-beat can generally be observed. In females the thoracic respiratory movement is more ample and active than in males, and in chests that are thickly coated with muscle or fat the heart's pulsation may be invisible and impalpable. Many modifications of these normal movements occur in disease. The movements may be increased or diminished in amplitude, in frequency, or in

rhythm, and abnormal movements may be noted. The chest is to be inspected from all sides, as well as from above. Careful palpation, the hands being warmed, and mensuration are also to be employed.

**Diminution of Movement.**—The respiratory movements may be decreased in frequency. Normally the respiration rate is from sixteen to twenty in adults, about twenty-five at five years, and forty-five in the new-born. In some forms of coma, in



FIG. 68.—THE SAME PATIENT AS IN FIG. 65, PHOTOGRAPHED TWO MONTHS LATER, AND A WEEK BEFORE HIS DEATH.

The ring at the base of the tumour is the limit of the pulsating area. A, the apex-beat.

collapse, in poisoning with opium, chloroform, or aconite, the breathing is slowed. The extent of the respiratory movements may be limited by pleurisy, with or without effusion, emphysema, pericarditis, peritonitis, fracture of the ribs, pleurodynia, paralytic conditions (spinal disease, peripheral neuritis), ankylosis of the costal articulations in arthritis deformans. In debility and collapse the breathing is shallow. Unilateral or localized immo



bility is seen in affections which prevent the proper expansion of the lung, as phthisis, pleural adhesions, pleural effusions, pneumothorax, collapse of the lung, pneumonia, obstruction of a bronchus, etc.

Diminution of chest movements due to circulatory defects is of but slight diagnostic importance, as even in health the apex-beat may be barely perceptible.

**Excessive Movement.**—Increase in frequency of breathing is observed in dyspnoea, in emotional disturbances, in fevers, more particularly in pneumonia, where the respiration rate is increased by both the amount of lung involved and by the fever.

Increase in the extent of respiratory movements, if general, may be due to diminished abdominal breathing from abdominal inflammation or tumours, ascites, meteorism. It may be caused by paralysis of the diaphragm or diaphragmatic pleurisy, and by hysteria. Local increase may be the result of disease in the opposite lung, throwing more work than normal on the sound organ. Retraction of the intercostal spaces, when considerable, indicates inspiratory dyspnoea, and is often seen in children suffering from broncho-pneumonia or obstruction of the upper air passages. When localized, it indicates circumscribed interference with expansion, as in blocking of a bronchus, pulmonary collapse, pleuritic adhesions, etc. Bulging of the interspaces generally denotes expiratory dyspnoea, as in asthma and emphysema. It may be seen during **expiration** above the clavicles in emphysema.

**Litten's Sign.**—The diaphragm phenomenon may at times be of assistance. It is obtained by placing the patient on his back with his feet toward the light, other side-lights being removed. A movement of the intercostal spaces opposite that portion of the side which is in contact with the diaphragm may then be seen as a linear shadow at right angles to the midaxillary line, moving downward with inspiration and, less distinctly, upward with expiration. The distance traversed by the shadow may be 3 inches or more from the sixth to the ninth rib. It is produced by a sucking in of the intercostal spaces as the diaphragm descends, and its presence is proof that the lung and the diaphragm lie against the thoracic wall at that spot, and are free to move. Its absence in patients whose chest-walls are not thickly covered with muscle or fat suggests pleural effusion, pleural adhesion, pneumothorax, pneumonia.

Various abnormalities of respiration impart irregular chest movements. The subject is discussed under *Dyspnoea* (p. 125).

Increase of the movements of the chest, due to affections of the **organs of circulation**, are varied and instructive.

**Apex-Beat.**—The portion of the chest-wall which is normally moved by the ventricular contraction corresponds accurately enough for clinical purposes to the lowest and outermost portion of the organ—namely, the apex of the left ventricle. In adults this area, known as the **apex-beat**, is about 1 inch square, and is situated, when the patient is in the upright or dorsal posture, in the fifth left interspace, somewhat internal to the nipple line, or rather over 3 inches from the midsternal line. Owing to variation in the shape of the chest, the position in normal subjects may be slightly removed from that just described. In children it is commonly a space higher, and in old people a space lower, than the fifth. By turning the patient on his left side the apex-beat is displaced somewhat outward. Displacement to the right is less readily effected. Fixity of the apex-beat, in spite of change of posture, indicates adhesions or contractions in the lungs and pericardium.

Alterations in the position, force, or extent of the apex-beat are of the first importance in diagnosis. When, however, the chest is misshapen from any cause, this sign loses much of its value.

**Displacement of Apex-Beat.**—The apex-beat may be displaced in any direction, either with or without change in the force and extent of the area of movement. This may be the result of disease of the heart itself, or of the lungs, pleuræ, abdominal organs, and other structures.

1. Displacement downward and to the left is usually the result of hypertrophy and dilatation of the heart, and particularly of the left ventricle. It may reach the eighth interspace and the midaxillary line.

2. Displacement upward and to the left is, with the exception of pericardial exudation, almost always due to lesions other than those of the heart. The pericardial sac may be pulled in that direction by contracting affections of the left lung and pleural adhesions, such as fibroid phthisis, chronic pneumonia, removal of left pleural effusion, serous or purulent, with retracted lung. The heart may be pushed upward and to the left by right pleural effusions, by mediastinal tumours, or by abdominal pressure from ascites, tumours, or meteorism.

3. Displacement directly outward to the left may occur to a moderate degree from the causes just mentioned, and is often produced by enlargement of the right ventricle.

4. The heart may be pushed directly downward by aneurism of the arch of the aorta, by tumours of the upper mediastinum, or by emphysematous lungs.

5. Displacement of the apex-beat to the right may be the result of left pleural effusion, tumour of the left lung, contraction of a sclerotic right lung, with pleural adhesions. The apex may be felt as far to the right as the right nipple line. Inversion of the organ; will naturally cause displacement of the apex-beat to the right.

An unusual position of the apex-beat is sometimes observed—that is, well inside the region of superficial cardiac dulness. (See Percussion of Thorax, p. 459.) This is most probably due to pericardial effusion, but may be found in mitral incompetence. In the former condition the explanation is obviously that the dulness extending beyond the apex-beat is produced by the effusion, and not by the heart substance, which does not extend so far in that direction. In mitral incompetence the escape of blood at the beginning of the systole may prevent the sudden hardening and change of shape of the heart, which normally occurs immediately the mitral valves are closed by ventricular contraction, and before the intraventricular pressure has been raised sufficiently to permit the blood to escape into the aorta (the systolic closure time). The impulse would thus be slightly retarded, and would only reach the chest-wall when the shortening of the ventricle had raised the apex a little (Sahli).

Changes in the **force**, **extent**, and **character** of the apex-beat must be carefully noted. As already stated, diminution of the impulse at the apex is of secondary importance. A fleshy, oedematous, or emphysematous chest-wall, or the heart-stroke falling directly upon the posterior surface of a rib instead of on the soft tissues between the ribs, may be enough to abolish the apex-beat. In this case the situation of the heart's apex can usually be defined by noting the spot at which the heart's first sound is best heard with the stethoscope. Pericardial effusion or adhesion, pulmonary emphysema, and weakened ventricular contraction from dilatation, muscular degeneration of the heart, or from general debility, are the usual pathological causes of weakened or absent apex-beat.

An increase in the force of the impulse, which has a slow heaving, rather than a sudden knocking character, and which is not only displaced downward and outward, but is increased in extent, indicates hypertrophy of the left ventricle.

An increase in the force of the impulse, with a knocking or slapping rather than a heaving character, and with probably an extended, but not a displaced, apex-beat, suggests a functional disturbance. It is seen in the palpitation of emotional or mental excitement, after physical exertion, and as an effect of tobacco, tea, alcohol, etc. An increase in extent of the impulse results from retraction of the lung from the præcordial region owing to disease of the lung, and extreme thinness of the chest-wall may produce a similar effect.

**Præcordial Pulsation.**—Non-apical thoracic pulsations may be observed in affections of the heart and bloodvessels. In order to compare the time of the movement of one pulsating region of the chest with another, a matter of considerable diagnostic importance, we may amplify the movements by applying to the spots under observation indicators of some light material. These may easily be made of elongated pyramids of cotton-wool, about 3 inches in length, adhering by the base to the skin smeared with vaseline. By this means, not only the rhythm, but also the direction of the pulsation is demonstrated.

Pulsation in the first or second right interspace close to the sternum may be evidence of an aneurism of the ascending aorta. It is sometimes seen or felt when the lungs have been retracted from the front of the great vessels by disease, the vessels not being necessarily affected. Pulsation in the corresponding region on the left side is also commonly due to perceptible heaving of the vessels in systole. It sometimes occurs from dilatation of the pulmonary artery. Mitral disease is a common cause of pulsation in both these areas, the lungs being pushed aside by the dilated auricles and ventricles consequent on this valvular lesion, and the pulsation in the aorta and pulmonary artery being magnified by the ventricular enlargement. Under these conditions the closing of the semilunar valves may often be felt as a distinct **diastolic shock** in the pulmonary area. A similar diastolic shock may be felt in the pulmonary area in conditions favouring forcible closure of the aortic valve, e.g., Bright's disease or aortic aneurism.

While the pulsation from aortic aneurism occurs most fre-

quently near the second right costal cartilage, it may also be found close to the left of the sternum in the first three interspaces, also behind the sternum, which may have suffered erosion, or in the episternal notch.

Pulsations below the level of the third rib are usually due to heart affections, and above that level to vascular changes. On the right of the sternum from the third costal cartilage downward, pulsation denotes either displacement of the heart or dilatation of the right auricle, or, possibly, of the right ventricle. To the left of the sternum, pulsation below the level of the third costal cartilage denotes either hypertrophy and dilated right ventricle or retracted left lung. In the second and third interspaces the hypertrophied left auricular appendix may cause a presystolic pulsation. If the pulsation is observed above the left nipple line, and in the third or fourth interspace, it is usually due to a displaced apex-beat.

A rare form of pulsation is that communicated to the chest-wall from the heart through an empyema.

**Pulsation of the Epigastrium.**—This is not strictly thoracic, pulsations of the liver and of the epigastrium may be referred to here, as they so frequently depend upon thoracic disease. Pulsation of the liver, due to tricuspid insufficiency, causes an expansion of the whole organ with each systole, and may be observed by firmly compressing the liver between the left hand laid on the last three ribs behind, and the right hand on the right hypochondrium and costal margin in front. In the epigastrium movement may be both seen and felt, and may be systolic or post-systolic in time. If systolic it is usually produced by the heart, and may be observed in health as well as when the heart is acting forcibly, as in emotions and from exercise. It is an evidence of dilated and hypertrophied right ventricle, the result of mitral disease. Displacement of the apex-beat to the right from any of the causes mentioned above may produce epigastric pulsation, or the movements of the heart may be transmitted to the surface through the liver. If the pulsation is found to occur immediately after the apex-beat, it is dependent on the aorta, and not the heart, for its direct origin. The commonest cause is the so-called **irritable aorta**, observed in persons of a neurotic temperament, especially if they are the subjects of gastric affections or of anæmia. The aortic pulsation may be transmitted to the surface through a tumour. The pulsation may in rare instances be due

to an aneurism, in which case the movements are distinctly lateral or expansile. The subject of epigastric pulsation is further considered in the article on Abdominal Abnormalities of Shape, Movements, etc., at p. 12.

Movements of **retraction** of some portion of the chest-wall, or of the epigastrium (unconnected with respiration), are at times to be seen. Occasionally they occur in health, especially at the apex, but are more commonly the result of pericarditis. Inflammation of the serous membrane lining the pericardial sac usually causes a greater or less amount of **adhesion** between the visceral and parietal surfaces, and it may be that the pericardial sac is sufficiently inflamed to effect its adhesion to the adjacent thoracic wall and other neighbouring structures. In this case the heart's movements will be imparted to the chest-wall in the form, not only of pulsations, but also as retractions of various spots. The extent and force of these movements are often considerable, and are to be accounted for by the hypertrophy which results from the obstruction caused by adhesion, whereby greater exertion than normal on the heart's part is required in order to maintain efficient circulation. If valvular disease should accompany the pericarditis, as is often the case, the hypertrophy will, of course, be even better marked, and the consequent impulses and retractions more accentuated. Dilatation of the heart's cavities ensues from inflammatory and degenerative changes in the myocardium, with the result that the movements become feebler and less heaving in character. The following physical signs of adherent pericardium may be mentioned in this connection: Systolic retraction of the lower end of the sternum and of the epigastrium; systolic retraction at the heart's apex; systolic retraction to the left of the sternum, below the third rib; systolic retraction of the tenth and eleventh interspaces below the left scapula (**Broadbent's sign**); immobility of the apex-beat on change of posture; a palpable diastolic shock at the apex; inspiratory swelling of the jugular veins (**Kussmaul's sign**); diastolic collapse of the jugular veins; weakening or disappearance of the pulse during inspiration (**pulsus paradoxus**). An apical systolic murmur, even in the absence of valvular disease, may accompany adherent pericardium, owing to dilatation of the left ventricle. Pain in the præcordial region, dyspnœa, palpitation, cough, vomiting, and other symptoms of cardiac mischief may be present, while enlargement of the liver, ascites, and

œdema, especially in young subjects, are common accompaniments. It must be remembered that many cases of this affection are latent, presenting insufficient physical signs to enable one to recognize the condition.

Vibratory movements of the chest, perceived by palpation, are of considerable diagnostic importance. They originate in different regions and manners.

**Vocal Fremitus.**—A very instructive palpable vibration is that known as **vocal fremitus**, originating at the vocal cords during the act of phonation. The vibratory movements are transmitted thence to the surface of the chest through the columns of air contained in the trachea and bronchi, and through the intervening tissues, to the observer's hand resting upon a portion of the chest-wall with which the lung is in contact. In order that vocal fremitus be distinctly perceived, it must, in the first place, be produced with sufficient force and amplitude of vibration. This is best effected when the vibrations are comparatively infrequent—that is, when the voice is low pitched and when the sound produced is fairly loud. In the second place, the vibrations thus efficiently evoked must pass through good conductors of sound in order that we may perceive them distinctly. The uninterrupted air columns contained in the trachea and bronchi conduct vibrations excellently; the spongy lung tissue consisting largely of a mass of small collections of air separated from each other by membranous septa, forms a less efficient conductor. The chest-wall, if thin, conducts moderately well. These tissues may be altered so as to improve or to impair their conductivity. Thus the air columns in the lung may be interrupted, and the vocal resonance is diminished in consequence. This may occur exceptionally in some cases of inflammation of the lung, where the bronchi become blocked with inflammatory exudation (**massive pneumonia**). The interposition of fluid or gas in the pleural cavity diminishes the conductivity of the thorax. In pneumothorax and in pleural effusion, serous, purulent, or sanguineous, vocal fremitus is therefore diminished. An increase in thickness of the chest-wall (*e.g.*, œdema, fat) also lessens the fremitus. The contrary condition of increased conductivity is found where the imperfectly conducting spongy lung tissue is replaced by infiltration (provided the bronchi remain patent), by collapsed vesicles, by tumours, by cavities, or by enlarged bronchi. Vocal fremitus, therefore, is increased in pneumonia,

collapse of the lung from any cause, phthisis, and occasionally by bronchiectasis.

**Friction Fremitus.**—The vibrations caused by two roughened surfaces rubbing together can usually be best perceived by the stethoscope (see p. 421). They may at times be felt by the hand placed over the affected part as a trembling or grating movement synchronous with respiration or with the heart's beat. In the former case the fremitus is commonly a sign of pleurisy, but may also be found over the lower thoracic regions in peritonitis affecting the liver or spleen. When coinciding with the heart's rhythm it indicates pericarditis, and is to be distinguished from the vibratory movement or **thrill** caused by valvular lesions. This is a more useful sign of disease, and is most commonly due to endocardial affections causing obstruction of one of the orifices (especially the mitral), and, less frequently, to insufficiency of the valve. When resulting from mitral obstruction, it occurs as a fairly rough vibration felt at the apex-beat immediately before the impulse of the heart. It may be felt at the base of the heart in aortic stenosis, when it is systolic in time. Mitral and aortic incompetence may on rare occasions give rise to thrill, and a systolic thrill may be observed in aneurism. As a rule, a murmur is to be heard in all cases where thrill is felt; indeed, the vibrations producing a thrill are the identical movements causing the audible murmur, and, in order that they may be palpable, they must have a sufficient force, and should be comparatively infrequent. Occasionally, and especially in mitral stenosis, a thrill is felt when the murmur is inaudible. This is doubtless due to the condition just mentioned—the infrequency of the vibrations.

**Bronchial Fremitus.**—In bronchitis, bronchiectasis, phthisis, asthma, etc., the movements of air passing through fluids and catarrhal and constricted passages or cavities cause a palpable vibratory movement of the chest-wall, the bronchial fremitus. It is best observed in children, but is of trifling diagnostic value, as the condition is more advantageously investigated by the stethoscope.

### **THRILL (*Frémissement cataire*).**

A vibratory movement, palpable on the surface of the chest in the vicinity of the heart, due to a valvular lesion (usually obstructive) of that organ. (See Thorax, Shape, etc., p. 478.)



**THROAT, Painful Conditions of.**

Pain in the fauces is a very frequently occurring symptom. Inflammatory affections of the throat are always painful, a burning or smarting feeling being the rule. The lymphatic tissues and mucous membrane of this region are especially exposed to the attacks of micro-organisms and their toxins, the resulting affections being acute and chronic pharyngitis, follicular tonsillitis (often rheumatic), phlegmonous or suppurative tonsillitis (quinsy), scarlet fever, mumps, chronic tonsillitis (which is often associated with adenoids), diphtheria, ulcers of the fauces (follicular, cancerous, tubercular, or syphilitic).

Injury to the tissues is a not infrequent cause of pain in the throat. Thus one finds œdema, inflammation, and ulceration of the throat resulting from the swallowing of irritant poisons, foreign bodies, excessively hot food or fluids, and from scalding by steam.

The conditions giving rise to sore throat, and their chief distinguishing features, are compared in the table on p. 476.

**THYROID GLAND, Enlargement of.**

Hypertrophy of this organ is of more surgical than medical interest. Only one general disease need be mentioned in which this symptom occurs—namely, exophthalmic goitre (Graves' disease, Basedow's disease). It can usually be recognized with ease by the presence of one or more of the accompanying symptoms. The most important of these are excited action of the heart, exophthalmos, and tremor.

**TIME OF HEART MURMURS.**

It is essential to ascertain the exact period in the cardiac cycle at which the murmur takes place in order to discover the nature of the lesion causing it. (For an account of the methods of timing the murmur, and of the significance of their occurrence in the respective periods, see p. 436 *et seq.*)

**TONGUE, Abnormalities of.**

As an index of disease of wider distribution than the buccal cavity, the tongue deserves careful attention.

Local affections are of surgical rather than of medical interest in most cases. They include: Glossitis, ulceration from local

TABLE OF PAINFUL THROAT AFFECTIONS

Symptoms.	Acute Pharyngitis.	Chronic Pharyngitis.	Secondary Syphilis.	Follicular Tonsillitis.	Phlegmonous Tonsillitis.	Diphtheria.	Scarlet Fever.
<b>Pain</b> ..	Smarting or burning	Slight	Very slight	Considerable	Severe	Moderate	Considerable
<b>Fever</b> ..	Moderate	Little or none	Slight	Considerable	High fever	Moderate	Considerable
<b>Seat of the lesion and its nature</b>	The fauces are generally injected, the mucous membrane thickened. Streptococci and other non-specific organisms may be found	Mucous membrane of the fauces thickened generally; the follicles enlarged and perhaps ulcerated	On posterior wall of pharynx, pillars of fauces, soft palate, uvula, and tonsils. Shallow ulceration ('snail-tracks')	Tonsils inflamed and moderately swollen. Follicles inflamed, with plugs of mucus or false membrane, which peels off, leaving surface of tonsil uninjured. No specific micro-organisms found	Tonsils much enlarged, large, red, tense. Often suppurate	Fauces injected. Membrane seen on soft palate, pillars of fauces, and tonsils; on removal a raw and bleeding surface left. Löffler's bacillus is found	The fauces generally are injected, dusky and swollen
<b>Other prominent symptoms</b>	Moderate constitutional disturbance	Persistent cough or clearing of throat. Mouth breathing. Imperfect bodily development	Signs of syphilis elsewhere	General pains over body and constitutional disturbance	Constitutional disturbance often severe	Constitutional disturbance severe. Cervical glands considerably enlarged	Pulse rate unduly rapid. The rash and period of its invasion are characteristic

irritation, injury from corrosive poisons, gumma, cancer, smoker's patch, lacerations, or scars of healed wounds (possibly the result of epilepsy), etc.

In regard to medical affections, the tongue should be examined as to its coating, colour, tone, size, and movements.

**Coating.**—A thin whitish fur is found in health in many individuals; it occurs in pyrexia from any cause, in digestive disorders, and among smokers. The fur may be thick and yellowish, or brownish in more severe gastric disorders, fevers, or among those who abuse alcohol and tobacco. If the coating is thinner or absent at the tip, sides, and some distance up the middle line of the tongue, while the rest of the tongue is well coated, the condition suggests typhoid fever. A dry fur, brown or dark in colour, is characteristic of prostration and fever; it is found in the typhoid state from any severe disease. A white fur, through which the bright red papillæ project, is known as the **strawberry tongue**, and is seen in fevers, and more especially in the early stages of scarlet fever. The same term is sometimes applied to a red tongue without fur, with projecting papillæ, which is also seen in scarlet fever, though less characteristic of the disease. Paralysis of one side of the face usually causes that side of the tongue to be more furred than the sound side, owing to the diminished friction of the parts. Carious teeth, tonsillitis, and other inflammations of the mouth give rise to a thick coating, which may be partial in its distribution. The fur may be coloured by drugs (black by bismuth, iron, or charcoal; white by carbolic acid) or by food.

**Colour.**—The tongue is pale in anæmic conditions—*e.g.*, chlorosis, chronic Bright's disease, etc.; red in tubercular disease, in the early stages of scarlet fever, in the typhoid state (when it is also dry); bluish in cyanosis; dark in Addison's disease; yellow in jaundice.

**Tone.**—A firm condition of the tongue suggests bodily vigour. On the contrary, a flabby soft tongue, on which the impressions of the teeth are plainly marked, indicates a debilitated and often anæmic state.

**Size.**—Increase in size of the tongue is usually the result of inflammation (glossitis); it may also be seen in cretinism and myxœdema. A slight enlargement is seen in the flabby tongue of anæmia. Tumours of the tongue give rise to enlargement, often of one side. Decrease in its size may be observed in conditions

of prostration—*e.g.*, typhus fever, advanced typhoid fever, profuse hæmorrhage, cholera. Lesions of the hypoglossal nerves or of their nuclei will cause marked atrophy of the tongue associated with paralysis (see below). In this condition the surface of the tongue is wrinkled, and either the whole organ or a portion of it is shrunken.

**Tremor of the Tongue.**—A familiar instance is the rather coarse tremor of acute and chronic alcoholism. The trembling seen in



FIG. 69.—HEMIATROPHY OF TONGUE.

First noticed some years ago, immediately after scarlet fever.

the tongue of neurotic persons and of conditions of prostration is of a similar character. Fibrillary twitching is seen in atrophic conditions—*e.g.*, in bulbar paralysis. It also forms an important sign in general paralysis of the insane, and in multiple sclerosis.

**Paralysis of the Tongue.**—Unilateral paralysis is shown by the deviation of the tongue toward the affected side when it is protruded; the paralysed side is flatter than the sound half, and if the

lesion is nuclear or infranuclear (see Trophic Disturbances, p. 487, and Decreased Movements, p. 225), the portion of the organ involved will be wrinkled and shrunken (see Fig. 69). Bilateral lingual paralysis with atrophy is, as a rule, a sign of bulbar paralysis. This affection may be part of the more widely distributed analogous affection of the spinal cord (progressive muscular atrophy), or may be rarely seen in advanced cases of locomotor ataxia, or may exist as an independent degenerative process affecting the bulbar motor nuclei. Unilateral lingual paralysis, with atrophy, may be due to the same causes as the bilateral affection; it may be the result of hæmorrhage or embolism affecting the hypoglossal nucleus, or of a lesion of the nerve.

Unilateral paralysis without atrophy is the effect of a lesion interrupting the motor tract, from the cortex to the hypoglossal nucleus, but not including the latter; the usual position for this lesion is the internal capsule. A bilateral lingual paralysis without atrophy is produced by a two-sided lesion above the nuclei—the so-called **pseudo-bulbar paralysis**.

Ulcers of the tongue are usually local affections. Of diagnostic interest are the lacerations due to the tongue being bitten in an epileptic fit, and the small ulcer of the frenum caused by the lower incisors in whooping-cough.

### TONIC SPASM.

A single, continuous, involuntary contraction of certain muscles or groups of muscles, producing a stiffness or rigidity of the limb or part affected. The conditions under which this symptom may be observed are described in the article on Increased Muscular Contractility, at p. 249.

### TOPHI (*L. tophus*, limestone).

Deposits of sodium urate, occurring in individuals who are the subjects of chronic gout. They form chalk-like masses around the joints, in the borders of the ear, in the eyelid, etc.

### TORTUOUS ARTERIES.

As a rule, tortuosity of the arteries is an evidence of senility, or of premature degeneration of the arterial walls. It may, however, be observed in the temporal artery in cases which are apparently free from arterio-sclerosis or from senile changes.

**TRACHEAL TUGGING (Oliver's Sign).**

The left bronchus passes under the arch of the aorta (see Fig. 21, p. 108), and may, if dilated, cause the bronchus to be depressed with each pulsation. This movement of the bronchus is transmitted to the trachea, and may be observed as a visible or palpable downward thrust of the larynx, synchronous with the heart-beat.

In order to demonstrate the movement in cases where it is not clearly visible, such obvious cases being rare, the observer stands behind the seated patient, who retracts his head somewhat and keeps his mouth closed; the two forefingers, placed on the sides of the cricoid cartilages, draw upward and keep tense the trachea; with each pulsation a *tug* or downward traction of the trachea is felt. Instead of standing behind the patient, some observers prefer to support the larynx with the finger and thumb of one hand from the front.

While the sign is strong evidence in favour of aneurism, it is not pathognomonic, as a mere dilatation of the aorta may be sufficient to produce it, or a pulsating sarcomatous growth in the mediastinum may be the cause. It is even seen at times in perfectly healthy, strongly-acting hearts.

**TRANSVERSELY GROOVED CHEST (Harrison's Sulcus).**

Often occurs in association with the rachitic chest and with the pigeon-breast, or may occur in otherwise normal-shaped chests. It consists in a groove commencing at the lower end of the sternum, passing out and slightly downward toward the infra-axillary region; it may occur as a simple eversion of the costal margins (see p. 467).

**TRAUBE'S SEMILUNAR SPACE.**

That portion of the thoracic surface bounded below by the left costal margin from the ensiform cartilage to the tenth rib; above by the dulness arising from the left lobe of the liver and the heart, and by the pulmonary resonance; to the outer side by the splenic dulness. This space is tympanitic on percussion, owing to its contact with the stomach, and is of interest to the diagnostician because its dimensions, as measured by the tympanitic note, are constantly diminished by disease in the adjacent structures. The most characteristic change is the encroachment

in its upper regions of the dulness due to left pleural effusions; the reflection of the pleura being extensible, permits of the descent of fluid in the pleural cavity to a lower level than that of the normal pleural membrane, so that in some cases of left pleural effusion there is very little clear area left in the semilunar space. Enlargements of the heart, spleen, and liver likewise encroach upon the space (see p. 453 and Fig. 65).

### TREATMENT, Result of.

As an aid to the formation of a diagnosis, the results obtained by treatment may occasionally be of service. The opportunities for availing one's self of this source of information are restricted, as the existence of a *specific* for a certain class of affection can be instanced but rarely.

It is mainly diseases of syphilitic origin that this line of evidence can be utilized. The disappearance of symptoms arising from a cerebral tumour under antisyphilitic treatment is a gratifying and not infrequent occurrence, which forms the strongest possible argument in favour of a diagnosis of syphilis in such a case. A similar course in many of the lesions due to this poison (*e.g.*, primary sore, iritis, secondary and tertiary throat affections, gumma of the tongue, bone disease, skin affections, etc.), is equally instructive.

The effect of iron in certain heart affections may aid the diagnosis. A murmur indicative of mitral incompetence is often found in an anæmic subject, whose heart can be shown to have increased beyond the normal size. Under treatment by iron the murmur may disappear, and the heart may return to the normal dimensions, showing that the lesion was probably one of **relative incompetence**, depending on degenerative changes in the heart muscle leading to dilatation of its cavities, the orifices being otherwise intact.

The ready control of pyrexial attacks by quinine suggests malaria as a cause, and the failure of iron to cure anæmia in cases that are apparently independent of any primary cause raises the suspicion of pernicious anæmia. In both these instances, however, we have in the blood examination a means of investigation beside which the deductions from the result of treatment seem insignificant.

An affection which is commoner than is generally supposed is

the pyelitis seen mainly in female children, and due to the presence of the *Bacillus coli communis* in the urine. (See *Urine, Abnormalities*, p. 513.) Here the urine is acid. Holt and John Thompson have shown that by causing the urine to become alkaline (by the administration of alkaline drugs) the symptoms promptly disappear, to reappear if the drug be prematurely discontinued; this reaction of the disease to an alkaline drug may be regarded as a point of diagnostic value.

A severe attack of articular pains with elevation of temperature, which yields readily to salicylates, is probably acute rheumatism.

### **TREMOR** (*L. tremere*, to shake).

Rapidly recurring contractions of small groups of muscles, sufficiently forcible to move slightly their insertions, are observed in many morbid conditions. The finest variety of these clonic spasms are the **fibrillary twitchings** seen in progressive muscular atrophy and other atrophic and cachectic conditions. The amplitude and force of the contractions vary in different affections; there is only a difference in degree, but not in kind, between fibrillary twitchings, tremor, and convulsive spasms. The varieties of tremor and the diseased conditions in which it may be observed are discussed at p. 253 *et seq.*

### **TRICHTERBRUST** (Funnel-Chest).

A chest presenting a depression or groove at the lower end of the sternum. It may be congenital or may arise in childhood, or may even be produced in adult life by pressure long continued on the region. (See *Thorax, Shape*, etc., p. 468.)

### **TRICUSPID AREA.**

That portion of the chest-wall occupied by the lower end of the sternum and the parts immediately adjacent on either side. At this region sounds generated at the tricuspid orifice can be heard best (see pp. 435, 437).

### **TRISMUS** (Lock-jaw).

One of the chief symptoms of tetanus is an involuntary spasmodic contraction of the muscles of mastication, whereby the patient is prevented from separating the jaws. This group of muscles is usually the first to be affected, and is so characteristic



of the disease that the name lock-jaw is often applied to the affection rather than to the symptom. A similar spasticity of this group of muscles may at times be seen in cases of strychnine-poisoning, should the patient survive. In this condition the jaws are affected later in the course of the complaint and in severe cases.

## TROPHIC DISTURBANCES.

**Hypertrophy:** of excessive use; of excessive nutriment; as a result of various morbid processes.

**Atrophy:** of excessive destructive metabolism; of insufficient nutriment; of disuse; of post-inflammatory changes, of disease of the nervous system; of disease of the muscles

Infantile paralysis—Progressive spinal muscular atrophy—Bulbar paralysis—Amyotrophic lateral sclerosis—Lesions of motor nerves—Syringomyelia—Landry's paralysis—Progressive neural muscular atrophy.

Disease of the muscles: pseudo-hypertrophic muscular paralysis, Erb's juvenile form of progressive muscular dystrophy; the facio-scapulo-humeral form—Dystrophy.

Superficial tropho-neuroses: Paget's glossy skin; erythema; vasomotor disturbances (lividity, cyanosis, gangrene); herpes zoster; pemphigus; ichthyosis; pigmentary changes; changes in the hair, nails, epidermis—Perforating ulcer—Bedsores—Morvan's disease—Affections of the teeth

Neurotrophic affections of the bones and joints: Charcot's joint, tabetic foot—Trophic lesions in locomotor ataxia, syringomyelia, infantile paralysis, and peripheral nerve affections.

The tissues of the body in disease may differ from those in health in many respects. The changes observed result from disturbance of the nervous organs controlling nutrition, from defective nutriment, and from local disease. In examining the patient we have to ascertain if the organs and tissues of his body are—(1) increased in bulk (**hypertrophy**); (2) decreased (**atrophy**); or (3) altered in texture (**dystrophy**).

1. **Hypertrophy** of an organ or of any structure in the body is the result of—

(a) Excessive use of the part, as may be seen in the muscles of the limbs, the heart, etc.

(b) An excessive amount of nutriment supplied to the body, causing an accumulation of fat.

(c) Various morbid processes (apart from excessive nourishment). These include—(a) degenerative changes—*e.g.*, the muscles (sec

p. 491), kidneys, etc.; ( $\beta$ ) inflammatory changes—*e.g.*, subcutaneous inflammatory swellings, enlargements of the tonsils, salivary glands, etc.; ( $\gamma$ ) gaseous distension of hollow organs or tissues—*e.g.*, meteorism, pseudocystitis, surgical emphysema; ( $\delta$ ) dropsical or other effusions into serous cavities and subcutaneous tissues; ( $\epsilon$ ) new growths.

2. **Atrophy** of tissues or organs occurs in the following conditions:

(a) **Emaciation from Excessive Destructive Metabolism**, as in wasting diseases, and from **insufficient or unsuitable nutriment**. The two acts of metabolism, construction and destruction, should, under normal circumstances, keep pace together. In many morbid states the destructive or **katabolic** metabolism outstrips the constructive or **anabolic**, resulting in a loss of tissue. This may be accurately observed by noting carefully the body-weight at regular intervals, due allowance being made for dropsical fluids when they are present. Emaciation results most frequently from disease of the alimentary tract; very frequently and rapidly from acute pyrexial diseases; from chronic diseases with fevers—*e.g.*, phthisis, suppuration; from diabetes mellitus; from all varieties of malignant growths. When this emaciation is accompanied by general loss of vitality and loss of function it is termed **marasmus** or **cachexia**.

(b) **Disuse of the Atrophied Parts**—*e.g.*, atrophy of muscles after the removal of splints or other impediments to movement; senile changes in disused organs (breasts, uterus, etc.) or tissues (skin and subcutaneous fat). Atrophy of disuse must be carefully distinguished, in the case of muscles, from that due to disease of the nervous system. There may be a combination of both factors, as in sciatica. Here the muscular wasting is partly due to the interference with nervous trophic control, owing to neuritis, and also to disuse of the limb. The familiar atrophy of disused tissues in old age needs no further comment.

(c) Changes occurring in tissues after **Inflammation or Injury**. Orchitis, whether due to direct injury or to metastatic inflammation from mumps, may be followed by atrophy of the organ. Atrophy of the mucous membrane of the stomach is found in one form of chronic gastritis. Atrophy and degeneration are familiar results of inflammation in nervous tissues; and the *lineæ albicantes* (see p. 188), sclerosed and atrophied lines in the skin which had been subjected to undue strain, are produced by tumours, the

pregnant uterus, enlarged mammae, or excessive fatty deposit on the thighs and elsewhere.

(d) Muscular wasting consequent on **Disease of the Nervous System**. A trophic or nutritive influence is exercised by, or by means of, the nerves supplied to muscles and other tissues. When a motor nerve is cut, the portion of it which is peripheral to the injury becomes wasted (Wallerian degeneration). The wasting is not confined to the nerve, but is continued to its attached muscle fibres, which undergo a much more rapid atrophy than could be explained by simple disuse. A similar wasting is seen in the testicle after section of the spermatic cord: and in the disease of the joints known as **Charcot's joint** (see p. 494) the trophic changes are to be accounted for by disease of the nerves supplying them.

In the case of muscles this trophic influence proceeds from the cells, of which the efferent nerve fibres are axis-cylinder processes (**axons**)—namely, the so-called ganglion cells in the anterior cornua grey matter of the spinal cord (see Fig. 28, p. 204). Injury or disease affecting the ganglion cell or any portion of its axon, in such a manner as to interfere with the passage of nerve impulses to the muscle, causes atrophy of all the nerve tract peripheral to the seat of the injury. It also causes atrophy of the muscle fibres in which the damaged nerve fibres end. This portion of nerve tract (ganglion cell to peripheral termination) is spoken of as the **lower segment of the motor tract**. Diseases of the lower segment are, therefore, characterized by muscular atrophy. On the other hand, lesions of the brain or spinal cord, not involving the ganglion cells in the anterior horns of the cord, have no deleterious effect on the nutrition or **tone** of the muscles.

Muscular atrophy of nervous origin is, therefore, found in the following condition, which are also instances of diminished muscular contractility (see p. 213):

(i.) **Infantile Paralysis (Acute Anterior Poliomyelitis)**, an acute inflammatory affection of the anterior cornua of the spinal grey matter, probably of infective origin. It occurs in early childhood, with sudden onset, accompanied by constitutional disturbance and fever. A partial recovery usually results, followed by extreme wasting and defective growth of the affected region.

(ii.) **Progressive Spinal Muscular Atrophy**, a chronic degeneration, rather than an inflammation, primarily affecting the anterior

horns and the ganglion cells contained therein. The respective nerve roots, nerve trunks, and attached muscle fibres are involved in the degeneration. The cervical portion of the cord is commonly first affected, the small muscles of the thumb and little finger or the shoulder muscles often being the earliest to show the wasting. The disease usually commences in young adults, and is of a slowly progressive character.

(iii.) **Progressive Bulbar Paralysis** is a similar morbid process affecting the motor nuclei in the medulla and pons, producing



FIG. 70.—PROGRESSIVE MUSCULAR ATROPHY.

Muscles of right shoulder are wasted, as is also the lower half of the left pectoralis major.

wasting and paralysis of the muscles supplied by the nerves arising therefrom.

(iv.) **Amyotrophic Lateral Sclerosis (Charcot's Disease)** is the term applied to the affections just referred to under (ii.) and (iii.), to which a degenerative lesion of the lateral columns is added. The result is that, in addition to the wasting and paralysis of those diseases, there occur a spasticity and an increase of the deep reflexes.

(v.) **Lesions of the Motor Nerves.**—The causes are: (a) Neuritis, due to toxic states—*e.g.*, rheumatism, diphtheria, alcoholism,

lead and arsenical poisoning, diabetes, etc. : to injury to the nerve or to involvement in inflammation of neighbouring tissues : to exposure to cold. ( $\beta$ ) Tumours of various descriptions occurring in or near nerves. ( $\gamma$ ) Pressure by splints, crutches, etc. ( $\delta$ ) Wounds of nerves—*e.g.*, cuts, stabs, gun-shots, etc. (see p. 214 *et seq.*).

(vi.) **Syringomyelia**.—A wasting of the muscles of the hand similar to that of progressive muscular atrophy is usual. Characteristic sensory and trophic disturbances are present, and are described at pp. 234, 304 and 494.

(vii.) **Landry's Paralysis**.—An acute atrophic affection which causes paralysis, commencing in the feet and ascending rapidly. It often ends fatally before atrophy has time to appear.

(viii.) **Progressive Neural Muscular Atrophy**.—A chronic form of peripheral neuritis occurring in childhood, and affecting, in the first place, the peronei and other muscles of the foot. Later the remaining leg muscles and those of the hands and arms are involved. Pain and anaesthesia are present.

(*c*) **Disease of the Muscles**.—A well-defined group of affections of the muscles may be recognized, the distinguishing features of which are atrophy of the muscular fibres, with or without hypertrophy of the connective tissue. They are now regarded as diseases of the muscles themselves, without dependence upon any central or peripheral nerve lesion. They have been described by Erb as 'progressive muscular dystrophy,' and by some observers are regarded as forms of one disease. Three chief varieties are recognized :

- a. Pseudo-hypertrophic muscular paralysis.
- $\beta$ . Erb's juvenile form of progressive muscular dystrophy.
- $\gamma$ . The facio-scapulo-humeral form.

In addition, intermediate varieties have been observed which do not conform sufficiently to any type to enable one to add them to either of the groups, or to form them into a class for themselves.

The following are the chief features which these groups possess in common: Heredity is an important factor. The tendency to degeneration seems to be congenital and inherited, though the morbid process may not be observed for months or years after birth. The muscles selected by the disease are attacked in a different order and grouping from those affected by disease of the nervous system (see tables, pp. 234 to 238).

Babinski and Onanoff explain the selection by showing that the muscles involved are those earliest developed in the fœtus.

3. **Dystrophy, or Perverted Nutrition.**—Structural change in the tissues occurs in practically all local and general diseases, including many of the so-called functional affections. In this place, however, we consider only those changes produced by interference with the normal trophic control exercised by nervous tissues over the state of nutrition of the remaining tissues and organs of the body. The most important of the trophic changes so brought about may be grouped into two classes: (a) Trophic affections of the **skin** and its appendages; (b) of the **bones and joints**. The muscular dystrophies have just been referred to in discussing atrophy (p. 491).

(a) **Superficial Trophoneuroses:** (i.) **Paget's 'glossy skin,'** best seen in the fingers, which become tapered to the tip, with transversely and longitudinally-curved nails. The skin becomes thin, smooth, shining, dry, red, burning and painful, often due to inflammation of, or injury to, a peripheral nerve. (ii.) **Erythema** may arise under similar circumstances. It may have the appearance of chilblains. (iii.) Vaso-motor disturbances may give rise to **lividity, cyanosis,** or even **gangrene,** as seen in Raynaud's disease (see p. 48). (iv.) **Herpes zoster (zona),** a papular eruption, becoming vesicular and pustular, following the course of a superficial nerve on the trunk, limbs, or face. An inflammation or hæmorrhage has been observed in the posterior root ganglia and in the Gasserian ganglion. (v.) **Pemphigus** may form, in consequence of neuritis, along the course of a nerve, also in central nervous diseases, as syringomyelia or general paresis. (vi.) **Ichthyosis and pigmentary changes** have been attributed to neuritis. (vii.) **Hair** may become white in parts, may hypertrophy, grow brittle, or fall out. (viii.) **Nails** may be shed or become grooved transversely or longitudinally. (ix.) **Epidermal callosities and corns** sometimes owe their origin to nerve affections. The corn may give rise to a **perforating ulcer,** which may be painful, but more frequently is painless; perhaps leading by a sinus to carious bone in the foot. 'Perforating ulcer occurs in locomotor ataxia, Friedreich's disease, general paralysis of the insane, compression of the nerves or spinal cord by tumours, in peripheral nerve degeneration following neuritis, due to alcohol, diabetes, or leprosy; also after injuries of nerves or spinal cord, and in spina-bifida, in which the peripheral nerves were implicated

before quitting the spinal canal. In all these various diseases and injuries a common factor, peripheral nerve degeneration, is the condition which, if it do not actively excite the skin lesion



FIG. 71.—CRETIN CHILD, AGED THIRTEEN; ADULT WOMAN SUFFERING FROM MYXEDEMA; NORMAL CHILD, AGED THIRTEEN.

that ultimately leads to perforating ulcer, passively permits it through deficient innervation' (Hopkins). (x.) **Bedsore** (de-

cubitus). An undue facility in the production of gangrene of the skin and subjacent tissues by pressure is observed in diseases of the nervous system, especially in extensive disease or injury of the spinal cord, and more particularly of its grey matter. This must be distinguished from gangrene due to obstruction of the circulation. (xi.) **Morvan's disease**, an affection in which painless whitlows appear on the fingers, causing deep ulceration, and even necrosis of the terminal phalanges, together with œdema, bullæ, and injury to the nails. It is found in cases of syringomyelia. (xii.) The **teeth** may become carious very rapidly, or may loosen and fall out painlessly, as in locomotor ataxia.

(b) **Neurotrophic Affections of the Bones and Joints.**—The occurrence of these conditions is practically pathognomonic of **tabes dorsalis** or **syringomyelia**. The arthro- and osteopathies occur in about 3 to 4 per cent. of the observed cases of tabes, and in perhaps 20 per cent. of the much rarer disease syringomyelia. The same lesions may, indeed, be found in some cases of **general paresis**, but only in those instances where general symptoms of tabes dorsalis are present. It must be remembered that general paralysis and tabes have many features in common, and are probably nearly related, if not identical, in their pathology. In **leprosy** similar bone and joint lesions have on rare occasions been observed, and are due to the accompanying peripheral nerve lesion. The rarity of its occurrence in a rare disease deprives it of any importance from a diagnostic point of view.

The bone lesion is a rarefying osteitis. There is a loss of inorganic constituents, which, instead of constituting 67 per cent., as in normal bone, only amount to 30 to 40 per cent., the proportion of organic material, on the contrary, being about doubled. This renders the bone quite fragile, and it is frequently broken by such slight force as merely turning in bed. While the limb may be the seat of neuralgic pains, a striking feature in the lesion is the absence of pain on movement of the fragments of broken bone in the limb. The fracture heals readily, an abundant callus being thrown out, but its subsequent absorption may result in a fresh fracture.

The joint affection is to some extent a modification of the condition just described, the ends of the bones entering into the joint being the seat of a similar rarefying osteitis. At the same



time a tendency to overgrowth is observed, as exostoses, with ossification of tendinous attachments, and even of synovial fringes. Fractures into the joint, spontaneous, or the result of slight injury, occur, giving finally the characteristic **Charcot's joint**. The clinical aspect of the arthropathy is as follows: the joint in the earliest stage often suddenly become distended with fluid, which may infiltrate the surrounding tissues, causing a solid œdema. The skin becomes white, shining, and stretched; crepitus or loose fragments may be felt; the limb is abnormally mobile at the joint, and is not painful on manipulation. The effusion then tends to subside, and the osseous structures may undergo absorption—*e.g.*, the head of the femur may disappear. At the same time osteophytes are formed in greater or less abundance. It is usually noticed that in the ball-and-socket joints, such as the hip and shoulder, absorption is more active and osseous outgrowths are less abundant, than in the hinge joints, such as the knee and elbow. The final result is total destruction of the joint, with abnormal mobility, and perhaps dislocation. On the other hand, the bony outgrowths may, less frequently, cause osseous ankylosis. An extreme degree of destruction is sometimes seen in the so-called **tabetic foot**, in which, owing partly to absorption of bone and partly to dislocation of the metatarsus under or over the tarsus, the toes seem as if they were directly articulated on to the tarsus. This must be distinguished from the necrotic and septic condition of the foot, the ultimate result of a perforating ulcer referred to above.

In comparing these lesions as occurring in locomotor ataxia and syringomyelia, the following points of distinction may be noticed: in **tabes** the lower limb is the seat of the lesion in 75 per cent. of the cases, and the knee twice as often as the hip; rarely in the smaller joints except in the case of the tabetic foot. There may be more than one joint affected, in which case the lesion is symmetrical. If the upper limb is affected, it is in a late stage of the disease, when the cervical portion of the cord becomes involved. In **syringomyelia** a larger proportion of the cases show arthropathies, and, as might be anticipated from the commoner situation of the spinal lesion, the arm is much more frequently affected than the leg. The arthropathies are often multiple, but are generally asymmetrical. There is a considerable tendency to necrosis.

In **infantile paralysis** an apparent dystrophy of the bones may be observed, but it is merely deficient growth due to the want of the stimulus of the muscular contractions.

Trophic changes in the bones and joints may occur from lesions in the **peripheral nerves** (apart from leprosy), but are rare and of slight clinical importance. Similarly, **cerebral lesions** have been reported as causing arthropathies, but the occurrences are rare and without characteristic features.

**Summary.**—Diseased tissues may vary from those in health in three main directions—hypertrophy, atrophy, and dys.rophy.

Hypertrophy is the result of overuse, overnutrition, and a variety of diseased conditions (degenerations, inflammations, gaseous distension, fluid effusions, new growths).

Atrophy is due to (a) excessive destructive metabolism; (b) insufficient or unsuitable nutriment; (c) disuse; (d) post-inflammatory changes; (e) disease of the nervous system; (f) disease of the muscles.

In atrophy due to disease of the nervous system the lesion involves the peripheral neurons of the motor tract, and is exemplified in the following diseases: infantile paralysis, progressive spinal muscular atrophy, bulbar paralysis, amyotrophic lateral sclerosis, lesions of the nerves (neuritis, tumours, injury), Landry's paralysis, syringomyelia.

Atrophy due to disease of the muscles is seen in three main forms—pseudo-hypertrophic muscular paralysis, Erb's juvenile form of progressive muscular dystrophy, and the facio-scapulo-humeral form.

Dystrophy, or perverted nutrition, of nervous origin, is observed as superficial trophoneurosis, and neurotrophic affections of the bones and joints. The muscular dystrophies have been referred to in speaking of atrophy.

The superficial dystrophies include the following: Paget's 'glossy skin,' erythema, lividity, cyanosis, gangrene, herpes zoster, pemphigus, pigmentary changes, ichthyosis, changes in the hair and nails, epidermal callosities and corns, perforating ulcer, bedsores, Morvan's disease, carious or loosened teeth.

Neurotrophic affections of the bones and joints occur in tabes dorsalis, syringomyelia, general paralysis of the insane, leprosy. A rarefying osteitis giving rise to fractures and an overgrowth of bone round the joints are the chief morbid processes. Charcot's joint and the tabetic foot are two well-known examples.

**TROUSSEAU'S SIGN.**

In cases of tetany, pressure over the nerves or arteries of the affected limb will bring on a recurrence of the spasm, provided the attack has not completely passed over.

**TUBE-CASTS (Renal Casts).**

The varieties of tube-casts and their clinical significance are discussed in the article on the Abnormalities of the Urine (p. 508).

**TUBERCULOSIS (Tests for).**

See Calmette's reaction (p. 97); Moro's test (p. 202); Von Pirquet's reaction (p. 562).

**TUBULAR BREATHING.**

On listening over a patch of consolidated lung, and especially in pneumonia, the breath-sounds have usually a characteristic clear, blowing, almost whistling quality. While this is doubtless in some measure due to the highly-conducting character of the consolidation, it is largely the result of added qualities locally produced in the affected region (see p. 412).

**TYMPANITES** (Gr. *τύμπανον*, a drum).

A distension of the abdomen by gas (see p. 7).

**UFFELMANN'S TEST.**

A means of detecting the presence of organic acids in a fluid. (See Examination of the Stomach, p. 398.)

**UMBILICAL REGION.**

The portion of the abdomen immediately adjacent to the umbilicus. It is arbitrarily limited for purposes of description by four lines: an upper horizontal line joining the lowest points of the two tenth ribs, and a lower horizontal line joining the two anterior spines of the ilia. Laterally it is bounded by two vertical lines passing through the middle point of Poupart's ligament.

In thick abdominal walls (from fat or œdema) the umbilicus is retracted; in ascites it is flattened and stretched, or even project-

ing if the fluid is abundant. It projects in pregnancy and in umbilical hernia. Swelling in the umbilical region may be caused by gastric dilatation or ptosis, by intussusception, floating kidney, tumours of the liver, gall-bladder, and peritoneum.

Enlargement of the veins in the neighbourhood of the umbilicus (the blood current in them being directed away from that point) indicates an obstruction to the blood return in the portal circulation—*e.g.*, cirrhosis of the liver, or thrombosis of the portal vein. Occasionally under these circumstances the enlarged veins are arranged in a radiating fashion with the umbilicus as a centre, and are then known as the **caput Medusæ** (see p. 98).

In this region visible movements of peristalsis may possibly be gastric, but are more frequently intestinal; in the former case the pylorus is almost certainly constricted. Pulsatile movements are probably aneurism of the abdominal aorta, or possibly an excessively active uninjured aorta. The movements of the fœtus in the pregnant uterus may be felt in this region.

Pain in this region may be an indication of obstruction of the bowels. It is not uncommon for the pain of strangulated inguinal hernia to be referred to this area; omental hernia may cause considerable pain. The conditions mentioned above as causing swelling in this region are also likely to produce pain. (See Pain, p. 274.)

### UNCONSCIOUSNESS (Coma).

The sum of various impressions received from external sources by means of afferent nerve impulses of all descriptions, together with recalled impressions stored in various regions of the brain, are combined to form the conscious state of health. Under a variety of circumstances the consciousness may be disturbed, and it is almost invariably the brain itself that is at fault, rather than the peripheral arrangements for receiving and transmitting impulses.

Different degrees of unconsciousness are to be observed :

(a) **Somnolence (lethargy, sleepiness, hebetude, narcosis)** is a condition in which the consciousness is lost, but can be more or less readily recalled by rousing the patient. It is seen in the healthy sleep following severe physical labour and exhaustion, in alcoholic, chloral, and opium poisoning; in uræmia; in slight degrees of intracranial pressure; in acromegaly, and in hysteria. In the last-named condition the term lethargy is used to denote a more complete state of unconsciousness than is usually indicated by

the word ; it may closely resemble a deep sleep, but occurs under circumstances (emotional excitement) which would banish sleep from the healthy subject. Hysterical unconsciousness shows many grades and varieties. The lethargy may be transient, or, on the contrary, it may be lasting and profound, only distinguished with difficulty from death, and accounting for the occasional and rare occurrence of premature burial. The conditions of semi-consciousness evoked by hypnotic suggestion may be mentioned as cases in point ; one may also look on **somnambulism** as a state of partial (but often complete) unconsciousness. A disease caused by blood parasites, **trypanosomiasis**, or **African lethargy**, is characterized by somnolence. (See Blood Examination, p. 73.)

(b) **Stupor**.—The patient can with difficulty be roused from unconsciousness. The reflexes, of which the knee-jerk and the pupillary light reflex are the most important for diagnosis, are not as a rule abolished, and painful stimulation of the skin usually causes a movement of the part.

The conditions mentioned above in regard to somnolence may, when more intense, give rise to stupor.

(c) **Coma**.—A deep unconsciousness, from which the patient cannot be aroused by stimulation or irritation. It is a symptom of many affections, and like stupor, of which it is a more intense form, it is always a sign of grave import. As a rule, the more complete the unconsciousness the greater the danger. The loss of consciousness may come on gradually or suddenly ; in the latter event the cause will probably prove to be cerebral hæmorrhage or embolism, head injuries, heat-stroke, hysteria, or at times uræmia or diabetes. The last two affections are, however, more likely to give rise to a gradually supervening coma. If the unconsciousness should develop more slowly, and especially if it vary in intensity between mere lethargy and complete insensibility, it is due to a toxic blood state, or to a gradually increasing intracranial pressure. Of the toxic states may be mentioned: The infectious fevers—*e.g.*, typhoid, typhus, acute rheumatism, etc.—uræmia ; diabetes ; septicæmia ; acute yellow atrophy of the liver ; excessive venosity of the blood, as found in asphyxia ; poisons—*e.g.*, opium, alcohol, chloral, coal-gas, etc. Owing to the rigid and unyielding structure of the skull, intracranial disease is in most cases accompanied by pressure on the brain, with resulting unconsciousness, as well as in many cases motor or other disturbances. No doubt the insensibility produced by

TABLE COMPARING STATES

Symptoms.	Uræmia.	Opium-Poisoning.	Alcoholic Poisoning.	Apoplexy.
1. Mode of onset	Usually gradual	Gradual, but rapid	Gradual, but rapid	Often sudden
2. Degree of insensibility	Deep coma; cannot be roused	Deep narcosis; can usually be roused with difficulty	As a rule can be roused	Cannot be roused
3. Aspect ..	Pallid; perhaps œdema; has the aspect of renal disease	Face dusky, livid, or cyanosed	Flushed or cyanosed; rarely pale	Flushed, cyanosed, or grey
4. Condition of the muscles	Convulsions, twitching, or rigidity; sometimes paralysis	Sometimes convulsions	Twitchings or tremor	Hemiplegia commonly
5. Pulse ..	Infrequent; high tension	Full and infrequent	Full; frequent	Full; infrequent; of high tension
6. Respiration	Laboured; noisy	Slow; laboured; noisy; often Cheyne-Stokes	Deep; slow; sometimes stertorous	Slow; stertorous; sometimes Cheyne-Stokes
7. Temperature	Normal or subnormal; raised in convulsive attacks	Normal	Usually subnormal	Raised
8. Smell of the breath	Heavy, offensive	Odour of opium	Odour of alcohol, with fœtor	No distinctive odour
9. Pupils ..	Inconstant; may be widely dilated or of medium size	Markedly and equally contracted	Dilated	Variable; usually dilated, always inactive
10. Other prominent symptoms	Convulsions usually occur. Urine scanty or suppressed. Albumin present	Skin dry (except towards the end) and warm	History of alcoholic excess. Irritable or abusive when roused	Patients commonly elderly males. History of gout, arterio-sclerosis, lead-poisoning. Family history may indicate atheroma. Conjugate deviation often present, the eyes looking toward the lesion in most cases

## OF UNCONSCIOUSNESS

Meningitis.	Diabetes.	Hysteria.	Syncope.
Gradual	Gradual or sudden	May follow a convulsion	Sudden
Cannot be roused	Cannot be roused	Can be roused	Roused by stimulating the circulation
Cyanosed or pale	Cyanosed or pale	Flushed	Pale
Limbs often rigid and flexed. Often convulsions	Unaffected	Epileptiform convulsions common	Unaffected
Infrequent ; may be rapid if temperature raised	Normal and full	Unaffected	Weak or absent
May be frequent if temperature raised	Labourd and rapid ('air hunger')	Rapid, but not stertorous	Shallow and almost imperceptible, or sighing
Often raised	Subnormal	Unaffected	Unaffected
No distinctive odour	Sweet, fruity, 'like over-ripe apples')	Unaffected	Unaffected
Inconstant	Inactive	Equal ; normal size or dilated ; react to light	Widely dilated
Patients commonly youthful. Headache ; vomiting ; retraction of head in some cases. History of ear disease, tubercle. <i>Diplococcus intracellularis</i> in fluid (obtained by lumbarpuncture) in cases of epidemic cerebro-spinal meningitis	Most severe in youthful patients. Headache, vomiting, drowsiness often precedes coma. Sugar in the urine	Almost exclusively infemales, Coma resembles deep sleep, but caused by conditions which prevent normal sleep (emotional excitement, etc.). Eyelids are kept closed, and resist attempts to open them	Females more commonly affected than males. Eyes often remain open

destructive lesions of the cerebral tissue—*e.g.*, hæmorrhage, softening—results in some degree from the actual damage effected by the diseased process. The chief share, however, in the production of coma in these circumstances is the pressure of the accompanying extravasated fluids and infiltration. Of the intracranial affections causing coma the following, in addition to those mentioned above, are the most noteworthy: Meningitis (tubercular, pneumonic, epidemic cerebro-spinal, etc.); tumour and abscess of the brain; encephalitis; general paresis; thrombosis of the cerebral vessels or of the sinuses; disease in the cranial bones. A condition of apparent wakefulness, but in reality a muttering unconsciousness, is that known as **coma vigil**, seen in cases of low delirium (See Delirium, p. 121.)

**Syncope.**—The loss of consciousness due to failure of the circulation in the brain is in some respects different in its nature from coma. It is due to a sudden anæmia of the brain, and is recognized by the sudden pallor, sickness, weakness of pulse, sweating, and often dyspnœa.

The symptoms by which some of the more important and commonly occurring forms of unconsciousness may be distinguished, are enumerated and compared in the table on pages 500 and 501.

### URINE, Abnormalities of.

Quantity of urine passed daily—Colour—Odour—Translucency—Reaction—Density—Deposits: pus, blood, hæmoglobin, epithelium, casts—Uric acid—Purin bodies—Urates—Phosphates—Oxalates—Leucin and tyrosin—Cystin—Cholesterin—Fat—Micro-organisms—Albumin—Sugar—Acetone—Diacetic acid—Hydroxybutyric acid—Pentoses—Albumoses—Bile—Indican—Urea—Nucleo-albumin.

The habitual examination of the urine should be practised, not only in the case of patients seen for the first time, but also at suitable intervals. The methods recommended as best suited for practical clinical purposes, and which may be efficiently carried out by the practitioner, are described in the article upon the Examination of the Urine (p. 526). Here the abnormal conditions found on examination, and their diagnostic significance, are considered.

1. The **Quantity** of urine passed. Normally, in the adult it is about 50 ounces daily. Any important deviation from this amount may be pathological or merely physiological.



Increase in the quantity may be caused by—(a) inact. on of the sweat-glands, as in cold weather; (b) recent copious draughts of any fluid, but more especially of dilute alcohol; (c) hysteria, epilepsy, or simple emotional excitement; (d) drugs which exert a diuretic influence on the kidneys, or which raise the arterial tension in the kidneys—*e.g.*, digitalis, broom, caffeine, juniper, nitrate of potash, etc.; (e) absorption of dropsical effusions; (f) diabetes mellitus and insipidus; (g) granular or contracted kidneys (the small red kidney); (h) the atrophic form of chronic parenchymatous nephritis (the small white kidney); (i) the lardaceous kidney.

A considerable increase in the quantity of urine secreted is termed **polyuria**.

The removal of three-fourths of the kidney tissue experimentally is followed by an increase in the quantity of urine (Rose Bradford).

Decrease in the quantity is noted in (a) excessive perspiration, as in hot weather; (b) a relatively dry diet; (c) diminished absorption of fluid from the stomach in cases of gastric dilatation from pyloric stricture; (d) copious loss of fluids, as in diarrhœa, dysentery, severe hæmorrhage, vomiting, cholera; (e) fevers; (f) shock; (g) diminished arterial pressure in the kidneys and elsewhere, as in heart disease; (h) congestion of the kidneys; (i) the accumulation of dropsical effusions; (j) acute and chronic parenchymatous nephritis; (k) obstruction to the flow in the urinary passages.

2. The **Colour** is commonly a brownish shade of yellow ('amber-coloured'), but many shades of difference are compatible with health. As a rule, (a) the denser a specimen of urine, the darker the shade of yellow, the chief exception being the pale greenish yellow urine of diabetes mellitus, with a high specific gravity; (b) when the urine is copious it is usually pale in colour; (c) when a darker shade than normal, it may be due to fevers, where altered metabolism and increased hæmolytic changes occur; (d) when 'smoky' it indicates a small quantity of blood present (see p. 503); (e) when red or dark like porter, a larger quantity of blood; (f) shades of brown, light to dark, may mean either blood, carbolic acid, bile, or melanin; (g) greenish, or greenish-brown, bile, salol, carbolic acid; (h) whitish or yellowish urine may indicate the presence of phosphates, pus, or spermatozoa, or, if gelatinous, possibly chyle.

3. The normal **Odour** is diminished in urines of low density, and increased with higher specific gravity, when the latter is due

to larger excretion of urea. If (a) ammoniacal, it is probably due to putrefactive changes; (b) the odour of violets is produced by the administration of turpentine; (c) several substances give their own characteristic odour to the urine—*e.g.*, garlic, sandalwood, copaiba, cubebs, tolu; (d) in diabetes a sweetish odour is observed.

4. **Translucency.**—Normal urine should be clear when voided. (a) After standing, a cloud of mucus is deposited at the lower portion of the vessel. (b) When the urine is concentrated, and contains an excess of urates, these are thrown, on cooling, as a brick-red or pink precipitate. (c) Should the urine be alkaline, as is often seen in slight digestive disturbances, etc. (see below, Reaction), the fluid will be turbid when passed, becoming clear on the addition of a few drops of acetic or other acid. This is due to the presence of phosphate of calcium, or sometimes phosphate of magnesium, which are insoluble in an alkaline medium. The phosphates of sodium and of potassium are also normally present, but are not precipitated. (d) If the turbidity does not clear up on the addition of an acid, it will probably prove, on microscopical examination, to be pus (see below, p. 506). (e) Rarely, a whitish turbidity is due to chyle, passed in the course of filariasis, and it has been observed, still more rarely, without evidence of the presence of the parasite. (f) A turbidity is produced by blood, dusky or 'smoky' when in small or moderate quantity; it may be thick, porter-coloured, or bright red when more copious (see below, p. 506).

5. **Reaction.**—Normally, this is acid, but at the height of digestion the acidity is much reduced, and the urine may be neutral or even alkaline, particularly with a vegetable diet. The acidity is not due to free acid in the urine, but to acid phosphate of soda. The reaction is **alkaline** after prolonged cold baths, in dyspepsia—especially of neurotics—in anæmia, debility, and the administration of alkaline drugs. Holt has shown that, by rendering alkaline the acid urine of children suffering from pyelitis due to *Bacillus coli*, a prompt improvement is effected. In affections of the lower urinary passages, especially those which interfere with the complete evacuation of the bladder—*e.g.*, enlarged prostate or stricture of the urethra—alkaline and ammoniacal urine is common. It is easier to produce alkaline reaction in acid urine, by means of drugs administered internally, than to change alkaline urine into acid by the administration of acid-forming drugs. (For this purpose the best drug is benzoic acid.)

Acidity of the urine is increased by exercise, hot baths, fevers, and by all conditions in which the concentration of the urine is increased. In gout, diabetes mellitus, acute and chronic rheumatism, and when the diet is chiefly composed of animal food, the increase in acidity is marked. In this condition of urine amorphous urates are likely to be deposited, and, after standing, uric acid crystals may be found.

6. **Density.**—The average specific gravity is about 1020 in health, the limits of healthy density being usually 1015 to 1025, but it may normally be found much outside these figures—*e.g.*, copious drinking reduces, and profuse sweating raises, the density. The chief solid in the urine is urea, and alterations in the density most frequently depend on variations in (a) the quantity of urea by the kidneys, and (b) the bulk of water which has passed through them. Chloride of sodium is also an important ingredient of the solids. A healthy man passes, say, 1,500 grammes of urine in twenty-four hours, of which 1,440 grammes are water and 60 grammes are solids. Of the solids, 35 grammes are urea, and 16.5 grammes are sodium chloride, the balance being composed chiefly of alkaline and earthy phosphates and sulphates (Halliburton). The presence of albumin has only a slight effect in raising the density.

An increase in the specific gravity is noted, as above stated, when the individual has been sweating profusely, in fevers, and when the urine is scanty. The various conditions mentioned above under 'Quantity' (p. 498), which show a diminution in the amount of fluid, exhibit, then, a urine of high density—*e.g.*, vomiting, diarrhœa, acute nephritis, etc.

On the other hand, a very high specific gravity is commonly found in diabetes mellitus, where, instead of a scanty urine, the quantity is invariably increased. Here the increased density is mainly due to the presence of sugar, though there is often an increase of urea as well, produced chiefly by the nitrogenous diet which is usually prescribed. (See Sugar, p. 521.)

Diminished density is found in conditions which increase the quantity of urine (with the important exception just mentioned). It is observed in many conditions of cachexia, in neuroses, in diabetes insipidus. As shown by Dr. Rose Bradford, loss of kidney tissue is followed by an increase in the amount of fluid excreted by the remaining portion of the organ, with a corresponding fall in the specific gravity. Hence the latter is lowered

in the small red kidney, in the small white kidney, in hydro-nephrosis, and in cystic disease of the kidneys.

7. A **Deposit** may be observed in the urine, either immediately after being voided or on standing for some time. A microscopical examination must in every case be made. (See Urine, Examination of.) Before proceeding to this, observe the naked-eye appearance of the sediment.

(a) **Pus.**—A creamy deposit with clear, acid, supernatant urine; or a thick, slimy, partly coagulated, whitish-yellow sediment in turbid, alkaline urine, indicates in both cases pus. Under the microscope a multitude of colourless, rounded cells, larger than red blood-corpuscles, with a divided nucleus, is seen.

If from the urethra, it is small in quantity, and is probably due to gonorrhœal urethritis. A non-specific urethritis does, however, occasionally occur in gouty subjects, and in connection with prostatic or bladder disease.

Pus derived from the bladder is usually in an alkaline urine, and may be in semiclotted, ropy, or slimy masses. It may be due to tubercular disease, calculus, cystitis, prostatic enlargement, or stricture. Tubercular disease of the ureters or kidneys, renal calculus, pyelitis from the invasion of micro-organisms (including *Bacillus coli communis*), and abscess of the kidney, may be the cause of pyuria. In those conditions the urine is usually acid, the pus may be in large or small quantities, and is often intermittent in its appearance. These latter distinctions do not always obtain, as the renal disease may be an accompaniment or a sequela—*e.g.*, surgical kidney—of lesions in the bladder or urethra.

Lastly, it must be remembered that pus found in the urine may not be derived from the urinary passages. It may come from the uterus, vagina, or vulva in females, or from beneath the prepuce in males. When pus is filtered from the urine the clear fluid may contain a small quantity of albumin derived from the pus, and not of renal origin. If, however, the amount of albumin is considerable, it has probably come from the kidney.

(b) **Blood.**—A dark, chocolate-coloured, or red deposit, sometimes like coffee-grounds, or possibly clotted, in irregular or string-like masses, is produced by blood from the urinary passages (*hæmaturia*). On microscopical examination the red cells are found, separate (not in rouleaux), often distorted in shape, though the biconcave disc without a nucleus can be recognized easily.

Blood, like pus, may come from any portion of the urinary tract. At the meatus and in the urethra gonorrhœal ulcers and chancres may be the cause. It would, under these circumstances, appear in the first portion of the urine voided; or it may be observed exuding from the meatus, independently of micturition. Prostatitis and varicosities about the prostate, or lesions of the orifice of the bladder, from stone, may be the cause of the hæmorrhage, which may be intermittent. There may in these conditions be frequency of micturition, with pain in the penis or hypogastrium, particularly felt at the end of evacuation. If it is voided soon after its effusion it is red, in both cystic and urethral lesions; whilst from the kidney the blood is darker, having undergone chemical change. Ulcers of the bladder, generally tubercular and villous, and malignant growths are, in addition to the conditions just mentioned, the chief causes of bleeding from the bladder.

Hæmorrhage from the upper urinary passages (ureter, pelvis, and kidney tissue) may be due to renal calculus, tubercular disease, malignant tumours (sarcoma or carcinoma), or to acute, subacute, or even chronic nephritis, and to granular kidney. In acute fevers of a malignant type (see below, p. 508) and in hæmophilia (renal epistaxis) hæmaturia is often seen.

In tropical countries parasites cause renal hæmorrhage—*e.g.*, *Filaria sanguinis hominis* and *Bilharzia hæmatobia*. Hæmophilia and scurvy are occasionally the causes of similar hæmorrhage.

*Hæmoglobinuria*.—In certain infrequent cases we find the blood pigment in the urine, and on microscopical examination an amorphous reddish-brown deposit, with, perhaps, a few broken or imperfect red corpuscles. Here there has been a destruction of the red cells, an excessive hæmolysis, from some cause. The passing of blood pigment in the urine is the chief symptom of the disease named paroxysmal hæmoglobinuria, the pathology of which is uncertain. The blood pigment in this affection appears at intervals from some such exciting cause as cold or excessive exercise, and is accompanied by signs of severe constitutional disturbance. It has been observed in persons who are the subjects of Raynaud's disease, a disorder of the vasomotor system (see p. 48).

Another group of affections characterized by the appearance of blood pigment in the urine is toxic hæmoglobinuria. The red blood-corpuscles are so damaged by the presence of a poison that their pigment escapes into the plasma, and is removed therefrom

by the kidneys. This destructive action of certain toxic agents upon the red cells is termed **hæmolysis**. Various drugs have this hæmolytic action—*e.g.*, carbolic acid, chlorate of potash in large doses, arseniuretted hydrogen, carbon monoxide, etc. Quinine is stated by some observers to have this effect, the hæmoglobinuria of malaria being by them attributed to the large doses of quinine administered, whilst others refer this 'blackwater fever' to the destructive action of the parasite upon the red cells. The poisons of other infections, especially in severe cases, may have the same effect, as in yellow fever, scarlet fever, typhoid fever, and syphilis. In these affections, not only hæmoglobinuria, but also hæmaturia, may occur.

(c) **Epithelium**.—Under the microscope epithelial cells of various descriptions may be found. In the first place, squamous cells from the vagina in females may be distinguished as large, flat, polyhedral, faintly granular cells, with a small central nucleus. Bladder-cells may be flat and very similar to those of the vagina, but the deeper layers of epithelium from the bladder bear a strong resemblance to the cells from the ureter, kidney pelvis, and tubules of the kidney. The renal cells are usually smaller, with a relatively larger nucleus, more distinct outline, and darker granulation than those of the lower urinary tract. The prevailing type of cell in the latter situation is a flattened, cubical, tailed cell—a transition between the squamæ of the superficial layer of bladder cells and the more columnar type of the renal tubules. It is, however, often quite impossible to decide from which source the cells were derived, as they may be much altered by their separation from the mucous membrane, and by the subsequent action of the urine when alkaline. The presence of renal cells in the urine, when identified, indicates nephritis. They often resemble leucocytes, but the large undivided nucleus of the former serves to distinguish it.

(d) **Casts** are usually accompanied by albumin in the urine. They are microscopical objects of considerable diagnostic value. Their presence locates, with rare exceptions, disease in the kidneys. Moreover, the prevailing type of cast found assists us to distinguish between the varieties of renal affections.

Tube casts, as their name indicates, are moulds of the renal tubules. The material of which they are formed is a point upon which opinion differs, except in the case of blood casts, where they are obviously composed of fibrin. The other forms may be

(a) simple coagulated albumin from the blood, (b) material secreted by diseased epithelium, or (c) material formed from shed renal epithelium. In support of the last hypothesis is the fact that certain epithelial casts are evidently the coherent epithelial lining of the tubes, a lumen having been sometimes observed.

The following are the chief varieties of casts :

Hyaline casts—clear, almost transparent.

Epithelial casts—epithelium shed *en masse*, or hyaline casts with epithelial cells adherent or embedded.

Granular casts—studded with granules, which may be scanty or profuse, and which are probably the remains of disintegrated epithelial cells. The granules are therefore in part composed of fat droplets. When the fat droplets are a prominent feature the casts are termed—

Fatty casts.

Blood casts—coagulated fibrin in which red blood cells are embedded, or hyaline casts with adherent red cells.

Leucocytic casts contain, or are mainly composed of, white blood-corpuscles.

Waxy casts—pale, clear, often yellowish, highly refractive; probably changed and degenerated epithelial casts, or may be simple hyaline casts which have been retained a long time in the tubules and have undergone degeneration.

The diagnostic value of casts is by some clinicians considered trifling. Real help, however, can often be secured by a careful study of the prevailing type of cast present. In the first place—

(i.) Casts, when found in albuminous urine, indicate renal disease. A possible exception to this statement is the functional albuminuria which is sometimes accompanied by the excretion of hyaline casts.

(ii.) Blood casts are found in acute nephritis, and in chronic nephritis if hæmorrhage is occurring. Renal hæmorrhage from any cause may produce blood casts.

(iii.) Fatty casts appear in chronic hypertrophic or parenchymatous nephritis, and rarely in renal sclerosis or interstitial nephritis.

(iv.) Epithelial casts and granular casts are found in parenchymatous nephritis, and rarely in the contracted kidney.

(v.) Hyaline casts are of the least diagnostic value, as they may occur in any form of renal disease, and occasionally when the kidneys are believed to be healthy (see above). In jaundice

without obvious renal disease bile-stained hyaline casts may be found.

(vi.) Waxy casts occur independently of lardaceous disease of the kidneys, where, indeed, they are very rarely found. They may be seen in any form of nephritis.

(vii.) Lastly, it must be remembered that it is the **prevailing type** of cast that we look to for help in the diagnosis. Any of the casts above mentioned may be found in any form of nephritis, but the variety of cast which is most numerous in any specimen of urine is our guide.

(c) Various **Crystals** and **Amorphous Salts** may be seen in the deposit—viz.:

(i.) **Uric Acid**.—In normal urine this is present in small quantity, 8 to 10 grains daily being excreted, and is all in combination in the form of urates. If an acid urine is allowed to stand for some time, crystals of uric acid may be seen as a scanty reddish deposit. Chemically, uric acid may be detected by the 'murexide' test (p. 538). Microscopically it is seen as pink crystals of various shapes, rhombic, dumb-bell, lozenge, star, whetstone, and lance-head, and yellow or brownish in colour.

Excess of uric acid, like that of urea (*q.v.*, p. 525) is a result of excessive proteid metabolism, and is noticed in febrile and wasting diseases, in phthisis, diabetes, pernicious anæmia, scurvy, leukæmia, and after an attack of gout. Certain drugs increase its excretion—*e.g.*, salicylic acid, colchicum, and euonymin. In acute rheumatism, in the uric acid diathesis, after prolonged or severe exercise, whilst a diet mainly nitrogenous is used, the excretion of uric acid is increased.

A decrease in the excretion is observed before and during an acute attack of gout, in anæmia, in chronic nephritis, with a milk diet, during the administration of quinine, caffeine, sodium carbonate, and other alkalis, and of iodide of potassium.

(ii.) **Purin Bases**.—The xanthin or alloxuric bases form, with uric acid, a group of derivatives of proteid metabolism. Their presence in the urine is due to the decomposition of tissue nuclein, or to the ingestion of food containing these bodies (nucleins). They may thus be divided into two groups: endogenous purins, derived from tissue changes, and exogenous purins, derived from food containing purins. The former is the chief source of their formation, and it is with reference to the changes



in the tissues that the estimation of their presence in the urine is of interest. While the methods for determining the quantities of purin bodies present in the urine are hardly suitable for routine clinical investigations, they have of late received considerable attention, and the technique involved has been considerably simplified, notably by the apparatus introduced by Dr. Walker Hall. This observer summarizes the results of the estimation of pathological variations in the amount of urinary purins thus:

Uric acid is increased in alcoholism with enlarged liver, carbon monoxide poisoning, cirrhosis of the liver, gout, leukæmia, neurasthenia and migraine, pneumonia, sepsis, scurvy.

Uric acid is decreased in anæmias and gout.

Xanthis are increased in adiposity, in diabetes (owing to excessive meat ingestion), diphtheria, scarlatina, nephritis, hyperthyrea.

(iii.) **Urates.**—Amorphous urates of sodium, potassium, and calcium are seen microscopically as granular, opaque masses. They are sometimes so deposited as to resemble granular casts. Crystalline forms of the sodium and calcium salts occur in acid urine, the former as balls (plain or spiked), 'hedgehog' forms, and stars (the latter arranged in stars or bundles of needles. Ammonium urate appears in similar forms in alkaline urine. It is, therefore, usually found in company with phosphates. It is the amorphous urates which form the ordinary brick-red or lateritious deposit so frequently observed in disorders of the alimentary system, serious or trivial, or even in health.

(iv.) **Phosphates.**—Calcium phosphate occurs as an amorphous deposit along with the crystalline ammonio-magnesium phosphate in alkaline urine. The latter, commonly (and erroneously) called 'triple phosphates,' is found as stars, coffin-lids, knife-rests, and feathers. The potassium and sodium salts, unlike the earthy, are freely soluble, even in an alkaline medium, and do not, therefore, occur as deposits.

The presence of precipitated phosphates may be due merely to diminished acidity of the urine, and not to an excessive quantity of the salts. It is, therefore, in many cases of no clinical importance. (See Reaction, above, p. 504.) On the other hand, an actual increase of the phosphates (phosphaturia) is found in rickets, osteomalacia, in leukæmia, in severe anæmia, in wasting and inflammatory diseases of nerve tissue, and in wasting

diseases generally. An alkaline urine with deposition of phosphates is seen in dyspepsia, and especially in neurotic subjects.

(v.) **Oxalates.**—The oxalate of lime is found usually in acid urine as small octahedral, or, more rarely, as oval or dumb-bell shapes, the familiar 'envelope' crystal being an octahedral with an apex presented towards the observer.

Oxalates may often be found, after urine has stood for some time, in certain digestive disturbances. Injudicious or excessive food, certain vegetables (rhubarb, tomatoes, and onions) in susceptible persons, want of exercise, anxiety, are the more common causes. It is sometimes observed in diabetes, when the sugar diminishes. Its continued presence causes the formation of calculi, irritation of the urinary tract, and even albuminuria.

(f) **Leucin and Tyrosin.**—These bodies are normally oxidized into urea, but are found in serious disease of the liver, notably in acute yellow atrophy; also in phosphorus-poisoning, pernicious anæmia, and sometimes in typhoid fever.

Tyrosin appears as needle-shaped crystals arranged in bundles, sheaves, or stars. Leucin is found as spheres, light brown in colour, with radial or concentric striation. It is less soluble than tyrosin, but both bodies are usually found in the same specimen of urine.

(g) **Cystin** is a rare deposit. It occurs as thin, transparent, hexagonal plates in acid urine. Its chief diagnostic value lies in the fact that it may, when plentiful, cause stone in the bladder.

(h) **Cholesterin** is also a rare sediment. It is found in thin yellowish, rhombic plates, with one or more notched corners. It occurs in jaundice, fatty degeneration of the kidneys, and chyluria.

(i) **Fat.**—In fatty degeneration of the kidneys oil droplets are found free in the urine. In chyluria these are so numerous as to give a milky appearance to the fluid.

(j) **Micro-organisms.**—The presence of bacteria diffused through a specimen of urine imparts to it a turbidity, and on moving the fluid a curious opalescent, wavy movement of the cloud in the urine is seen. The opacity differs from that produced by urates, phosphates, pus, etc., in showing no tendency to settle in a few hours.

The methods to be employed in the detection of the various micro-organisms are described in the article on the Examination

of the Urine (p. 528). The following varieties occurring in the urine are those which have diagnostic interest :

(i.) *Gonococcus* is usually detected in the urethral discharge, and in the fibrinous filaments often observed in the urine of cases of chronic gonorrhœa ; it is less frequently discovered in the urine in cystitis.

(ii.) *Tubercle bacillus* occurs in the urine in tuberculosis of the prostate, bladder, ureters, or kidneys. In many cases the organism is difficult to discover, so that its absence should not be too much relied upon as an evidence against tuberculosis.

(iii.) *Streptococci* and *staphylococci* are found in the urine in septic and suppurative affections of the urinary tract.

(iv.) *Bacillus typhosus* is sometimes found in the urine in cases of typhoid fever, and may at times be detected a considerable time after the fever has disappeared. The organism is difficult to discover, and its identification in the urine is not practicable as a routine clinical investigation.

(v.) *Bacillus coli communis*, which is a normal inhabitant of the intestine, is at times to be seen in the urine. In such cases the urine is found to be acid, and often contains pus. It usually gives rise to cystitis, but may be the cause of a form of pyelitis, usually found in female children, in which there is a hectic temperature, acid urine containing pus, and often a history of preceding constipation or bleeding from the bowel.

8. **Albumin.**—The presence of serum-albumin or of globulin, or of both bodies, in the urine is demonstrated by the various tests described at p. 531. While it is true that such urine must be declared abnormal, it is not certain that in every case the kidneys or urinary passages are diseased.

Two main groups of conditions characterized by albuminuria are to be distinguished: (*a*) Those in which the urine arrives in the urinary passages free from albumin, but gathers it on its way to the urethral meatus; (*b*) those in which albumin is transferred into the urinary passages during the process of excretion of the fluid.

The first group is mainly of surgical interest, and will be only briefly considered.

*Pus* is the commonest source of non-renal albuminuria. If the pus be in considerable quantity, and its cellular elements be removed by filtration, a small amount of albumin will be found ;

a copious deposit of albumin after removal of the pus cells will, therefore, point to a concurrent renal affection. The clinical significance of pyuria is referred to above, at p. 506.

*Blood of non-renal origin gives rise to albuminuria, but it is frequently a sign of kidney disease (see p. 506).*

It must also be borne in mind that pus, blood, epithelial cells, and debris may reach the urine from extra-urinary sources, especially in the form of vulvar and vaginal discharges in the female, and subpreputial in the male.

*Albuminuria of Renal Origin.*—The proteids of the blood may under certain circumstances traverse the capillary walls and the epithelial lining of the urinary passages, to appear in the urine. In many instances this is obviously due to a diseased condition of the renal epithelium, and so commonly is albuminuria apparently the result of renal disease, that its presence is by many regarded as a sign of renal inefficiency. No doubt the efficiency of the kidney can be more accurately judged by observing if it is able to elaborate from the blood a concentrated saline solution, than by ascertaining that it does not remove proteid matter from the blood. Indeed, Sir A. E. Wright, who has created this department of renal pathology, relegates to the antiquities of medicine the view that albuminuria is a sign of renal inefficiency. Until, however, the methods for ascertaining the respective salts-contents of the blood serum and of the urine are more within the reach of the clinical observer than those afforded by cryoscopy, or by Wright's ingenious hæmolytic process, the examination of the urine for albumin will remain an important procedure in the diagnosis of kidney affections. (See the article on Blood Examination, p. 77.)

Three groups of conditions exhibiting albuminuria may be distinguished: (i.) The so-called functional or physiological albuminuria; (ii.) albuminuria of pathological origin, without definite disease of the kidney; (iii.) albuminuria with definite kidney lesion.

(i.) In a considerable number of cases a small quantity of albumin may appear in the urine, either continuously or at intervals, without any other evidence of ill-health. The use of the term **physiological** or **functional albuminuria** in these cases is open to objection, as in many instances, at least, the condition of the urine is due to pathological changes in the kidney.

This group was defined by Grainger Stewart as comprising

those cases 'where exercise or exertion, diet, exposure to cold, mental emotion, and obscure psychic influences account for the symptom.' Several varieties of functional albuminuria have been described—viz., **periodic, intermittent, cyclical, or paroxysmal.** Here the albumin appears at certain times of the day, to disappear later, and perhaps reappear the next day, or it may be present for weeks or months together, and then disappear for a long period. **Albuminuria of adolescents** is a distinct and important variety. It is chiefly observed in boys, and is in many cases associated with undue activity of the genital organs. This may be caused by nutritional changes in the renal tissue of central nervous origin, the genital and renal trophic centres being closely connected. The amount of albumin is usually small. It may be increased by proteid food or by exertion, and may therefore be absent in the morning. In some cases it is not found when the youth is in the recumbent position, but appears as soon as he assumes the upright attitude—the so-called **postural or orthostatic albuminuria.** Functional albuminuria in some cases occurs while digestion is proceeding, and is then termed **dietetic albuminuria.** Again, it may be produced by excessive exercise, by exposure to heat or to cold.

In any of the forms of functional albuminuria one may find hyaline casts in the urine at times, but the albumin is temporary, and there is an absence of those cardio-vascular changes which characterize renal disease.

This condition is regarded by some authors as a mere physiological disturbance, without pathological significance. Saundby, Monro, and others state that functional albuminuria need not prevent an adolescent from being accepted by life assurance companies at ordinary rates. Wright has published the results of examining the salt-abstracting capacity of the kidneys in a series of cases of physiological albuminuria, and has found the organs efficient. By administering calcium salts to those patients he diminished or dismissed the albumin from the urine, owing to the consequent increased coagulability of the blood, while the administration of lime salts to individuals suffering from nephritis did not result in any diminution in the amount of albumin present. Wright concludes from these and other confirmatory observations that albuminuria in this class of case is the result of increased hydrostatic intracapillary pressure, combined with diminished coagulability of the blood, and that this form of

albuminuria is practically an exudation of lymph into the urinary tubules, which are lined by healthy epithelium.

On the other hand, a good deal can be said in favour of the widespread opinion that renal albuminuria is always an evidence of some change, either temporary or serious and progressive, in the kidney. In the first place, many of the cases of physiological albuminuria show definite formed renal elements in the urine, such as epithelial cells and casts in small numbers. In functional albuminuria increase of intracapillary pressure, which is recognized as a probable cause of the passage of albumin, may itself be the cause of definite, though slight, changes in the renal epithelium. Such changes do undoubtedly occur in the passively congested kidney mentioned below, where the damage results from excessive intravenous and intracapillary pressure. The temporary forms of albuminuria, such as febrile albuminuria and other varieties to be referred to presently, and which may be regarded as a step further on toward definite renal disease, show slight but distinct epithelial changes. These are the 'cloudy swelling' in the secreting cells of this as well as of other organs in those conditions. Again, in many cases there is no definite boundary between the temporary albuminuria due to morbid blood states, such as poisoning by cantharides, and the more serious and destructive acute nephritis.

A certain proportion of the cases of physiological albuminuria ultimately develop nephritis, though many cases have been recorded as free from albumin years after the functional affection was first noticed. It may also be remembered that the temporary febrile albuminuria in cases of scarlet fever and of diphtheria is often the forerunner of nephritis.

If a change does occur in the epithelium lining the urinary passages in cases of physiological albuminuria, it probably causes no serious or permanent injury to the organ, and may be regarded as analogous to catarrhal processes in other less vital mucous-lined tracts. Such a condition would denote an unusual vulnerability, which in the case of the kidney must be a serious consideration in forming a prognosis, and should make one hesitate in life assurance examinations to recommend the acceptance of such cases as an ordinary risk.

(ii.) *Albuminuria associated with Disease in Some Part of the Body, but without Definite or Serious Kidney Lesion.*—The affections comprised in this group differ from the foregoing in degree

rather than in kind. They include (a) **Febrile albuminuria**. Fever from any cause produces slight temporary changes in the renal epithelium, the so-called 'cloudy swelling' which commonly occurs in pneumonia, typhoid fever, scarlet fever, diphtheria, and phthisis. In the three last-named affections this transient albuminuria must be distinguished from the more serious kidney affections which sometimes occur at a later stage of the disease in question—namely, Bright's disease after scarlet fever and diphtheria, and amyloid disease of the kidneys as a result of tuberculosis. (b) **Morbid blood states** may cause slight or temporary albuminuria, as, for instance, scurvy, purpura, syphilis, anæmia, leukæmia, gout, chronic poisoning by lead, mercury, etc. (c) In certain **diseases of the nervous system** one finds temporary albuminuria at times—*e.g.*, epilepsy, tetanus, apoplexy, convulsions, injuries to the head, and also in exophthalmic goitre.

(iii.) *Albuminuria with Definite Kidney Lesion*.—This includes—  
(a) **Congestion of the kidneys**, either active or passive. The former is seen in early stages of nephritis, in poisoning by cantharides, turpentine, alcohol, and other drugs. Passive congestion is produced by obstruction to the venous return from the kidney, as in heart and lung diseases, or by pressure of abdominal tumours, including the pregnant uterus. (b) **Acute and chronic nephritis (Bright's disease)**. Here the lesion is simply more intense and destructive than those which cause albuminuria in the foregoing conditions; but even in these grave affections, or at any rate in the chronic forms, abnormalities in the urine may be the only symptom of disease obtainable. Indeed, albuminuria is in many cases the only readily obtained evidence of the inadequate activity of the kidney, for the healthy organs are accustomed to work with such a considerable 'margin of safety' that moderate deficiency is not perceived as a disturbance of the normal functions. Moreover, there is an attempt to compensate for the deficiency by transference of a portion of the defective organ's function to other regions—*e.g.*, the intestines—and the impaired excretion of the kidney may also to some extent be assisted by the increased activity of the circulation resulting from the hypertrophied ventricle. So long, then, as the margin of safety suffices to cover the imperfect renal secretion, and so long as the latter is assisted and compensated by the means just described, there will be no evidence, except the presence of albu-

minuria, of a morbid condition of the kidneys. As soon, however, as the kidneys, even with this assistance, are unable to successfully meet the need for elimination of waste products—in other words, as soon as compensation fails—renal incompetence becomes apparent. This is indicated by one or more of the various symptoms of uræmia, and by œdema. We may thus usefully compare the effects produced by kidney diseases with those resulting from heart lesions. In both cases compensation obviates all symptoms except those discoverable on examining the organ affected.

The boundary between febrile albuminuria and nephritis is sometimes ill-defined. For example, in scarlet fever and in diphtheria slight albuminuria is often found at the height of the fever, disappearing soon, as in other instances of febrile albuminuria. Subsequently acute nephritis may develop, with the usual signs of a definite renal lesion (œdema, increased arterial tension, abundant albumin or blood and casts in the urine). The initial or febrile albuminuria occasionally persists until the nephritis is established. Again, in pregnancy, as above stated, albuminuria may arise from a mechanically produced impediment to the venous return from the kidneys; but the nephritis which so often occurs in pregnancy is of less simple origin, as is shown by the profound metabolic changes arising in puerperal eclampsia, in which the liver takes an important share.

The albuminuria of nephritis varies in quantity, but is practically always abundant. In explanation of this statement it is understood that nephritis, or Bright's disease, is an acute or chronic inflammation of the essential excretory tissue of the organ, and of its supporting connective tissue; that is to say, in addition to acute nephritis, Bright's disease comprises the large white and the small white kidney. On this understanding the small red kidney is excluded from the inflammatory affections of the organ. There seem to be good grounds for accepting the view expressed by some authors that the small red or sclerotic kidney is not a nephritis, but is a primary renal affection, the result of arterial degeneration.

Turning again to the heart for the purposes of analogy, one notices a striking resemblance in the respective affections of the organs. In the heart valvular lesions may be the result of an inflammatory process, endocarditis, and these, like the inflammations of the kidneys, form a very large proportion of the cases of



failure of the organ. On the other hand, certain valvular heart lesions are due to a primary arterial degeneration, and differ fundamentally from inflammatory valvular disease in origin, symptoms, and progress. The primary arterial degeneration of renal disease differs from the inflammatory variety in precisely the same respects, and forms in all probability a separate and distinct affection from those inflammatory affections which we recognize as Bright's disease.

Adopting this view of the sclerotic kidney, we may say that albumin appears in considerable quantity in Bright's disease, which comprises three typical forms, with intervening gradations between the varieties—viz. : (a) acute nephritis; (β) chronic hypertrophic nephritis, or large white kidney; and (γ) chronic atropic nephritis, or small white kidney.

(c) The **small red kidney** is, then, the third form of kidney disease causing albuminuria (congestion of the kidneys and nephritis being the first two mentioned). The names usually applied to this affection are unsatisfactory. The term 'gouty kidney' implies a half truth, as a considerable number of the cases met with are quite unconnected with gout. Granular kidney is ambiguous, as the small white kidney presents a distinctly granular appearance on stripping off its capsule. Chronic interstitial nephritis, the name which is, perhaps, most used, should certainly be avoided, as, even if the above classification be rejected, this term makes no distinction between the small red and the small white forms, while if the former be removed from the group of nephritis it is inaccurate. The rather unwieldy term 'chronic arterial renal sclerosis' would, on the whole, be preferable. The quantity of albumin found in the urine in this affection is always small, and it may be absent at times. The urine is abundant, and a few hyaline or granular casts may be discovered.

(d) Lastly, albumin is found in large quantity (though at times it may be scanty) in **lardaceous** or **amyloid disease** of the kidney, which occurs in the course of certain wasting diseases. Here also the urine is abundant, and hyaline or granular casts may be found in small numbers.

As will have been observed from the foregoing statements, the quantity of albumin to be found in the urine of different affections varies considerably, and this fact may be of assistance in diagnosis. The following **summary** is therefore given :

*Quantity of Albumin in Urine.*

An abundant deposit of albumin is observed in—

- (a) Acute nephritis and hæmaturia from any cause.
- (b) Chronic hypertrophic nephritis when well established.
- (c) Lardaceous disease of the kidney.
- (d) In the final stages of all forms of renal disease, when the kidney has become inadequate.

A moderate quantity of albumin is observed in :

- (a) Slighter degrees of chronic hypertrophic nephritis.
- (b) Chronic atrophic nephritis, when well established.
- (c) Congestion of the kidney, active and passive.

A small quantity of albumin is observed in :

(a) Slight degrees of chronic hypertrophic and atrophic nephritis and of lardaceous disease.

- (b) Chronic arterial renal sclerosis.
- (c) Congestion of the kidney, active and passive.
- (d) Febrile and toxæmic conditions, and certain nervous affections.
- (e) Physiological or functional albuminuria.
- (f) Pyuria.

The following is a summary of the diseased conditions in which albuminuria may be observed :

Albuminuria is of (A) **Non-renal** and (B) **Renal** origin.

A. Non-renal albuminuria is due to hæmaturia, pyuria, and to the admixture of non-urinary discharges.

B. Renal albuminuria comprises :

(i.) Functional or physiological albuminuria, of which a variety of forms is recognized—*e.g.*, cyclical or paroxysmal albuminuria, albuminuria of adolescents, postural or orthostatic albuminuria, dietetic albuminuria, albuminuria due to excessive exercise, to exposure to heat or to cold, etc.

(ii.) Pathological albuminuria without definite kidney lesion :

(a) Febrile albuminuria, due to slight temporary changes in the renal epithelium ('cloudy swelling').

(b) Albuminuria from morbid blood states—*e.g.*, scurvy, syphilis, gout, anæmia, chronic lead and mercurial poisoning.

(c) Albuminuria from nervous diseases—*e.g.*, epilepsy, tetanus, apoplexy, head injuries, convulsions.

- (iii.) Pathological albuminuria, with definite kidney lesion :
- (a) Congestion of the kidneys, active and passive.
  - (b) Acute nephritis.
  - (c) Chronic hypertrophic nephritis (large white kidney).
  - (d) Chronic atrophic nephritis (small white kidney).
  - (e) Chronic arterial renal sclerosis (small red kidney).
  - (f) Lardaceous disease of the kidneys.

9. **Sugar.**—A small quantity of glucose is found normally in the blood (under 0·2 per cent.). When from any cause a larger proportion exists in the blood, it makes its appearance in the urine. Indeed, normal urine contains a minute quantity of grape-sugar, but not enough to be recognized by the ordinary tests. (See Urine, Examination of, p. 526.)

Sugar having been found in the urine, we must ascertain, in the first place, if it is glucose (see Pentose, p. 524); secondly, if it is persistent; and, thirdly, if the quantity of urine is increased. If these questions are answered in the affirmative, the case is one of diabetes mellitus. The urinary condition will be as follows: quantity increased; specific gravity rises above 1030, possibly up to 1060; reaction acid; colour, pale greenish-yellow; albumin often present; odour sweetish; glucose present in varying amount, 1 or 2 per cent. being a mild degree, 6 or 8 per cent. a severe degree of the affection. These features are quite sufficient for diagnostic purposes, but in corroboration we note the excessive appetite for liquids and solids, in spite of which the patient in most cases loses flesh—the so-called *diabète maigre*. This symptom is often striking—e.g., a patient at present under observation weighs 5 stones 5 pounds; two years ago she weighed over 12 stones. A certain proportion of cases, especially individuals over middle age, do not lose much weight, but keep in fairly good condition for years (*diabète gras*). It is cases of this description which often prove a pitfall to the practitioner who omits to examine the urine. His suspicions should be aroused by the appearance of one of the customary complications of diabetes, viz., cataract, boils, carbuncle, pruritus, or peripheral neuritis.

The pathology of the affection cannot be here discussed further than to remind the reader that it originates frequently in the pancreas. Hale White shows ('Guy's Hospital Reports') that simple atrophy is the form of pancreatic disease most commonly found in diabetes. Sometimes the affection is associated with hepatic or cranial disease.

## COMPARATIVE TABLE OF SYMPTOMS IN

Symptoms.	Acute Nephritis.	Chronic Nephritis.		Chronic Arterial Renal Sclerosis.
		Hypertrophic.	Atrophic.	
<b>Urinary Symptoms:</b>				
<b>Quantity ..</b>	Decreased	Decreased	Increased (Decreased in Pale	Increased terminal stages) Pale
<b>Colour ..</b>	Dark	Pale		
<b>Density ..</b>	Increased	Somewhat diminished	Considerably diminished	Markedly diminished
<b>Blood ..</b>	Usually present, scanty	Unusual	Unusual	Unusual
<b>Casts ..</b>	Numerous (hyaline, blood, epithelial, granular, and perhaps fatty)	Numerous (hyaline, epithelial, granular, fatty, waxy)	Numerous (hyaline, epithelial, granular, fatty, waxy)	Less numerous and smaller (hyaline and granular)
<b>Albumin ..</b>	Abundant	Usually abundant (Increased in	Less abundant last stages of	Scanty or absent the affection)
<b>Urea ..</b>	Decreased	Decreased	Decreased	Decreased
<b>General Symptoms:</b>				
<b>Edema ..</b>	Early and marked	Usually well marked	Often present in moderate degree	Often absent: usually slight
<b>Arterial tension</b>	Increased	Increased	Increased	Markedly increased
<b>Cardiachypertrophy</b>	Only in prolonged cases	Present	Present	Well-marked
<b>Uræmia ..</b>	Common	Less common than in acute	Fairly common	Very common
<b>Anæmia ..</b>	Moderate	Well-marked	Well-marked	Often absent
<b>Pain (lumbar) ..</b>	Sometimes present	Absent	Absent	Absent
<b>Fever ..</b>	Sometimes present	Absent	Absent	Absent

## DISEASES OF THE URINARY ORGANS

Congestion of Kidney—Active or Passive.	Physiological Albuminuria.	Amyloid Disease o. the Kidney.	Non-Renal Albuminuria.	Diabetes Mellitus (added for the Sake of Com- parison).
Decreased	Unchanged	Increased	Unchanged	Much in- creased
High-coloured	Unchanged	Pale	Unchanged or turbid	Pale
Increased	Often dimin- ished	Diminished	Unchanged	Considerably increased
Often present	Absent	Absent	Often present	Absent
Sometimes found, usu- ally hyaline, perhaps fatty or granular	Sometimes a few hyaline	A few hyaline, or rarely waxy casts	Absent	Absent, except when ne- phritis has supervened
Often abun- dant	Scanty deposit	Often abun- dant	Scanty de- posit on fil- tration	Sometimes found in advanced cases
Decreased	Unchanged	Slightly de- creased	Unchanged	Considerably increased (sugar pre- sent)
Well-marked, of cardiac distribution	Absent	Usually pre- sent, of car- diac distri- bution	Absent	Absent
Often dimin- ished in car- diac cases. Often in- creased	Unchanged	Usually un- changed	Unchanged	Unchanged
Present in car- diac cases	Absent	Usually absent	Absent	Absent
Uncommon	Absent	Rarely ob- served	Absent	Absent
Usually absent	Usually absent	Usually pre- sent	Often present	Often present
Often present	Absent	Absent	Often present	Absent
Usually absent:	Absent	Often present	Often present	Usually absent

The temporary appearance of sugar in the urine is common to many diseases, and is not to be diagnosed as diabetes. It may be merely a passing phase in dyspepsia, gout, asthma, hysteria, epilepsy, and other affections; or it may be due to the action of certain drugs and poisons, such as chloroform, chloral, carbonic oxide, ether, antipyrin, etc. In these conditions the sugar is usually small in quantity, and the amount of urine passed is not increased.

Associated with glucose there may be found at times **Acetone, Diacetic Acid, and Hydroxybutyric Acid.** The last-named of these bodies is recognized as the cause of the acidity of the blood observed in diabetes, and to it diabetic coma is attributed. Acetone and diacetic acid are derivatives of hydroxybutyric acid, and accompany it in the urine. These bodies are not found in normal urine, and their presence is indicative of a severe form of metabolic disturbance.

10. **Pentoses.**—This group of carbohydrates differs chemically from that (hexoses) to which glucose belongs. Pentosuria may be a temporary and unimportant condition, depending upon the ingestion of large quantities of fruits which contain pentoses, such as plums, cherries, etc. They are also found to persist in the urine of certain patients, independently of the nature of the food, constituting an anomaly in metabolism, the nature of which is not well understood. At times pentose is found to accompany glucose in the urine of diabetics. Pentosuria may give rise to an erroneous diagnosis of diabetes, as several of the tests for glucose also react to pentose (see Examination of Urine, p. 537). This is of some importance, as the diet suitable for glycosuria is not appropriate for pentosuria. The clinical signs found in cases of diabetes are absent in pentosuria, where the condition is only recognized by an examination of the urine.

11. **Albumoses.**—The conditions albumosuria and peptonuria may be taken as identical. It is not certain that true peptones occur in the urine. Albumosuria alone or combined with albuminuria may occur in the puerperal period, in acute yellow atrophy of the liver, in phosphorus-poisoning, in ulceration of the stomach or intestines, in acute infectious diseases, in chronic abscesses, in phthisis, in resolving pneumonia, in empyema, and in multiple myeloid sarcoma. If the urine contains albumin as well as albumose, the diagnostic value of the latter is trifling, as it may possibly be the result of changes occurring in the albumin

effected by the chemical reagents used in the test. When found alone, and when in considerable quantity, it suggests deep-seated suppuration, and in doubtful cases it would support a diagnosis of appendicitis, abscess of the brain, empyema, etc.

12. **Bile Pigments** are not normally found in the urine. Bilirubin and sometimes biliverdin appear when any hindrance to the outflow of the bile occurs, resulting in jaundice. (See Jaundice, p. 169.)

13. **Indican**.—A small quantity of indican is normally present in the urine. When in excess it points to the absorption from the bowel of products of putrefaction; hence it may be found in constipation or obstruction of the bowels. It is also increased in putrefactive suppuration, and by a diet largely composed of animal food.

14. **Urea** is the final product of proteid metabolism in the body. The nitrogen-containing waste substances, produced by muscular activity chiefly, and by other vital processes to a less extent, are transformed, mainly by the liver, into urea. It is also derived from nitrogenous food. Its extraction from the blood by the kidneys is the principal function of these organs. Urea, then, is normally found in the urine, and is the largest constituent of the urinary solids. (See above, under Density, p. 505.)

It will be at once seen that the quantity of urea excreted must vary within wide limits in health. On an average it amounts to about 2 per cent. of the total urine passed.

Any considerable increase in the quantity of urea (the condition is termed **azoturia**) indicates (*a*) an excessive nitrogenous diet, or (*b*) an excessive destruction of the nitrogenous tissues of the body, and may be observed in fevers and in diabetes.

Decrease of urea is observed in (*a*) starvation, or when the diet is mainly non-nitrogenous; (*b*) in disease of the liver, especially acute yellow atrophy, also cancer and cirrhosis of the liver; and (*c*) in renal inadequacy, as seen in Bright's disease, in cystic kidney, and sometimes in cirrhotic kidney.

15. **Nucleo-Albumin**.—This substance resembles mucin in its physical and chemical properties. It is found in abundance in inflammations of the bladder, ureter, and kidneys; also in leukæmia. It is of little diagnostic importance, except for the fact that it may be mistaken for albumin. The heat test, however, at once distinguishes the two substances (see p. 534).

**URINE, Examination of.**

Selection of a specimen of urine—Reaction—Specific gravity—Translucency—Deposits: phosphates; urates; oxalate of lime, etc.—Micro-organisms.

Albumin: heat test; Heller's test; picric acid test; biuret reaction; quantitative examination—Albumoses.

Blood: guaiacum test; Heller's test; hæmin test; spectroscopic test.

Pus—Mucin and nucleo-albumin.

Sugar: Fehling's test; fermentation test; von Jaksch's phenylhydrazine test.

Aceto-acetic acid—Hydroxybutyric acid—Acetone—Pentoses—Chlorides—Urea—Bile: Gmelin's test—Indican—Uric acid—Ehrlich's diazo reaction.

The following description of the methods to be adopted in examining the urine is an attempt to place within reach of the reader a short but sufficient guide to the clinical and practical examination of the urinary condition. The inferences to be drawn from the conditions observed are considered in the article on Abnormalities of the Urine (p. 502).

**Selection of a Specimen of Urine.**—An accurate estimation of the state of the urine may at times necessitate the collection of all the urine passed in twenty-four hours, the sample being taken from the mixed urine. In practice it is by no means always essential to take this precaution; it is quite sufficient in most cases to obtain a specimen which has been secreted three or more hours after a meal. At times one is not able to make sure of even this interval after food, but if on examination any abnormality is observed it will then be necessary to direct the patient, so as to secure a suitable sample. In collecting a twenty-four hours' specimen it is well to add to the vessel containing the urine a few drops of chloroform, carbolic acid, or toluol, in order to obviate putrefactive changes. When pus, blood, tube casts, epithelium, or micro-organisms are suspected, the urine should be examined at once, without waiting for a twenty-four hours' specimen. Tube casts, if allowed to remain in the urine glass for more than six hours, may become changed or even dissolved, so that the examination would be fallacious. In order to avoid errors arising from the addition of vaginal or urethral discharges to the urine, it is usually necessary in the case of females to have a catheter passed, and with males to direct that the first 2 or 3 ounces passed should be rejected, and the remainder of the bladder contents



preserved for examination. Should the urine have been passed without these precautions, and found free from abnormality, it will then be unnecessary to insist on a catheter specimen. If it should be considered desirable to make a bacteriological examination, the urine must be withdrawn by means of a catheter, with strict aseptic precautions.

The various characters of the urine are then to be methodically investigated, and the results noted. The following tests will generally be found sufficient for diagnostic purposes:

**Reaction.**—Test with litmus-paper. If the blue colour of alkalinity turns red on drying it is due to ammonia. If blue paper turns red and red turns blue with the same urine, the reaction is said to be **amphoteric**, and is the effect of both acid and basic phosphates in the specimen. On rare occasions it may be desirable to have a quantitative estimation of the acidity: this may arise in cases of gout or diabetes. The method of determining the total acidity is described in the article on the Examination of the Stomach (p. 397).

**Specific Gravity.**—Average healthy limits: 1015 to 1025. The ordinary urinometer is the best means of observing the density; there are no material advantages in 'beads' and other methods. See that all bubbles on the surface of the urine and on the urinometer are removed, and that the latter floats free in the fluid without touching the sides or bottom of the vessel. Read off the mark on the scale opposite the lowest point of the meniscus, or curved surface of the fluid. If the quantity of urine available be too scanty to float the urinometer, a small-sized instrument may be obtained, or the urine may be diluted; in the latter case, the last two figures of the resulting density are multiplied by the number of times that the dilute fluid is greater than the original. Thus, if 2 ounces only of urine were obtainable, and this diluted to 4 ounces, the specific gravity being then 1005, multiply the last figures by 2, giving a density of 1020.

A rough, and not very accurate, estimate of the amount of solids in the urine is arrived at by **Trapp's method**. This consists in doubling the last two figures of the specific gravity. The result is supposed to equal the number of grammes of solids in a litre of the urine.

The specific gravity varies with the temperature. In this country the urinometer is usually graduated for a temperature of 60° F. (15° C.). If the temperature of the room (that of the

urine) be much over or under that figure, add 1 to or subtract 1 from the reading for every 3 degrees Centigrade.

**Translucency.**—Normally, urine is clear when freshly voided, but on standing a few hours a cloud of mucus is observed. The various other substances which cause an opacity of the fluid are to be examined chemically and microscopically. The urine is allowed to stand in a narrow vessel for a short time, and the resulting **deposit** is examined.

About 2 drachms of the urine containing some of the sediment is placed in a test-tube, and about 5 drops of **dilute** acetic acid added. If the deposit disappears it is **phosphates**; if not, boil. If the deposit now disappears it is **urates**; if not, take a fresh quantity in the test-tube, and add a few drops of strong nitric or hydrochloric acid. If the deposit disappears it is **oxalate of lime**; if not, place a drop of urine from the bottom of the urine glass (withdrawn by means of a pipette) on a glass slide, and cover with a thin cover-glass. This is now examined first with a low power of the microscope, and then with a high power. It is not as a rule necessary to stain the film thus prepared, but if desired to do so, a small drop of gentian violet or weak iodine solution may be placed at the edge of the cover-glass and allowed to run under. Even where there is no visible deposit in the urine, it is often necessary to make a microscopical examination. Here the urine must be left in a conical urine-glass for some hours to settle, or, preferably, it may be centrifugalized; a drop is then carefully taken from the bottom of the fluid and examined microscopically. The different substances thus discovered are described in detail in the article on Abnormalities of the Urine (p. 506).

**Micro-Organisms.**—Among the objects found in the sediment are various micro-organisms. The usual clinical method of recognizing bacteria in urine is by staining a cover-glass preparation. When such examination does not allow of a definite recognition of the bacterium, laboratory methods must be adopted, such as culture or inoculation of an animal with the suspected fluid.

The following methods of staining micro-organisms in urine will generally be found sufficient for clinical purposes:

A catheter specimen of the urine is obtained with careful aseptic precautions. As a rule, it is necessary to centrifugalize the fluid, or at any rate to give the suspended matter time to form a sediment. In some cases of bacteriuria, where the micro-organisms

form a diffuse cloudiness, it may be necessary to add alcohol, which allows the bacteria to sediment. Ammoniacal urine is usually heated on a water-bath with dilute potassium hydrate solution before centrifugalizing. Where there is a deposit of urates these may be dissolved by warming with normal saline solution.

The staining is usually effected by—(1) Dilute methylene blue; (2) Gram's method; and (3) Ziehl-Neelsen method.

In each case a cover-glass is smeared with the sediment, and then allowed to dry in the air at ordinary temperature. It may be fixed by passing it three times rapidly through a Bunsen flame.

1. The methylene blue solution is made by adding 3 or 4 drops of a 5 per cent. solution of methylene blue in alcohol to a watch-glass of water.

The cover-glass is immersed in this dilute solution for a few minutes, say two to four. It is then washed in water, dried with filter-paper, and mounted in Canada balsam.

By this method all the forms of bacteria found in urine may be stained, but it is chiefly for the detection of the gonococcus that it is employed. In chronic gonorrhœa the filaments in the urine may be stained in a similar manner.

2. Gram's method is chiefly employed to discover the pyogenic cocci, staphylococci, and streptococci. It consists in—

(i.) Prepare a solution of aniline gentian violet. This is made by adding a few drops of aniline oil to about half a test-tubeful of water, shaking thoroughly for about a minute, then filtering the emulsion through a filter-paper previously wetted with water. To a watch-glassful of this aniline water add 3 to 4 drops of a 7 per cent. solution of gentian violet in absolute alcohol. Place the cover-glass smear in this stain for two minutes.

(ii.) Transfer the cover-slip to Gram's iodine solution (consisting of iodine, 1 gramme; potassium iodide, 2 grammes; water, 300 c.c.), and leave it there for two minutes.

(iii.) Wash in 95 per cent. alcohol until no more colour comes away.

(iv.) Wash in water.

(v.) Counterstain with basic fuchsin (3 or 4 drops of 10 per cent. alcoholic solution of basic fuchsin in a watch-glassful of water); leave it there for about half a minute.

(vi.) Wash in water, dry with filter-paper, and mount in Canada balsam.

With this staining the Gram-positive bacteria (the pus-forming cocci—*i.e.*, streptococci and staphylococci and tubercle bacillus) are stained dark blue or black, while the Gram-negative bacteria (gonococcus, *Bacillus typhosus*, *Bacillus coli communis*) are stained red.

3. Ziehl-Neelsen method:

(i.) The dried and fixed smear on the cover-slip is placed in a watch-glass of carbol fuchsin (fuchsin, 1 gramme; alcohol, 10 grammes; 5 per cent. watery solution of carbolic acid to 100 c.c.), and heated over a Bunsen flame or spirit-lamp carefully till it steams well. Leave it there for two minutes

(ii.) Wash in water.

(iii.) Dip into a 20 per cent. solution of nitric acid, and remove it in three to five seconds.

(iv.) Wash in water.

(v.) Wash in 60 per cent. alcohol till the red colour is lost.

(vi.) Wash in water.

(vii.) Counterstain with dilute methylene blue [see (1) above].

(viii.) Wash, dry, and mount in Canada balsam.

This method is intended to detect tubercle bacilli, and will be sufficient in cases where the bacilli are numerous. When, however, the smegma bacillus is present a definite diagnosis cannot be made without the performance of an inoculation experiment.

A convenient size of watch-glass for this work is about 2 to 2½ inches in diameter.

The Ziehl-Neelsen method may be somewhat simplified by pouring a few drops of the carbol fuchsin solution on to the cover-slip itself, held (by means of forceps) with the smear side uppermost. It may thus be heated by holding the cover-slip over the flame, and keeping it heated for two minutes.

The micro-organisms which may be found in the urine are—

1. Gonococcus, occurring as minute rounded or oval bodies, often in pairs with the contiguous surfaces flattened, or in groups and chains, and are chiefly found in the interior of pus cells. It differs from the pus-forming cocci in not retaining its stain by Gram's method. It is chiefly found in the urethral discharge and less frequently in the urine of cystitis cases.

2. Tubercle bacillus is seen as short beaded rods staining red by the Ziehl-Neelsen method. They are also stained by Gram's method, in which respect they differ from the smegma bacillus, from which they are with difficulty distinguished.

3. Streptococci and staphylococci occur as minute rounded organisms in chains or in groups, staining with Gram's method.

4. *Bacillus typhosus* (Eberth), a short, rod-like form with somewhat oval ends, staining in weak carbol fuchsin and other stains, but not in Gram's. It is often found in the urine at some period of typhoid fever, and may be occasionally found a long time after the fever has disappeared. The bacillus is difficult to discover, and its identification in the urine (or faeces) is not practicable as a routine clinical investigation, but may be reserved for the laboratory.

5. *Bacillus coli communis* resembles closely that of typhoid fever in appearance and in staining qualities. It is the chief organism present normally in the small intestine, and is also found in the large bowel. It occurs in the urine, which is acid, and may be the cause of cystitis and pyelitis. It must be borne in mind that the urine rapidly loses its acidity on standing in a vessel.

Other organisms of little diagnostic importance are to be found in the urine—*e.g.*, *Bacillus proteus vulgaris*, *Bacillus pyocyaneus*, etc. In the article on Abnormalities of the Urine the clinical significance of the different organisms is further considered.

**Albumin.**—The form in which proteids are most frequently found in the urine is serum albumin, which is often accompanied by globulin. The clinical significance of both bodies is the same (see p. 513). The following tests are sufficient :

1. **Heat.**—If the urine be turbid, filter; if alkaline or neutral, add 3 or 4 drops of dilute acetic acid. Heat up to boiling-point in a test-tube. If a white precipitate forms it is serum albumin or globulin. If only a trace of albumin be present, hold the test-tube against a black background, with a good light shining straight on the tube and background. Note that excessive acidity and alkalinity interfere with this test, the albumin being thereby changed to acid albumin or alkali albumin, which are not precipitated by heat.

2. **Nitric Acid (Heller's Test).**—Place about a drachm of strong nitric acid in a test-tube; hold the test-tube as nearly horizontal as possible without spilling the acid, and very gently pour down the sloping surface of the glass (preferably by means of a pipette) about 2 drachms of urine. Now carefully replace the tube in a vertical position. If albumin be present a precipitate will be formed; if only a very small amount of albumin be present the test-tube may have to stand for some minutes before

the cloud appears. The precipitate occurs as a disc of varying thickness and apparent density just at the junction of the two fluids—the so-called **gun-wad** precipitate.

**NOTE.**—**Mucin** is precipitated in this test as a haze, found chiefly towards the upper part of the layer of urine, and it does not disappear on boiling. **Hetero-albumose** also gives a similar cloud, which dissolves on heating and reappears on cooling. **Uric acid** and **urea** are crystallized as nitrate of urea in concentrated urine. Dilute the urine and again test, when no urea nitrate is thrown down. **Balsams**—*e.g.*, copaiba or turpentine in the urine (after administration by the mouth) are precipitated as a cloud with nitric acid. It clears up to some extent on heating, or it may be dissolved by an excess of ether.

3. **Picric Acid.**—Equal parts of urine and of a saturated watery solution of picric acid are placed in a test-tube. Albumin produces a whitish-yellow cloud, which intensifies on heating. This is a very delicate test, and detects a minute quantity of albumin.

**NOTE.**—A similar haze is caused if the urine contains alkaloids, such as quinine, also antipyrin, albumoses, and nucleo-albumin. In these cases the precipitate dissolves on heating and reappears on cooling.

4. **Biuret Reaction.**—To 1 drachm of urine add a considerable excess of caustic soda solution and 1 or 2 drops of a weak solution of sulphate of copper. If serum albumin or globulin be present a violet colour develops. If there be albumose the colour becomes rose-pink.

It is rarely necessary for diagnostic purposes to distinguish the different proteids which may at the same time be present.

The **quantity** of albumin in any specimen of urine may be roughly estimated by boiling and allowing the precipitate to settle for six or eight hours. The depth of the sediment may then be expressed as a fraction of the total height of fluid in the tube. Thus one may say there is a mere trace of albumin present, or albumin is one-sixth, one-half, or solid. A better method is to use **Esbach's albuminometer**, a graduated tube, in which the albumin, precipitated by picric acid, is allowed to settle for about twelve hours. The marking on the tube opposite the upper level of the deposit corresponds to the number of grammes of dried albumin present in a litre of urine. For example, if the sediment reaches to the line marked 4 on the Esbach tube, the

urine contains 4 grammes per litre—i.e., 4 parts in 1,000, or 0.4 per cent.—of dried albumin.

**Albumoses.**—Hetero- and deuterio-albumoses may occur, and are of doubtful diagnostic value. They may be recognized by the appearance of a precipitate with nitric and picric acids, which dissolves on heating and reappears on cooling (see above, p. 532). They are also precipitated by adding an equal quantity of a saturated solution of sodium chloride and a few drops of dilute acetic acid. They may, in addition, be distinguished by the biuret reaction (see above, p. 532).

**Blood.**—The urine may be very slightly or not at all changed in appearance if there be only a small quantity of blood present. With increasing quantities the urine is less translucent, is dusky, smoky, dark brownish-red, or porter-coloured. When the blood-corpuscles can be recognized by the microscope, the condition is termed *hæmaturia*. When, however, the chemical tests indicate hæmoglobin, but the microscope fails to recognize blood, the condition is known as *hæmoglobinuria*. The following tests may be used:

1. **Guaiacum Test.**—A few drops of freshly prepared tincture of guaiacum and a couple of drachms of ozonic ether are mixed in a test-tube. Pour gently (preferably by means of a pipette) a drachm or two of urine which has been previously acidified down the sloped side of the tube on to the surface of the guaiacum and ether. If hæmoglobin is present, a blue colour forms at the surfaces of contact.

2. **Heller's Test.**—Alkalinize the urine with a little liquor potassæ. A red colour or a red precipitate results, consisting of phosphates with blood pigment adhering.

3. **Hæmin Test.**—A little of the suspected sediment is placed on a glass slide, with a very small crystal or two of common salt. A cover-glass is placed over all, and a drop of glacial acetic acid is run under the cover-glass. Warm gently over a small flame, allow it to cool, and examine under the microscope. If blood be present, the mahogany-red rhombic crystals of hæmin will be found.

4. **Spectroscope.**—As a corroboration the absorption bands of blood pigment may be identified in the spectrum. A small pocket spectroscope can be obtained. A solution in water of the deposit obtained by Heller's blood test, or the suspected urine itself, when examined shows the bands.

**Pus.**—In addition to the microscopic examination, which is the most effective means of distinguishing pus, it may be recognized by its behaviour in an alkaline medium. In alkaline urine, or in urine rendered alkaline by the addition of liquor potassæ, it becomes ropy and coagulated. If a large quantity of pus be present, albumin will be found, even if the corpuscles be filtered out and if the kidneys be free from disease.

**Mucin and Nucleo-Albumin** are sometimes mistaken for albumin. They are precipitated by strong nitric acid in the contact method (see p. 532), but the precipitate is confined to the upper layers of the urine, the substances being redissolved by the excess of acid in the lower portions of the urine. They are unaffected by heat. Both bodies are precipitated by glacial acetic acid, especially when the urine is diluted, and both are slightly dissolved again by an excess of the acetic acid, mucin less so than nucleo-albumin. Nucleo-albumin is precipitated by picric acid, but mucin is not.

**Sugar.**—Glucose is the carbohydrate which is of most diagnostic interest in urinary examination. It exists in the normal urine, but in quantities too small to be recognizable by the ordinary clinical tests. Of the numerous tests in use, the following may be recommended as the most suitable for the practitioner :

1. **Fehling's Test.**—This test depends on the capacity possessed by glucose for reducing soluble cupric salts in hot alkaline solution to insoluble cuprous forms. The reagent is to be kept in two separate bottles, one containing a solution of cupric sulphate in water, the other a solution of Rochelle salts and caustic soda in water. An equal quantity of each fluid (say about a drachm), is taken in a test-tube and brought to boiling-point. If the reagent is not injured by chemical decomposition (to which it is very liable), the blue colour remains bright and without deposit. Now add a few drops of urine to the test-tube and heat again, but do not boil it too much. Proceed in this manner until a quantity of urine equal to that of the reagent has been added, and, if no change in colour and no precipitate appears, sugar is absent. If, however, on adding the urine the blue colour of the Fehling's solution be discharged, or if a yellowish-orange precipitate of cuprous oxide forms, glucose is probably present.

The chief defect of this test is the fact that a similar reduction



of the copper reagent is effected by urates when in excess in the urine, by uric acid, and by glycuronic acid. These substances can be precipitated by about one-fourth the bulk of urine of a hot 10 per cent. solution of acetate of lead, which does not precipitate sugar.

The quantity of sugar present can be determined by this test. Fehling's solution is made of such portions that all the cupric salt contained in 1 c.c. is reduced by 0.005 gramme of glucose. Hence, by noting the quantity of urine necessary to completely decolorize a given quantity of Fehling's solution, one knows the amount of glucose in the quantity of urine in question, and it is a simple calculation to arrive at the percentage of glucose in the urine, and so at a knowledge of the total amount excreted in a given time. The following example will explain the method:

Take 5 c.c. of each of the portions of Fehling's solution, mix them in a flask, and dilute with water to 50 c.c. The urine to be examined should have been previously boiled and filtered, in order to remove any possible albumin. Of the urine take 10 c.c. and dilute it with water to 100 c.c. Bring the Fehling's solution to boiling-point, and keep it gently boiling while the urine is slowly poured into it by drops from a burette. Remove the flame at intervals for a few seconds. As soon as the solution is decolorized (a difficult point to determine), and an abundant red precipitate has appeared, the quantity of diluted urine which has been expended is noted. Let us say, for example, that this amounted to 15 c.c. Then, since 10 c.c. of Fehling's solution were used, the 15 c.c. of dilute urine contain 0.05 gramme of glucose. As the urine was diluted ten times, 1.5 c.c. of urine contain 0.05 gramme of glucose; therefore 100 c.c. of urine contain  $\frac{0.05 \times 100}{1.5} = \frac{5}{1.5} = 3.3$ —that is, the specimen of urine contains 3.3 per cent. of glucose. The quantity of urine passed in twenty-four hours being known, it is now seen at once how much sugar is being passed in the day.

**2. Fermentation Test.**—This is a reliable test, but it requires several hours to complete it, and it is not always easy to procure active yeast free from starch. The urine must be made acid. Fill the closed graduated limb of a Doremus ureometer with the urine, in which a piece of yeast about the size of a pea has been dissolved. Leave it in a warm place—*e.g.*, on a mantelpiece over a fire—for about twelve hours. If any gas has accumulated

at the upper closed end of the tube, sugar had been present. While this specimen of urine is fermenting, two control experiments should at the same time be performed. One tube should be prepared with water, sugar, and yeast, in order to verify the activity of the yeast; another tube should contain the urine alone, without any yeast, to demonstrate the absence of gas-forming bacteria.

It is found that the specific gravity of sugar-containing urine is reduced by fermentation. For every degree of density lost the urine contains approximately 1 grain of sugar to the ounce: for example, a sample of urine before fermentation was 1038; after fermentation the specific gravity was only 1022. The difference is 16; therefore the urine contained about 16 grains of sugar to each ounce of urine.

The above two tests are generally sufficient, but if additional corroboration be required, the following is a delicate means of detecting even minute quantities of sugar:

**Phenyl-hydrazine Test** (von Jaksch).—Take 2 or 3 drachms of urine free from albumin in a test-tube; add 7 or 8 grains (as much as will lie on the point of a penknife) of phenylhydrazine hydrochloride, and about twice as much sodium acetate. Place the test-tube in a beaker of boiling water and boil for half an hour. Allow it to cool slowly, and if glucose be present a yellowish precipitate of phenyl-glucosazone appears. On examining under the microscope, this is seen to consist of needle-like crystals, arranged in sheaves, bundles, stars, etc. Sometimes the precipitate is amorphous; it must then be dissolved in hot alcohol, diluted with water, and the alcohol boiled off, when the crystals will be found.

A simplification (by Kowarsky) of this method may be preferred: 5 c.c. of urine are taken in a test-tube, to which are added 5 drops of pure phenyl-hydrazine, 10 drops of glacial acetic acid, and 1 c.c. of a saturated solution of sodium chloride. Boil for two minutes, cool gradually, and examine for the characteristic crystals.

**Aceto-Acetic Acid (Diacetic Acid).**—This acid does not occur in normal urine, but is found along with acetone. It is probably of more diagnostic importance than acetone.

**Test:** Add a few drops of dilute ferric chloride solution to fresh urine, and a precipitate of ferric phosphate appears. Continue adding ferric chloride till no more precipitate falls,

filter, and add a few more drops of the ferric chloride solution. A violet-red colour is produced when aceto-acetic acid is present.

This reaction only occurs if the urine has not been previously boiled. Antipyrin, salicylates, and carbolic acid also give the reaction, whether the urine has been boiled or not.

**Hydroxybutyric Acid ( $\beta$ -Oxybutyric Acid)**, when present, is invariably accompanied by aceto-acetic acid. There is no simple test for hydroxybutyric acid, so that the recognition of acetone is all that need be attempted.

**Acetone—Legal's Test.**—Add to the urine a few drops of caustic soda solution to alkalize; add a solution (0.1 gramme to 15 c.c. of water) of nitro-prusside of soda. If acetone is present, a ruby-red colour results. Acidify with acetic acid, and the colour darkens to a violet.

**Pentoses.**—These carbohydrates may be found in the urine with or without glucose (see p. 524). They respond to the reduction tests (Fehling's, etc.), and to the phenyl-hydrazine test, but (like glycuronic acid) they do not ferment.

**Bial's Orcin Test.**—Make a solution of 500 c.c. of 30 per cent. hydrochloric acid, 1 gramme of orcin, 25 drops of a 10 per cent. solution of ferric chloride. Boil 5 c.c. of the reagent, add a few drops, not exceeding 1 c.c., of urine. If pentoses be present, a green colour appears.

**Chlorides—Mohr's Method.**—Make two solutions—

1. Silver nitrate, 29.042 grammes; water, 1,000 grammes.
2. Potassium chromate, a 10 per cent. solution.

Take 10 c.c. of urine and dilute with 30 to 50 c.c. of distilled water. Add 2 or 3 drops of the chromate solution. Run in the silver nitrate solution from a burette until a red colour is obtained, remaining permanently in solution. Each c.c. of the silver solution used represents 0.01 gramme of sodium chloride. A deduction of 1 c.c. from the quantity of silver solution used should be made to allow for substances other than chlorides present in the urine which unite with the silver before the chromate has secured them.

**Urea.**—It is necessary to ascertain the quantity of urea present, as it is a constituent of normal urine. All the urine passed in twenty-four hours is collected in a large vessel, and the specimen for examination is taken from the mixture. The quantity passed is also to be noted. The amount of nitrogen gas given off as a result of decomposition of the urea by the action of an alkaline solution of sodium hypobromite is observed. This solution is

composed of 2 c.c. of bromine in 23 c.c. of a 40 per cent. solution of caustic soda. By the complete decomposition of the urea present, nitrogen to the volume of 35.4 c.c. to each 0.1 gramme of urea is evolved. The gas is measured in any convenient apparatus—Hind's modification of Doremus's ureometer is the most convenient. The hypobromite solution should be prepared fresh when required, capsules containing the necessary quantity of bromine being obtainable for the purpose.

**Bile.**—The presence of bile in the urine gives a colour of varying degrees of dark yellow to brown or green, with yellow froth. It stains linen yellow.

**Gmelin's Test.**—Filter the urine through white filter-paper; with a glass rod place on the stained paper a drop of fuming nitric acid (*i.e.*, nitric acid which has been exposed to light for some time, or which is partly reduced by the addition of a few crystals of cane-sugar). A play of colours surrounds the drop of nitric acid. The colours are due to the oxidation of the bile pigments bilirubin and biliverdin, and occur in this order: green, blue, violet-red, yellow.

**Indican.**—This body, found normally in the urine in small amount, is the indoxyl-sulphate of potash, and is detected by decomposing it with strong hydrochloric acid, releasing indoxyl. The latter is then oxidized by calcium hypochlorite, producing a blue colour.

**Jaffe's Test.**—To about 2 drachms of urine add an equal volume of strong hydrochloric acid; now add a few drops of a 1 in 20 solution of calcium hypochlorite till a blue colour, due to indigo blue, appears. Add about a drachm of chloroform and shake thoroughly; the chloroform then sinks to the bottom coloured blue.

**Uric Acid.**—In addition to microscopical examination, uric acid is detected by the murexide test, which also demonstrates urates. Place a little concentrated urine or deposit in a porcelain capsule; add a few drops of dilute nitric acid and evaporate to dryness—a yellowish residue is found; add a drop or two of ammonia, or expose the residue to the fumes of ammonia—a violet colour is produced; if a drop or two of caustic potash solution is now added, the colour becomes more blue in shade.

**Ehrlich's Diazo-Reaction.**—For the clinical significance of this reaction and the methods to be employed, see the article on the subject (p. 122).

**URTICARIAL ERUPTIONS.** See **Skin Eruptions**, p. 371.

**UTERINE REFLEX.**

This reflex act consists in the involuntary contractions of the muscular fibres of the uterus in response to peripheral stimulation. The stimulus may be applied to the womb, or may come from some other peripheral organ or region—*e.g.*, the breast. (See **Reflexes**, p. 345.)

**VALSALVA'S EXPERIMENT.**

The act of making a forced expiratory effort while the glottis is kept closed. By this procedure the heart's beat is rendered somewhat more frequent, and the passage of blood from the veins to the auricles is impeded (see pp. 443 and 447).

**VASOMOTOR REFLEX.**

A series of reflexes governs the blood supply to the different regions of the body. They are described at p. 346.

**VENOUS HUM (Bruit de Diable, Humming-Top Murmur, Nun's Murmur).**

A murmur which may be heard at the base of the neck in anæmic persons. It may be heard at times in healthy children, and in cases of exophthalmic goitre (see p. 444).

**VERTIGO.**

A sensation of instability. The power to maintain the balance is impaired or lost, and the patient experiences illusory movements, either of his own body or of surrounding objects, or of both. His gait, if he attempts to walk, is reeling, and he may fall if not supported. Vomiting or a sensation of nausea is a common accompaniment of these symptoms.

The maintenance of equilibrium is accomplished by the due co-ordination and regulation of the muscular contractions of the body, which automatically respond to afferent nerve impulses passing to the cerebral cortex from various peripheral organs and regions. These consist of: (1) the internal ear; (2) the retina; (3) the peripheral terminations of muscular sensory nerves; and

(4) the peripheral terminations of cutaneous sensory nerves. These nerve impulses pass from the periphery directly in the majority of instances to the cerebellum, whence the co-ordinated afferent impulses are transferred to the appropriate regions of the motor cortex, and possibly in part directly to the motor nerve nuclei in the anterior cornua of the cord. In consequence of this stimulation, motor impulses are being continually issued to the muscles in the periphery, whereby in a state of health the co-ordinated movements and the equilibrium of the body are perfectly maintained. It will thus be easily understood that any defect, either in the direction of defective or excessive afferent impulses, or in the cerebellar co-ordination of these impulses, will result in (among other defects) loss of equilibrium. The subject is further discussed in the articles on Inco-ordination (p. 245), and on Reflexes (p. 334).

While it is true that disturbances of any of the above-named afferent impulses may operate in the production of loss of balance, it is mainly those emanating from the internal ear or labyrinth that are responsible for producing the combination of symptoms comprised in the term 'vertigo.' It is by means of impressions originating in the semicircular canals, and conveyed thence to the cerebellum by the vestibular nerves, that the position of the body in space is made known to the consciousness of the subject. Any interference with this function will cause a conflict of impressions, which results in illusions as to the position of the body in relation to surrounding objects. A majority of the cases of vertigo are, then, to be attributed to such an interference, operating either directly or indirectly on the semicircular canals, or their connections with the cerebellum; thus, we have familiar instances in the vertigo caused by the pressure of accumulated wax in the external auditory meatus, or by its removal, and in the typical vertigo of Ménière's disease, which is recognized as due to active stimulation of the semicircular canals. The vertigo of arterio-sclerosis and of other circulatory defects—*e.g.*, heart disease, syncope, alcoholic intoxication, digestive disturbances—are probably the result of improperly controlled changes of blood pressure, which produce their effect on the fluids of the labyrinth. Similar circulatory irregularities account for the vertigo of neurasthenia.

An interference such as that just referred to, instead of involving the labyrinth or its nervous connection with the cerebellum, may

operate directly on the latter. The frequently occurring instances of **cerebellar ataxia** are examples of disturbed equilibrium, through lesion of the cerebellar portion of the nerve structures which preside over this function. The reeling gait resulting from cerebellar tumour, abscess, hæmorrhage, or other injury, especially if it involve the vermis, is the visible evidence of vertigo. (See Gait, p. 150.)

Sight also plays an important part in the maintenance of equilibrium; its relation to co-ordination of muscular movements is referred to at p. 246, and its connection with vertigo is even more marked. The act of directing the eyes toward any point in the field of vision is to a certain extent a reflex act, influenced by the judgment one forms as to the position of the body in relation to external objects. Should that judgment be vitiated by lesions of, say, the semicircular canals, the **innervation** or **strength sense** controlling the movements of the eyeballs is at fault, and visual impressions are in consequence misplaced on the retina. The involuntary attempts to follow the displaced images with the eyes gives the illusory sensation of moving objects, the so-called **objective vertigo**, as distinguished from **subjective vertigo**, in which the subject feels as if he himself were in motion. The vertigo produced by spinning movements of the body, both during the movements and on their sudden cessation, or by the sudden stopping of the body in the course of swift movement—*e.g.*, the sudden stoppage of a train, which may give rise to a degree of vertigo in susceptible persons—is accounted for in a somewhat similar manner. The disturbance of the semicircular canals by the constantly changing position of the body causes an automatic attempt on the part of the oculo-motor apparatus to fix the objects as they pass across the field of vision. If this attempt is successful, no special inconvenience ensues during the continuance of the movement of the body. If, on the other hand, it is unsuccessful, surrounding objects appear to be in motion, causing giddiness (objective vertigo). As soon, however, as the swift movement of the body is brought to an end, there is a sudden interruption in the more or less complete accommodation of the balancing mechanism and of the oculo-motor co-ordination, to the violent disturbance of the semicircular canals. The oculo-motor apparatus continues to make allowance for movements which have now ceased, and until a sufficient time has elapsed to enable it to adjust itself to the altered circumstances, the faulty retinal stimu-

lation causes the illusion of movement in surrounding objects and in one's own body.

The difficulty in adjusting the eye movements to objects which are in motion, or which are looked at from an unusual position, may be sufficient to cause vertigo in a susceptible individual, more especially if some emotion is introduced. A combination of fear, with the altered perspective, gives rise to the vertigo produced by looking from a height; merely looking at moving or revolving objects—*e.g.*, machinery—may be enough to cause vertigo in some individuals. Sea-sickness and the vertigo produced by swinging are mainly caused by disturbance of the semicircular canals. The vomiting centre is especially irritated by the violence of the disturbing movements, and the visual phenomena (objective vertigo) depend on the interference in the labyrinth from the same conditions as those referred to above, in speaking of the vertigo following whirling or spinning movements of the body. No doubt the retinal impressions of really moving objects are factors in the production of this form of vertigo, but they are quite subsidiary to the irritation of the semicircular canals.

Oculo-motor paralysis, by giving rise to misplaced retinal images, may also cause objective vertigo.

The ataxia and vertigo due to cerebellar disease frequently exhibits this peculiarity—that it disappears or grows considerably less while the patient is in the recumbent position, with the head at rest.

Disturbances of the cutaneous and muscular sensory nerves, while they commonly cause inco-ordination, are less frequently the origin of vertigo.

**Summary.**—Vertigo is mainly the result of disturbance of the semicircular canals and of the cerebellum. Ocular disturbances and interruptions to the passage of impulses by the muscular and cutaneous afferent nerves have also an influence in the production of the symptom. It is typically found in Ménière's disease; is seen in less intense form in digestive disturbances, especially when accompanied by flatulence; in arterio-sclerosis; in affections of the external ear causing pressure; in heart disease; in cerebellar disease; in cerebral lesions; in sea-sickness; in visual and emotional disturbances; as a result of spinning or swift movements of the body; and in alcoholic intoxication.



**VESICULAR BREATHING.**

The normal rustling sound heard on listening over the lung at some distance from the larger bronchi—*e.g.*, in the infra-axillary region. As heard, the inspiratory sound is about three times as long as the expiratory portion, though the act of expiration really occupies more time than that of inspiration. The nature and origin of the sound are considered at p. 408.

**VESICULAR ERUPTIONS.** See **Skin Eruptions**, p. 370.

**VISION, Disturbances of.**

Defective sight: minor disturbances of vision; diminished acuteness of vision. Defects of the field of vision: methods of examination; vision nulle and vision obscure; scotoma; hemianopsia; amaurosis; contracted field of vision.

Ophthalmoscopic appearance: optic neuritis; papillitis; optic atrophy; retinitis and neuro-retinitis; cataract; tubercle of the choroid.

In the following pages the evidence obtained by an examination of the pupils is not discussed, as it is of sufficient diagnostic importance to demand separate consideration. (See **Pupils**, p. 316.)

Defective sight may be the result of malformations or diseases of the eye itself, which cannot be suitably considered in a work of this description, and for which the reader is referred to the special treatises on eye affections.

1. **Various Minor Disturbances of Vision** may be complained of. A **yellow discoloration** of all objects is often perceived by patients suffering from jaundice, and by those who have been taking **santonin** internally. **Red vision** sometimes occurs in neurasthenia. Floating spots and nodular threads (*muscæ volitantes*) may be seen by persons suffering from digestive disturbance, hysteria, cardiac hypertrophy, cerebral anæmia and hyperæmia. **Flashes of light** occur in acute indigestion, in migraine, and they may form the aura of epilepsy. Patches of obscurity surrounded by a bright margin (**glittering scotomata**) are seen in migraine, and in irritative lesions of the cortex and of the cerebral meninges.

2. **The Acuteness of the Patient's Vision** must be investigated. This may be fairly satisfactorily ascertained by noting the degree of accuracy with which he can describe the contents of a bookcase or the subject of a picture at the opposite side of the

room. More accurate results are obtained by the use of Snellen's test types, displayed in a good light at a distance of 6 metres. Near vision is tested by directing the patient to read printed matter, observing the distance at which small print can be most easily distinguished. By these means defects of refraction, perception, and accommodation may be detected. Disorders of accommodation are considered in the section on the Pupil (p. 323).

In addition to local affections of the cornea, lens, and retina, atrophy of the optic nerve always, and optic neuritis sometimes, cause defective vision. These and other intra-ocular affections associated with disease elsewhere are considered below with the ophthalmoscopic examination (p. 550).

3. **Field of Vision.**—Defects of the field of vision are highly instructive. By this term is understood that space or area within which, the eye under examination being fixed upon any point and the other eye being closed, white objects can be distinguished. It extends on the temporal side of the eye for a little more than 90 degrees, and on the nasal side for almost 50 degrees. A normal eye can perceive an object at any part of this area, except at one spot, the 'blind spot,' 15 degrees to the outside and a little below the point upon which the gaze is fixed (the 'point of fixation'). From this blind spot rays of light passing through the pupil fall upon that portion of the retina occupied by the entering optic fibres, and in which the outer retinal layers are wanting. In the peripheral portions of the field of vision perception is much less acute than in the central regions, and this is especially so if the object be coloured. Certain colours are less easily perceived than others, green being the most difficult to see in the peripheral regions of the field of vision and blue the easiest.

In certain morbid conditions there may be blank areas in the field—that is to say, the corresponding portions of the retina may fail to transmit to the visual centre the stimuli received from impinging light rays. Of this failure the patient may be unaware, and only on examination is it brought to his and the observer's knowledge. The most accurate information on this subject is obtained by means of the perimeter, an instrument consisting of a semicircular metal band, graduated on its convex surface, commencing with 0 at its middle point, and rising to 90 degrees at each extremity; it is pivoted at its middle point, and by turning on that pivot it describes an imaginary hemisphere. The patient's eye occupies the centre of the hemisphere, and the fixation-point

is the pivot upon which the semicircle turns. A disc of white paper  $\frac{1}{4}$  inch in diameter is then slowly moved from the centre along the concavity of the horizontally fixed semicircle; the moment the disc disappears from the patient's sight its position is noted by means of the graduation, and marked on a chart. This is done in several different meridians with each eye, and the points marked on the chart are joined by a line, and so a figure enclosing the field of vision is formed.

A less accurate method of examining the field of vision, but one which yields sufficient information for many purposes, is the following, practised by the late Dr. W. A. McKeown:

The patient and examiner sit opposite each other, about an arm's length separating their faces; the patient's right eye and the examiner's left eye are closed; the two seeing eyes are steadily fixed on each other; the examiner's right forefinger is held vertically midway between his own and the patient's eyes, and is slowly moved to the right at an equal distance from each eye; the same movement of the finger is executed in several lines radiating from the point midway between the eyes. In every movement the finger should be lost sight of by both eyes at the same moment if the respective fields of vision are normal.

Defects of the field of vision may be unnoticed by the patient before his examination; he is in some cases no more inconvenienced by the blind area than he is by the physiological blind spot. This is the condition known as 'vision nulle,' and is to be distinguished from 'vision obscure' (Dufour), a darkened or indistinct vision which gives the patient cause for complaint. The former is usually the result of a lesion of the cortical centre of vision, while the latter depends upon a lesion lower in the visual path.

In cases where an imperfect field of vision is found the defective area is termed a **scotoma** (Gr. *σκότις*, darkness), which may be **peripheral**, **central**, **temporal**, or **nasal**, according to its position in the field of vision.

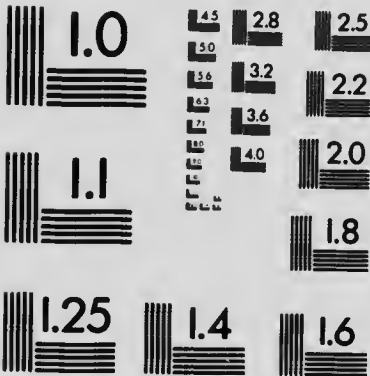
(a) **Hemianopsia**.—The most important defect in the field of vision is that in which one half of the field is blind; this is termed **hemianopsia**, and may occur in one or both eyes. The lesion causing it may be situated in any portion of the optic path, but, as will be seen by the accompanying diagram (Fig. 72), the effect varies according to the site of the interruption.

The commonest form of hemianopsia is that in which both eyes are affected; the defective halves of the fields of vision are **both on**



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the same side of the median line, and the boundary between the seeing and the blind halves of the retina is a vertical line drawn

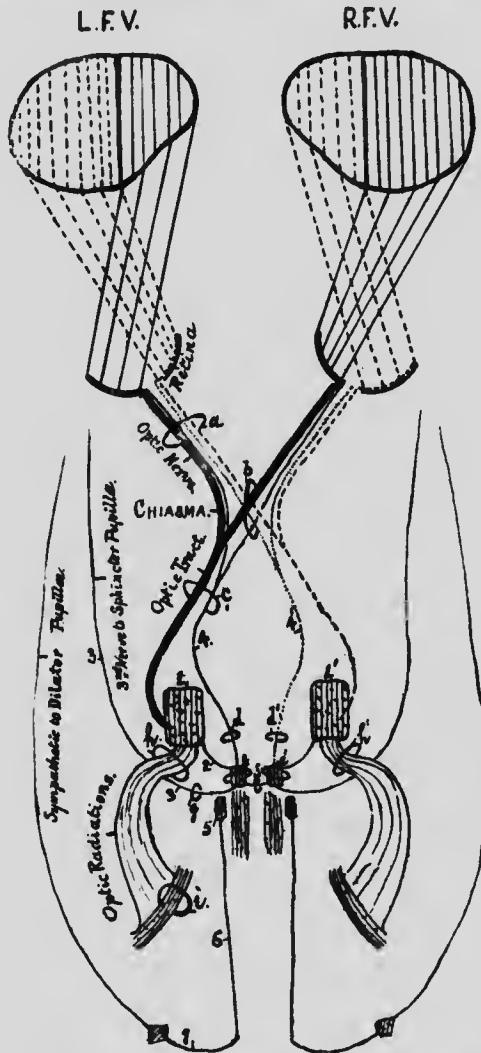


FIG. 72.—DIAGRAMMATIC REPRESENTATION OF THE VISUAL AND LIGHT REFLEX MECHANISM.

1, The primary optic nuclei; 2, Meynert's fibres; 3, motor fibres to the sphincter pupillæ; 4, afferent fibres to the iris nucleus; 5, 6, 7, nucleus and fibres of sympathetic to dilator pupillæ.

Situation of possible lesions: *a*, in optic nerve; *b*, in chiasma; *c*, in optic tract; *d*, in afferent fibres to iris centre; *e*, the iris nucleus; *f*, on the communicating fibres between the two iris centres; *g*, on third nerve; *h*, the internal capsule; *i*, the occipital cortex.

L.F.V., Left field of vision; R.F.V., right field of vision.

through the macula lutea. This is termed **homonymous hemianopsia** (Gr. *ὁμός*, the same; *ὄμμα*, a name; *ἡμιος*, half; *άν*, privative; *ὤψ*, the eye), the prefix left being added if the left half of the **field** (not the retina) is wanting, while absence of the right half of the field is named right homonymous hemianopsia. Owing to the partial decussation of the optic fibres at the chiasma, it will be seen by reference to Fig. 72 that this symptom will be produced by a destructive lesion in the cortical centre in the occipital lobe (*i*), in the optic radiations, in the posterior part of the internal capsule (*h*), in the primary optic nuclei (superior corpora quadrigemina, external geniculate bodies, and pulvinar of the optic thalamus), and in the optic tracts (*c*). **Bitemporal hemianopsia** is the loss of vision in the outer or temporal halves of each field (loss of function of the inner or nasal halves of each retina), caused by a lesion immediately in front of or behind the chiasma, or dividing it sagittally (*b*, Fig. 72), whereby the fibres proceeding to the inner halves of the retinae are interrupted. Owing to the limited area in which this lesion can occur, this form of hemianopsia is much rarer than the first mentioned.

**Unilateral and bilateral nasal hemianopsia** are such rare and improbable occurrences that a mere reference to them will suffice. They would be caused by a unilateral or bilateral lesion interrupting the laterally placed fibres in the chiasma, whereby the temporal half of the retina becomes blind.

Equally rare are **superior and inferior or altitudinal hemianopsia**, in which the upper or lower half of the field is absent.

Loss of one half of the field of vision, then, indicates a nuclear, a supranuclear, and an infranuclear lesion (to borrow the nomenclature of motor lesions) of the visual path. In order to localize the injury the following points may be of assistance:

(i.) Absence of hemiplegia, of paralysis of the cranial nerves, and of motor aphasia indicates a lesion of the visual path, which leaves the motor **tract** unaffected. This will most probably be found in the occipital lobe (*i*, Fig. 72), though even here lesions sometimes give rise to motor disturbances.

(ii.) The absence of sight in one half of the retina, without any sense of obscurity or darkness (*vision nulle*), also points to the visual centre in the occipital lobe as the probable site of the lesion, the blindness being more mental than physical.

(iii.) The absence of the pupil reflex when the blind half of the retina alone is stimulated by light, and its presence when the

sensitive half is stimulated, indicates a lesion interrupting the afferent visual and light-reflex fibres. As only one half of the retina is blind the lesion cannot be in front of the chiasma, nor can it be above the iris nucleus; it is, therefore, situated in the optic tract (*c*, Fig. 72). We are here assuming that the centripetal light-reflex fibres are distinct from the visual fibres, and that they leave the optic tract a short distance behind the chiasma. The demonstration of this immobility of the pupil in hemianopsia, known as **Wernicke's hemiopic pupillary immobility**, is no easy matter, and its absence need not prejudice the localizing diagnosis. The subject is more fully considered in the article on the Pupil, at p. 316 *et seq.*

(iv.) If the hemianopsia be bitemporal, the inner half of each retina being insensitive, the lesion must be in the chiasma (at *b*, Fig. 72).

(v.) The absence of the visual forms of aphasia (*q.v.*, p. 374), such as alexia, optic amnesia, etc., together with hemianopsia and symptoms of a lesion of base of the brain, point to the optic tract as the probable site of the interruption.

The diseased conditions giving rise to these lesions are usually the organic brain affections referred to elsewhere, such as hæmorrhage, thrombosis, embolism, meningitis, tumours, fractures of the base of the skull, abscess, etc. In some affections which produce functional disturbances of the nervous system without known anatomical lesions hemianopsia may occasionally be observed—namely, in hysteria, epilepsy, migraine, fatigue, and digestive disturbances. Lesions of the chiasma are most commonly caused by tumours of the pituitary body, as seen in acromegaly, where bitemporal hemianopsia is a not uncommon symptom.

(*b*) **Total Loss of Sight in One Eye** (apart from local affections of the eye), the other being normal, is due to a lesion of the optic nerve—*e.g.*, tumour, optic atrophy. If, in addition to blindness of one eye, we find temporal hemianopsia of the other, a tumour of the pituitary body pressing on the chiasma and on one optic nerve is probable.

**Amaurosis.**—This term was formerly restricted to those cases of total blindness in which there is no obvious lesion of the eye—such conditions, for example, as migraine, hysteria, electric shock, poisoning by quinine, salicylate of soda, etc. The word 'amaurosis' is, however, used at present in a wider sense, to signify blindness from any cause.



(c) **Contracted Field of Vision.**—The field of vision may, on examination with the perimeter, be found concentrically contracted, the peripheral regions being insensitive to light. This may be a symptom of glaucoma, of optic atrophy, and of neuroses—viz., hysteria, neurasthenia, and traumatic neurosis or 'railway spine.'

(d) **Colour Field of Vision.**—As mentioned above, the area of the field of vision for colours is less extensive than that for white light, green having the smallest field and blue the largest after white. In central scotoma (see next paragraph) and in hysteria we find abnormalities in this respect, either contraction of the colour's field or change in its usual extent being observed.

(e) **Central Scotoma, or Toxic Central Amblyopia** (from Gr  $\alpha\mu\beta\lambda\acute{\upsilon}\varsigma$ , dulled;  $\acute{\omega}\psi$ , the eye), are the terms used to identify a loss of acuteness of vision, especially as to colours in its earlier stages, and often an extreme dimness of sight in the central portion of the retina. The affection may come on rapidly with a misty dimness, but no loss of sight. On examination it is found that the centre of the field may be normal for white, but green may be unrecognized, or may seem grey, and other colours may be mistaken; at the periphery of the field nothing abnormal may be discovered. As the affection progresses sight is still further diminished, but on removal of the cause normal vision may return. The cause of this affection in most cases is the abuse of alcohol and tobacco; it is also seen in diabetes, uræmia, poisoning by quinine, iodoform, etc.

4. **Oculo-motor Disturbances.**—Disturbances of vision may be due to disorders of the third, fourth, and sixth nerves. The chief interferences with sight are double vision (diplopia) and erroneous projection, both of which are considered with the paralyses of the oculo-motor nerves at p. 215 *et seq.*

5. **Ophthalmoscopic Appearance.**—An examination of the fundus oculi by means of the ophthalmoscope yields valuable information. While it is undoubtedly difficult for the beginner to interpret accurately what he sees with this instrument, or even at times to see anything worth interpreting, a little systematic practice and acquaintance with the laws of physiological optics will enable any medical practitioner to obtain much useful information from this source. As stated at the beginning of this article, local affections of the eyes are omitted from our notice.

The observer must naturally make himself familiar with the

appearance of the normal fundus, and especially of the optic papilla and disc.

(a) **Optic Neuritis.**—Instead of the pale pink clearly defined disc, we may note that the papilla is swollen and hyperæmic, the margins hazy or 'woolly,' the central vein increased in size, while the central artery may be of normal size or contracted. We may observe all grades of intensity of this disturbance, which is due to inflammation of the optic nerve, or **optic neuritis**. In extreme cases the papilla is swollen into a dome-shaped projection, the veins greatly distended, and evidence of secondary vascular and inflammatory changes in the surrounding retina may be seen. Thus greyish or white striæ or spots may surround the disc, while flame-shaped patches of hæmorrhagic origin are seen on and near the papilla. This is the condition which was formerly known as **choked disc**, but which is more accurately termed **papillitis**. In many cases optic neuritis, even in its severer grades, causes little or no interference with sight, the field of vision and acuteness of vision remaining normal. In other instances, however, the disturbance of vision is considerable, even more, perhaps, than one might expect from the ophthalmoscopic appearance.

The changes that are found in the papilla are those of inflammation and obstructed venous circulation—viz., venous hyperæmia, œdema, proliferation of connective tissue and infiltration of round cells. This condition arises most frequently as a result of **tumours and abscess of the brain** (syphilis, tubercle, cancer, glioma); also from **meningitis, hydrocephalus**, and occasionally in spinal disease—viz., **tubes dorsalis** and **myelitis**; also rarely in **peripheral neuritis**. Tumours and inflammations of the orbit may also give rise to the affection, in which case it is often unilateral, while in the occurrences of cerebral origin it is almost always bilateral. Lastly, optic neuritis may be a symptom of more general disturbance in which the nervous system takes part. Thus we may find it in **Bright's disease** (associated with retinitis—a neuro-retinitis), **chlorosis**, in **rheumatism**, in **lead-poisoning**, **syphilis**, apart from tumours, **suppression of menstruation**, and **exposure to cold**.

The occurrence of optic neuritis, as a consequence of brain tumours is very constant, and it is at times surprising how a small cerebral tumour may cause an intense papillitis. While intracranial pressure is responsible for the neuritis in most cases,

it must be admitted that there is a true neuritis present, and not merely a venous stasis, as the name 'choked disc' or 'stauungs-papille' would lead one to suppose. The hyperæmic origin of the inflammation is doubtless the obstruction to venous and lymph return from the nerve sheath, which is revealed by enlargement of the retinal veins and œdema of the papilla and optic nerve. Intracranial pressure which has been rapidly developed will produce greater disturbance of the circulation than that which has more gradually supervened; hence a comparatively small but rapidly-growing tumour may cause a more intense optic neuritis than a larger tumour of slower growth. The presence of a small tumour is insufficient to increase the intracranial pressure by its mere bulk, but it may directly cause irritation or obstruction to venous return, and so favour serous effusion into the third or lateral ventricles, which would materially increase the pressure inside the skull. In meningitis also effusion is mainly responsible for the optic neuritis which is so often seen, especially in the tuberculous form.

(b) **Optic Atrophy.**—Instead of a swollen, hyperæmic papilla we may find the disc pale and hollowed on the surface ('cupped'), the vessels shrunken, and often outlined by two white lines representing their thickened coats. This condition is due to atrophy of the optic nerve, which may be either a primary disease of the nerve or one secondary or consecutive to pre-existing disease in the nerve or brain.

(i.) Primary optic atrophy is frequently found associated with disease of the spinal cord. The affection probably commences at the papilla, which shows a bluish-white colour and distinct outline.

Of spinal diseases, locomotor ataxia is most frequently accompanied by optic atrophy. It has been observed that those cases of tabes exhibiting optic atrophy early in the spinal affection may never become ataxic.

In multiple sclerosis and in general paralysis of the insane optic atrophy is also, but less frequently, found.

A hereditary form of the affection is described, and it is believed to be due to exposure to cold, sexual excess, diabetes, lead-poisoning, and alcoholism.

(ii.) Secondary optic atrophy gives rise to changes in the disc very much like the primary form. The outline is not so sharp. the colour is a duller white, and the retinal vessels are smaller.

Optic neuritis frequently precedes atrophy, especially if the neuritis has lasted for a considerable time. Pressure may cause atrophy without producing neuritis, and may be due to tumour, hæmorrhage, fracture of cranial bones, inflammatory exudation. Enlargement of the pituitary body or distension of the third ventricle may press upon the chiasma. Embolism of the central artery of the retina and retinitis may also give rise to atrophy of the nerve.

In both forms of atrophy sight is invariably affected in proportion to the extent of the atrophy. Not only is vision less acute and the field of vision restricted, but there is usually colour blindness.

(c) **Retinitis.**—There may be a diffuse cloudiness of the fundus, especially of its central regions. The papilla may be congested, swollen, and indistinctly outlined; the retinal veins may be engorged; bright or dark red hæmorrhagic patches and white exudations and degenerations may be observed. These changes, which are usually bilateral, indicate **retinitis**, or, when the papilla shares in the inflammation, **neuro-retinitis**.

The diagnostic value of this affection is considerable, as it is seen in various forms associated with different diseases.

(i.) Albuminuric retinitis is found most frequently with the contracted kidney, but may occur with any form of nephritis (in 15 to 25 per cent. of the cases of chronic nephritis—Osler). It occurs late in the disease as a rule, though its result—impaired vision—may be the first inconvenience the patient has to complain of. The fundus shows the changes above-mentioned, the papilla being usually involved, while patches of hæmorrhage and fatty degeneration are distributed concentrically or radially round the disc or macula.

(ii.) Hæmorrhagic neuro-retinitis may occur in vascular and cardiac affections, such as arterio-sclerosis, aneurism, valvular disease, and cardiac hypertrophy. In these affections retinitis is often unilateral.

(iii.) Hæmorrhagic or inflammatory retinitis and neuro-retinitis are seen in various forms in diabetes, in pernicious and other forms of anæmia, in lead-poisoning, in infectious diseases.

(iv.) Purulent retinitis may occur from septic embolism in the course of pyæmia or septicæmia, commencing as multiple retinal hæmorrhages, and proceeding to general inflammation of the eye, or **panophthalmitis**.

(v.) Pigmented retinitis is seen as a number of pigmented spots distributed towards the periphery of the fundus, and gradually encroaching upon the central regions. The patient may, in consequence of this situation of the lesion, be able to read and distinguish objects fairly well near the point of fixation, while he is unable to find his way about with comfort and safety. This affection usually appears in children whose development is defective from hereditary causes—*e.g.*, congenital syphilis, consanguinity of parents.

(d) **Cataract.**—The fundus may be invisible owing to opacity of the lens; or the opacity may be in striæ, flocculi, or dots. Light thrown obliquely on the lens will show up the opacity better than the transmitted light from the ophthalmoscope. This obstruction to vision is commonly due to advancing age, but also occurs in diabetes. It is occasionally congenital, and also arises in the course of diseases and injuries of the eye.

(e) **Tumours** may occur in the eye in conjunction with similar disease elsewhere. The only one of diagnostic interest is **tubercle**, which may be seen as one or more round, yellowish spots in the choroid, usually near the disc, occurring in the later stages of miliary tuberculosis. They are not likely to occur in tubercular meningitis, unless the affection has been generalized.

**Summary.**—The disturbances of vision which are of diagnostic value are:

1. Minor disturbances: yellow or red discoloration *isœre* volitantes, flashes of light, glittering scotomata.
2. Acuteness of vision, near and distant, may be defective.
3. Defects of the field of vision: **diminished area of the field, scotomata.** The defective area of the field of vision may occur in several different forms. (One half of the field may be invisible, a condition termed **hemianopsia**. This may be **homonymous** (the right or the left halves of both fields blind), **temporal** (the temporal half of one field absent), **nasal** (the nasal half of one field absent), **bitemporal**, **binasal**, **superior**, or **inferior** (the upper or lower half of the field deficient).

The field of vision may be concentrically contracted, the peripheral regions being invisible; or the central portion of the field alone may be defective (**central scotoma**). Complete invisibility of the field of vision means total blindness of the affected eye. Changes in the field of vision for colours are sometimes observed.

4. Oculo-motor disturbances may cause disorders of vision—  
e.g., **double vision, erroneous projection.**

5. Defects of vision discovered by the ophthalmoscope: **optic neuritis, optic atrophy, retinitis, cataract, tubercle of the choroid.**

### VISION NULLE.

In certain affections of the visual apparatus, especially lesions of the cortical centre of vision, the patient has abnormal 'blind spots' in his field of vision, of which he is unaware until they are elicited by an examination. This is known as **vision nulle**, which is to be distinguished from **vision obscure** (*q.v.*, below), a condition in which the patient is aware of the defect in his field of vision. (See Vision, Disturbances of, p. 547.)

### VISION OBSCURE.

A darkened or indistinct vision affecting certain regions of the field of vision (scotomata). The defect is due to a lesion of the visual apparatus below the cortical centres of vision. The patient is aware of his defective sight, in which respect the affection differs from that known as vision nulle (*q.v.*, above), in which the subject is unaware of the presence of the scotoma until it is elicited by examination. (See Vision, Disturbances of, p. 547.)

### VOCAL RESONANCE.

The sounds produced in the larynx by speaking, coughing, etc., may be heard at the surface of the chest as a droning, buzzing sound. The intensity of the sound varies not only with the degrees of loudness of the laryngeal sound, but, what is more important for the diagnosis, it varies much with differences in the conducting capacity of the lung and other structures intervening between the larynx and the stethoscope. Increase of vocal resonance (**bronchophony**) usually implies a higher degree of conductivity of these tissues than normal, while weakened vocal resonance, as a rule, signifies the contrary condition (see p. 415).

### VOCAL FREMITUS.

Palpable vibrations originating in the vocal cords during phonation, and transmitted thence via the bronchi and tissues to the chest-walls, where they may be felt by the observer. In various diseased conditions the fremitus may be exaggerated, diminished, or lost. The subject is considered at p. 477.

**VOICE, Abnormalities of.**

The quality of the speaking voice may be altered by disease situated in the larynx, or involving its innervation. Affections of the respiratory passages and mouth, and general disturbances of health also produce an influence on the quality or intensity of the voice sounds. We omit in this place the consideration of speech defects, which are discussed at p. 373.

**Hoarseness and Loss of Voice (Aphonia).**—Defective or irregular vibration of the vocal cords from any cause produces hoarseness of all degrees, from a slight loss of resonance to complete loss of voice. The commonest cause of defective vibration is catarrhal inflammation involving the cords. The familiar 'cold,' commencing as an infection of the fauces or nasal cavity, may spread by continuity, producing laryngitis, tracheitis, or bronchitis, disturbing the vocal function in its course. Exposure of the throat or air passages to cold, and prolonged or violent use of the cords in speaking, singing, or coughing, may set up a local catarrh. Among the specific affections of the larynx causing hoarseness are diphtheria, syphilis, and tuberculosis. In the last-named conditions evidence of the disease elsewhere usually makes the diagnosis clear. The presence of tubercular disease in the lungs or other organs suggests, but nothing more, the diagnosis of tubercular laryngitis. The hoarseness in such cases may be due to coughing, and examination of the larynx must, of course, be carefully effected. Other local causes of hoarseness which may be borne in mind are: tumours of the larynx, cicatricial contractions, foreign bodies, œdema of the glottis, inflammatory or malignant disease of the œsophagus or pharynx.

Disease of the laryngeal nerves (superior and inferior branches of the vagus), or of their central connections, interferes with the voice. In addition to bulbar paralysis and diphtheritic or other forms of peripheral neuritis, these nerves may be injured by the pressure of tumours, enlarged lymphatic glands, enlarged thyroid, aneurism of the carotid, subclavian, and innominate arteries or aorta. The course of the inferior laryngeal nerve, which innervates almost all the laryngeal muscles, renders the vocal cords very liable to paralysis from aneurism of the aorta or right subclavian artery, or from mediastinal tumours. Pleurisy or phthisis of the apex of the lung may also be a source of irritation or pressure to the recurrent nerve, especially on the right side.

Loss of sensation in the larynx also follows injury to these

nerves, so that foreign bodies have a tendency to enter the larynx.

Paralysis of all the muscles of the larynx, occurring in the course of bulbar paralysis or of peripheral neuritis (rarely from lesion of both inferior laryngeals), causes aphonia, with inability to cough, and without dyspnoea, except on deep inspiration.

The voice is deeply pitched and hoarse; particles of food easily enter the larynx, and are coughed out without difficulty: paralysis of the crico-thyroid muscle from lesion of the superior laryngeal nerve or its nucleus.

The voice may be normal, but there is inspiratory dyspnoea: bilateral paralysis of the adductors. This dangerous condition may occur in bulbar paralysis, locomotor ataxia, and in hysteria. It may result from laryngitis, or from pressure on the vagi.

The voice is lost, coughing is possible, there is neither dyspnoea nor stridor: paralysis of the adductors, the result of over-use of the voice, of laryngeal catarrh, or of hysteria (**hysterical aphonia**).

The voice is hoarse and rough, and is easily tired; there may be cough, but dyspnoea is absent: unilateral abductor paralysis. This is the condition most commonly resulting from aneurism involving one recurrent nerve; the adductors may also be affected, in which case the voice becomes still weaker.

**Nasal Voice.**—Normal speech requires the assistance of the nasal and naso-pharyngeal cavities for the correct pronunciation of certain words (*e.g.*, 'sink'), while others can only be properly spoken if the naso-pharyngeal cavity is completely shut off by means of the palate from that of the mouth and pharynx (*e.g.*, 'sick'). When this is not effectually performed, the result is what is usually known as the **nasal** or **open nasal voice**—*i.e.*, talking through the nose. Under these circumstances the word 'sick' would be pronounced 'sink,' or if, as is often the case in the affections causing this abnormality, gutturals are impossible, 'sick' would sound something like 'sing.' Diphtheritic paralysis, perforation of the palate (commonly syphilitic), cleft palate, or a mere habit of speech, are the causes of this defect. The contrary condition, an inability to place the nasal cavity in acoustic communication with the buccal, gives rise to what is commonly, but incorrectly, termed the **closed nasal voice**. On attempting to say, for example, the words 'sink and swim, the result is 'sick add



'swib,' which, instead of being an instance of the nasal voice, is one from which the proper nasal quality is absent. The distinction is, no doubt, of minor importance, but it shows a confusion of ideas which may as well be corrected, because the conditions producing the **non-nasal** quality of voice are the exact opposite of those causing the open nasal voice. So long as both these vocal abnormalities are included in the term 'nasal voice,' there will be misapprehension in the minds of some as to the respective conditions underlying them. The non-nasal voice is the result of nasal and naso-pharyngeal polypi, rhinitis, adenoids, or any other obstruction in that region.

The presence of swelling in the fauces or pharynx (compromised by enlarged tonsils or acute pharyngitis, rarely retro-pharyngeal abscess), imparts an intonation to the voice which can be termed nasal, though it may to some extent affect the nasal resonance. The peculiar 'throaty' quality can at once be recognized.

Delirium from any cause is often indicated by weakness of the voice. In pneumonia, phthisis, pleurisy, or other pulmonary complaint, the voice is materially affected, being weak, toneless, and broken; the same may be said of heart affections. Hysteria has been already mentioned as a cause of aphonia.

## VOMITING.

The vomiting reflex arc—Central vomiting—Reflex vomiting—Vomiting of childhood—Relation of vomiting to the act of eating—Vomiting independent of food—The time at which vomiting occurs—The relation of pain to vomiting—The relation of nausea to vomiting—Projectile vomiting—Quantity of vomit—Bilious vomiting—Fæcal vomiting—Free hydrochloric acid—Organic acids—Bloody vomit.

The act of vomiting is a reflex, the centre for which is situated in the medulla, in intimate relation with the respiratory centre. The afferent limb of the reflex arc consists of: The trigeminal and glosso-pharyngeal nerves, which are responsible for vomiting when the fauces are tickled; the vagus, through which the stimuli producing gastric irritant vomiting reach the centre; sensory nerves from various organs—*e.g.*, kidneys, testicles, ovaries, etc.; the nerves from other organs of special sense—*viz.*, olfactory, optic, auditory.

The centre may be directly stimulated, as in the case of cerebral

and cerebellar lesions, by emotions, by a toxic state of the blood, as in uræmia, etc.

The efferent limb of the reflex arc is formed by the vagi, the splanchnics, the phrenics, and the spinal nerves to the abdominal muscles.

Two classes of vomiting may be distinguished:

1. **Central Vomiting**, the result of direct stimulation of the vomiting centre.

2. **Reflex Vomiting**, the result of nervous impulses reaching the vomiting centre from the periphery, and set up by injuries and disturbances of many organs, including those originating in the stomach. It is incorrect to restrict the use of the term 'reflex vomiting' to those cases where the act is caused by disturbances at a distance from the stomach—*e.g.*, in the testicle or gall-bladder—as the vomiting of gastric irritation is equally a reflex act.

The relation of the act of vomiting to disease may be best understood by a consideration of the following points:

1. **Infants and Young Children** reject the stomach contents promptly when local irritation in the stomach renders their presence there objectionable. In these little patients, it must be remembered, vomiting is very frequently an early sign of acute diseases—*e.g.*, the exanthemata.

2. **The Period at which Vomiting occurs after Eating** should be inquired into. If it follow immediately on swallowing, it is suggestive of regurgitation rather than vomiting—that is, a return of the food from the œsophagus before it has reached the stomach. A stricture high up in the gullet causes instant return of the swallowed matter, while a stricture at the lower end near the stomach may permit the food to rest in the dilated gullet a quarter of an hour or so before regurgitation. On the other hand, true vomiting may occur immediately after eating, when the stomach is in a condition of irritability—*e.g.*, in acute gastritis, in gastric ulcer, or in cancer of the stomach. In these affections, however, vomiting is more frequently delayed. In cases of chronic gastritis the interval between eating and vomiting is usually considerable (one to two hours), and here the vomited food is found in a still undigested condition.

3. **Vomiting without Regard to the Presence or Absence of Food in the Stomach** occurs in conditions in which the vomiting centre is stimulated either (i.) directly, as in cerebral and cerebellar tumours, in meningitis, in emotional disturbances, and in

toxæmias; or (ii.) **indirectly** by stimuli reaching the vomiting centre from the periphery, exclusive of the stomach—*e.g.*, lesions of the uterus, breasts, testicles, kidneys, ureters, bladder, gall-bladder, gall-duct, bowels, lungs, pharynx (the cough in pertussis and in phthisis produces vomiting by irritation of the pharynx), and lastly from the organs of special sense—*e.g.*, the vomiting of glaucoma, vomiting produced by unpleasant sights and smells and by the ear disturbance of Ménière's disease.

4. **The Time at which Vomiting Occurs.**—In pregnancy vomiting and nausea commonly occur as soon as the patient rises from bed in the morning, the change of posture being sufficient to set up nervous impulses from the uterus or its appendages to the vomiting centre. In chronic alcoholism a bout of retching, with nausea and vomiting is a usual occurrence when the patient first rises in the morning, owing to the presence of catarrhal secretion in the stomach, which has accumulated during the night. In dilatation of the stomach, especially when due to pyloric contraction, but also in atonic dyspepsia, vomiting may only appear at longer intervals, perhaps every two or three days, and then copiously.

5. **The Relation of Pain to Vomiting.**—The pain of gastric ulcer and of acute dyspepsia is usually markedly relieved by vomiting. In chronic gastritis and in gastric cancer the pain is as a rule somewhat relieved by vomiting, but not to the same extent as in gastric ulcer. The pain from the passage of gall-stones and of kidney-stones, and that from peritonitis of all descriptions, is not relieved by vomiting. It is noteworthy that the pain of appendicitis in its early stages is often located in the epigastrium or about the umbilicus, and is not relieved by vomiting.

6. **The Relation of Nausea to Vomiting.**—In almost all cases peripherally started vomiting, and in some instances central vomiting (*e.g.*, emotions), is accompanied or preceded by nausea. Its absence is important, as it often indicates a lesion in the cranial cavity. The influence of a cerebral tumour or effusion is in many cases exercised upon the vomiting centre, without the production of nausea.

7. **Projectile Vomiting** is the sudden expulsion of the stomach contents without preliminary retching, and is often observed in intracranial lesions. A free, copious, and continuous vomiting almost a regurgitation, is sometimes found in peritonitis.

8. **The Quantity of Vomited Material** may be trifling, being in cases of gastric catarrh and gastric ulcer often little more than saliva and mucus. In dilatation of the stomach, as already mentioned, the vomit may be very copious, several pints being usually evacuated at a time, and a recurrence may take place in a few days.

9. An examination of the **Contents of the Stomach** is often required. The methods to be adopted are indicated in the article on the subject, at p. 391. Here the clinical significance of the result of the examination is considered.

(i.) **Food, mucus, and saliva** are found in all conditions causing vomiting, and are of no diagnostic value.

(ii.) **Bile** is regurgitated from the duodenum into the stomach in persistent vomiting from any cause. It occurs in acute and chronic gastric catarrh, in vomiting from other reflex origins besides the stomach, and particularly in intestinal affections. In obstruction of the bowels, acute and chronic, and in peritonitis, bilious vomiting is constantly observed.

(iii.) **Fæcal vomiting** is a further stage of the reverse peristalsis causing bilious vomiting. It occurs in obstruction of the bowels and in peritonitis, and is said to have been seen in hysteria.

(iv.) **Free hydrochloric acid in excess (hyperchlorhydria)** is an evidence in favour of gastric ulcer. It sometimes occurs in nervous dyspepsia, in acute and in chronic gastric catarrh. **Absence or diminution of free hydrochloric acid (hypochlorhydria)** is the rule in cancer of the stomach, in dilatation of the stomach from atony or from chronic catarrh, and sometimes from cicatricial contraction of the pylorus. At times this condition is found in nervous dyspepsia, in anæmia, and in fevers. **Free lactic acid** is often found where free hydrochloric acid is absent, and is a sign of abnormal fermentation. **Supersecretion of the gastric fluids** is found in catarrh of the stomach, in the gastric crises of locomotor ataxia, in gastric neuroses, and sometimes in gastric ulcer.

(v.) **Blood** may be vomited in large or small quantities (**hæmatemesis**). It may be shed in the respiratory passages or in the œsophagus and swallowed, to be afterwards ejected from the stomach. It may be the result of (a) ulcer of the stomach or ulcer of the duodenum. In these cases the blood is usually vomited at considerable intervals, and may be copious. (b) Cancer

COMPARATIVE TABLE OF THREE COMMON STOMACH DISORDERS

	Chronic Gastritis.	Gastric Ulcer.	Gastric Cancer.
<b>Family history</b>	Unimportant	Unimportant	Of slight importance if positive
<b>Age</b> .. ..	Any age	Young adults	Over middle age
<b>Sex</b> .. ..	Either	Females commonest	Males rather more frequently
<b>Previous diseases, habits, and occupation</b>	A previous history of alcoholism, unsuitable or excessive food, or sedentary habits is in favour of this diagnosis	Chlorosis, previous dyspepsia, carious teeth, constipation, indoor occupations, support diagnosis of gastric ulcer.	Of little or no importance
<b>Pain</b> .. ..	Neither severe nor localized	Often severe, usually localized	Severe; variable site; aggravated by food
<b>Tenderness</b> ..	Slight or absent	Present	Present
<b>Nausea</b> ..	Slight	Variable	Generally intense
<b>Vomiting</b> ..	Usually moderate	Variable; possibly that of dilatation	Commonly present; probably that of dilatation
<b>Hæmatemesis</b>	Rare	Copious, at long intervals	Scanty, at short intervals
<b>Free hydrochloric acid</b>	Diminished	Increased	Absent
<b>Organic acids</b>	May be present	Absent	May be present
<b>Tumour</b> ..	None	Rare	Present in 75 per cent. of cases
<b>Duration of disease</b>	Indefinite	Indefinite	From one to two years, sometimes longer
<b>Loss of weight</b>	Moderate	Moderate	Considerable

of the stomach. Here the blood is usually less copious, and only appears at a late period of the disease, when cachexia is well advanced. It may be vomited frequently and at short intervals. (c) Gastric catarrh may infrequently cause hæmatemesis, but streaks of blood may be found in the vomit after any prolonged attack of vomiting. (d) Obstruction to the venous return in the portal vein causes congestion and varicosities of the capillaries and venules in the stomach (and elsewhere in the portal circuit). This often gives rise to fairly free hæmorrhage into the stomach, causing hæmatemesis. The condition is furnished by cirrhosis of the liver, the 'nutmeg liver' (cardiac), and cancer of the liver. (e) Diseases of the spleen. (f) Corrosive poisons and other injuries—*e.g.*, passing the stomach-tube. (g) Aneurism of the aorta, opening into the œsophagus or stomach. (h) Blood states giving rise to hæmorrhages—*e.g.*, purpura, scurvy, septic inflammations, acute yellow atrophy of the liver. (See Hæmatemesis, p. 153.)

As a rule, there is no difficulty in detecting blood when present in the vomit. It may at times, however, be simulated by drugs (chiefly bismuth and iron). It is best recognized by means of the hæmin test, described at p. 533.

### VON PIRQUET'S REACTION.

An inoculation into any part of the skin of a 25 per cent. solution of Koch's old tuberculin, a control experiment being carried out on another surface with salt solution. In cases of active tuberculosis, and in a large proportion of latent or cured tuberculosis, the skin reaction occurs—*i.e.*, a red papule about 1 centimetre in diameter appears within a few hours, or it may be only after twenty-four hours. Pustules or vesicles may accompany the papule. The papule is surrounded by an inflamed area, and remains for five or six days or longer.

Many modifications of this method have been advocated—*e.g.*, friction of the skin with any old tuberculin (Lignière); inunction of an ointment composed of old tuberculin in lanoline (Moro, see p. 202); pure new tuberculin rubbed on to the surface of a partly healed blister (Woodcock).

All these skin reactions have the fault of being too sensitive, showing evidence of tuberculosis even where there is no active disease.

### WADDLING GAIT.

A mode of progression in which the legs are placed widely apart, the shoulders thrown back, and the body as a whole is inclined backward. The patient advances in a rolling, waddling fashion, with the lumbar spine arched forward. This type of gait is observed in cases of large abdominal tumours, ascites, pregnancy, obesity, and in pseudo-hypertrophic paralysis.

**WASSERMANN'S REACTION.** See p. 85.

### WATERBRASH (Pyrosis).

A term used to indicate the regurgitation of a quantity of fluid from the stomach, accompanied by a burning pain in the epigastrium. The fluid may be largely saliva, in which case it is alkaline in reaction. It may be mainly gastric secretion, and acid in reaction. It is a symptom of indigestion, and is commonest in adult females.

### WEBER'S SYNDROME.

An alternate paralysis consisting of oculo-motor paralysis on one side, with loss of power in the opposite arm and leg, due to a lesion in the crus on the side of the third nerve paralysis.

### WERNICKE'S PUPILLARY REACTION.

By carefully focussing a cone of light on to the retina, Wernicke has shown that in cases of hemianopsia the light stimulus falling on the blind side of the retina gives rise to no contraction of the pupil, but stimulation of the sentient part of the retina causes the usual pupil reflex (see p. 323).

### WESTPHAL'S PUPIL REACTION.

A contraction of the pupil occurring on the production of 'Bell's phenomenon'—that is, the rolling up of the eyeball under the upper lid when the latter is unable to close, either because of facial paralysis or of its being held open by the observer in order to produce the symptom. The pupil is seen to contract under these circumstances in some cases of locomotor ataxia and of general paralysis of the insane.

**WESTPHAL'S SIGN.**

This term is given to loss of the knee-jerks, the cause being any affection which interrupts the reflex arc. The various conditions giving rise to diminished or lost reflexes are discussed in the articles on the reflexes, at p. 334.

**WIDAL'S REACTION.** See **Blood Examination**, p. 84.

**WILLIAMS' TRACHEAL RESONANCE.**

Consolidation of the apex of a lung commonly gives a dull note on percussion. In some cases, however, owing to the proximity of the trachea to the spot percussed, and owing chiefly to the high conducting qualities of the consolidated lung, the percussion-sound has an added tympanitic resonance, the tracheal resonance. It is found that this sound, like that described by Wintrich (p. 461), is lowered in pitch if the mouth is closed, and raised if the mouth is opened.

**WINTRICH'S SIGN.**

On percussing over a pulmonary cavity which communicates with a bronchus, the resulting tympanitic or amphoric note is raised in pitch if the patient opens his mouth, and the pitch is lowered when the mouth is closed (see p. 461).

**WORD-BLINDNESS.**

Inability to understand the meaning of written or printed words on the part of a subject who was previously familiar with the use of the written language. It is observed in some forms of aphasia. (See **Disorders of Speech**, p. 379.)

**WORD-DEAFNESS.**

One of the evidences of aphasia, the patient being unable to comprehend any spoken or (commonly) written words. Here the memory or conception of the sound now uttered cannot be recalled, as the centre where such ideas are stored, or their pathway to the motor speech centre, is destroyed. (See **Disorders of Speech**, p. 380.)



**WORMS, Intestinal.**

The presence of parasites in the intestines is generally detected by their discovery or that of their ova in the fæces. In this country four varieties are most commonly met with—viz., the common round-worm (*Ascaris lumbricoides*), the thread-worm (*Oxyuris vermicularis*) the beef tape-worm (*Tania saginata*), and the pork tape-worm (*Tania solium*). Other less frequently occurring varieties are: *Ankylostomum duodenale*, *Trichocephalus dispar*, and *Bothriocephalus latus*. The conditions under which these parasites occur, and their distinguishing characteristics, are discussed in the article on the Fæces, p. 143.

**WRIST-CLONUS.**

The flexor muscles of the hand are to be stretched by firm pressure on the palm of the hand. So long as pressure is kept up, clonic flexing movements of the hand are seen. This rarely occurs in healthy subjects, but is an evidence of exalted reflex action. (See Reflexes, p. 339.)

**WRIST-JERK.**

The hand is flexed and the tendons of the stretched extensor muscles are tapped. In many cases of health, and in conditions where the reflexes are generally increased, the stroke evokes an extending contraction. A similar flexing movement of the hand may be caused by a stroke on the flexor tendons at the wrist, the hand being first fully extended. (See Reflexes, p. 339.)

**X RAYS IN DIAGNOSIS.**

Apparatus required—Mode of action—Fluoroscopy—Radiogram—Examination of the radiogram—Results obtained by X-ray examination of the various regions, organs, and tissues.

A. Thorax: (1) trachea; (2) lungs and pleuræ; (3) heart.

B. Muscles.

C. Abdomen:

(1) stomach; (2) intestines; (3) liver; (4) gall-bladder;  
(5) kidney; (6) ureters; (7) urinary bladder; (8)  
tumours.

D. Arteries.

E. Lymphatic Glands.

F. Limbs: (1) bones; (2) joints.

G. Vertebral Column.

The assistance of the X rays was early taken advantage of in the diagnosis of surgical conditions. Their use in the field of medicine has been more gradually extended as improved technique and experience have shown that many pathological conditions may be detected.

The proper appreciation of an X-ray examination requires an amount of knowledge only to be expected from a medical man, and considerable experience is requisite in the interpretation of the findings in many cases.

For an examination certain electrical apparatus is required. The electricity may be obtained from a friction machine—a good static machine will yield electricity of sufficient tension for the production of X rays. In this country the induction coil is more commonly used to get high-tension electricity. A current of low tension, which may be obtained from a storage battery or from direct current mains, with a resistance, passes through the primary coil of an induction machine, an interrupter being placed in the circuit. At each 'make' of the primary current, electricity of lesser quantity but of higher tension is induced in the secondary coil, the terminals of which lead to a focus-tube.

The X rays are produced by an electrical discharge at high tension through a tube which has been exhausted of air to a high degree of rarefaction. A perfect vacuum is, of course, not obtainable.

The discharge at this degree of vacuum is invisible. From the negative terminal inside the tube, the cathode rays are repelled in a straight line, and are reflected as X rays at a right angle, from a heavy target placed in their path and set at an angle of 45 degrees. It is only within certain limits of vacuum that the X rays are given off, and their power of penetration depends on variations within these limits.

A low-vacuum tube has little penetrating power, and such an object as the hand will be opaque. As the vacuum rises, so does the penetrating power, the rays passing readily through the skin and soft tissues, the bones and dense structures standing out in marked contrast. With a still higher vacuum even the bones are, to a certain extent, penetrated: details of their structure are shown, and the contrast with the softer tissues is not so well defined. At a very high vacuum the contrast is so ill-defined that the picture is not definite. Finally, with increasing vacuum, the resistance in the tube—for the electricity passes by means of the

molecules of air still present—rises so high that the discharge takes place preferably through the air around the glass of the tube.

The X rays are invisible. In order to demonstrate them two methods are therefore employed, depending, firstly, on their power of causing certain substances to fluoresce when acted on, and, secondly, on their chemical effect on a photographic plate.

A screen coated with platinocyanide of barium, when irradiated by X rays, shows an intense green fluorescence. An object which offers obstruction to the rays, when placed between their source and the screen, causes a shadow.

Similarly, when such an object is interposed between a photographic plate protected from light and the focus-tube, and the plate subsequently developed, a radiographic picture is given.

Fluoroscopy, or examination by the screen, is quicker and easier. The object may be observed from several different points of view, and movements may be observed and followed, such as those of the diaphragm, heart, etc. The disadvantages are that finer shades may not be detected, and the examination cannot be prolonged, owing to the danger to the patient and to the observer from the action of the rays. The apparatus also is apt to be unduly strained in attempting to get clearer definition.

By taking a radiogram we get a permanent record, which may be studied by several persons at leisure; finer shadows may be observed which escape the eye when the screen is used; the patient is exposed to the rays for a certain limited time, and the apparatus is not strained to any extent.

In examining a radiogram several points should be borne in mind:

1. The negative will generally be found to be the most serviceable. It gives more detail than the best print taken from it.
2. The negative gives a representation of a shadow viewed from in front, the transparent parts being dark, the bones and dense structures being, by contrast, white. A print from the negative is similar to the shadow as seen on the screen.
3. The size of the shadow is proportional to the distance of the part from the plate and of the focus-tube from the plate, owing to the divergence of the rays from their source.
4. The sharpness of the shadow is proportional to its distance from the plate. So, in the chest, a radiogram taken with the

plate at the back shows the posterior portions of the ribs clear and sharp, while the sternum is large and not clearly defined. The reason of this is that the source of the rays is not a point, but a small plane. The further the obstructing bony edge is from the plate the more room will there be for the rays, striking this edge from different points of the plane in different lines, to diverge before reaching the plate.

5. The amount of detail shown depends, *inter alia*, on the degree of hardness or penetrating power of the focus-tube, so with a soft tube the bones will be quite opaque, while with a hard tube the cancellous architecture will be shown.

6. A very limited amount of information is given by a single radiogram as to the depth of a structure. The only thing one can say is, that the sharper the image the more likely is it to be near the plate. To get this information two radiograms are taken in the same position, the focus-tube being moved between the exposures laterally about 3 centimetres. The two pictures are then viewed in the stereoscope.

We have now to consider the results obtained by means of X-ray examinations of the different organs and tissues of the body, and to discuss the diagnostic value of the information so obtained.

**A. The Thorax.**—1. The trachea shows as a transparent band in front of the vertebræ. A stricture may be here detected.

2. **The Lungs and Pleura.**—Fluoroscopy. The apices should be clear; any opacity is generally due to pneumonia or tubercle. A diffuse shadow suggests congestion, often unilateral, more so in pneumonia; here the whole lobe is usually uniformly dark. Tubercle has a more mottled appearance. If the patient takes a deep breath and the darkness perceptibly lessens, the lesion is probably congestive and recent. Where fibrosis has taken place the lung will be wanting in elasticity, and will not expand with the enlargement of the chest. The slope of the ribs should be observed. Where fibrous contraction has taken place the ribs show the 'roof-tiling' arrangement. In emphysema the lungs are unduly transparent, and the ribs are more widely separated and horizontal. In the commonly associated chronic bronchitis the bronchioles are thickened, and their branches are seen especially towards the root.

A tumour of the lung shows a shadow with a rounded outline,

while an aneurism shows as a rounded shadow, coming from the roots of the great vessels with an expansile pulsation. When large, an aneurism may press on the bronchi or vessels, giving rise to congestion and opacity of the area of lung involved. Tumour is often associated with some pleural effusion, which is basal, and leads to obscurity of the arch of the diaphragm.

The diaphragm forms an arch, with the convexity upwards. Its movements should be observed; it descends 2 to 3 inches on



FIG. 73.—PHTHISIS.

The radiogram is that of a woman of twenty-three, and shows early tubercular infiltration of both apices. There were night sweats and marked wasting, but the physical signs at the apices were ill-defined at the period when the radiogram was taken. Later on, with the breaking down of the lung tissue the physical signs of phthisis became obvious.

deep inspiration. A unilateral impaired movement, when associated with dulness at the apex, is characteristic of early phthisis. Pleural effusion causes opacity to the outer side and below; the lung is retracted towards its root, and is here seen as a clear area. The upper limit of the effusion is not clearly defined.

In pyopneumothorax the upper border of the effusion is horizontal, and varies with the position of the patient. The heart-

beat or percussion of the chest will cause a wave, which may be detected along the upper border. Above the effusion the chest will be unusually transparent.

Old thickened pleura, from contraction of the fibrous tissue, causes the ribs to lie close together and the heart to be drawn to the side of the lesion.

Empyema and clear effusion cannot be certainly differentiated. The former is said to cause a denser shadow. Foreign bodies,

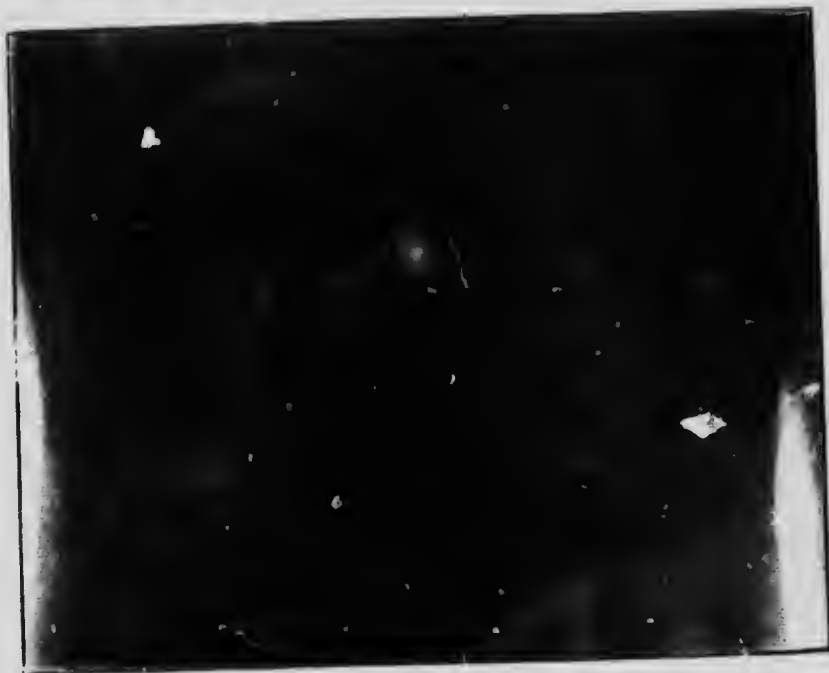


FIG. 74.—TUMOUR OF THE LUNG.

A tumour of the lung first diagnosed by means of this radiogram in July, 1905. The patient died in January, 1907, the growth having distended the side of the thorax to an enormous extent.

such as pins, coins, etc., are at once detected, and may reveal the cause of a cough and bronchitis when wholly unsuspected.

Those features not dependent on movement are better studied on the negative. Here in tubercular disease calcification may be recognized distinct from infiltration.

3. **The Heart.**—It was first convincingly demonstrated by the X rays that the heart is suspended from the great vessels, and does not, as was supposed, rest on the diaphragm. The observation gives a ready explanation of the cardiac embarrassment caused

by an overloaded stomach or meteorism pressing up the diaphragm. The heart is on rare occasions situated on the right side; this may be at once recognized. Transposition of viscera at times gives rise to great difficulty and mistakes in diagnosis.

By instantaneous exposures the heart may be radiographed in systole or diastole. If the focus-tube be placed a long way from the patient, the amount of divergence of the rays between the heart and the plate will be negligible, and a measure of the size of the heart may be obtained.

B. The **Muscles** are not readily defined from subcutaneous and other soft tissues. Calcification of muscles occasionally occurs, a traumatic myositis ossificans being recognized. Idiopathic myositis ossificans shows a remarkable X-ray picture (see Fig. 76).

C. **The Abdomen.**—It is in the region of the abdomen that most advance has been made in recent years. The whole of the digestive tract may now be studied by means of the bismuth meal. A thick emulsion of the carbonate or oxychloride of bismuth, when swallowed, casts a dense shadow, and its passage along the whole alimentary canal may be observed.

A flood of light has been thrown on the subject of intestinal disorders by this means, the work of Hertz, of London, having been of great service.

The act of deglutition may be followed and a stricture of the **oesophagus** is at once located.

The **stomach** is shown, as the bismuth clings to the walls, though a great part of the meal gravitates to the bottom of the stomach. An hour-glass stomach will be shown. The peristaltic waves of the pyloric region and spasm or stricture at the pylorus may be seen. The time necessary for the complete emptying of the stomach is also easy of determination. The bismuth enters the **duodenum**, and may be observed here within half an hour after the ingestion of the meal. Obstruction, due to kink of the duodeno-jejunal junction has been detected. In the **small intestine**, owing to the large area over which the bismuth becomes spread, a diffuse shadow is given, unless there be a stricture, most often due to the cicatrization of tubercular ulcers. In such a case the bismuth will be retained, showing a more dense, sausage-shaped tumour above the obstruction. In the lower end of the ileum there is sometimes delay in the passage of the intestinal contents, and here we may see a long cylindrical mass.

The **cæcum** is normally reached in about four and a half hours after the ingestion of food. As the bismuth meal collects in the cæcum it causes a dense shadow, in contrast with that of the small intestine. We can thus note the time in which the cæcum begins to be filled, and delay of the meal in the small intestine can readily be demonstrated. According to the findings with the bismuth meal, a loaded cæcum would appear not to be so frequent as has been supposed.

The passage along the **large intestine** is very slow, and in this region we get a clear picture. As malignant stricture is most frequently met with in the large intestine, this method of examination is of the greatest assistance in accurately locating the position of the lesion. Obstruction at the flexures due to kinking may also be diagnosed.

It has been shown that many cases of chronic constipation are due to retention of fæces in the **rectum**, a result of habitual disregard to the calls of Nature, the bowel becoming in consequence less sensitive. It is of importance that this condition should be appreciated, as the efficient treatment of constipation depends on a proper recognition of its cause.

The **liver**, being a dense structure, may be seen in outline, in contrast with the lungs and intestine. Owing to their want of density, **gall-stones** are not readily defined, but in some cases their shadow is quite discernible.

In the **kidney** calculi throw a shadow. Care should be taken to have the patient well purged beforehand, as concrete masses in the intestine may be mistaken for kidney-stones. By passing a metallic ureteral bougie and then taking a radiogram, the position of a stone in the **ureter** may be verified. As a good radiogram now shows the outline of the kidney itself, much more help will probably be forthcoming in the future in kidney affections.

Calcified glands in the mesentery are occasionally portrayed as irregular opacities, usually of small size.

The **bladder** may be distended with air and radiographed, when it shows as a clear rounded area; or it may be injected with bismuth or other opaque salt, and then examined. The action of the sphincter vesicæ has been so studied.

Stereoscopic examinations give more accurate information on these points.

**Tumours** of the abdomen cannot generally be detected when not readily found by other means.





FIG. 75.—RADIOGRAM OF THE LEG AND ANKLE-JOINT, TAKEN THROUGH SPLINTS. The calcified anterior tibial artery is clearly shown. Patient was a man aged forty-five.

D. The **Arteries** may at times be seen on the plate to be thickened and calcified (see Fig. 75). This is a subject of which much more will probably be heard in the future, as we have in this method a new means of studying the problem of arteriosclerosis.

E. A radiogram of the neck in a case of enlarged **Lymphatic Glands** of the Hodgkins type has shown calcification pointing to a tubercular element.

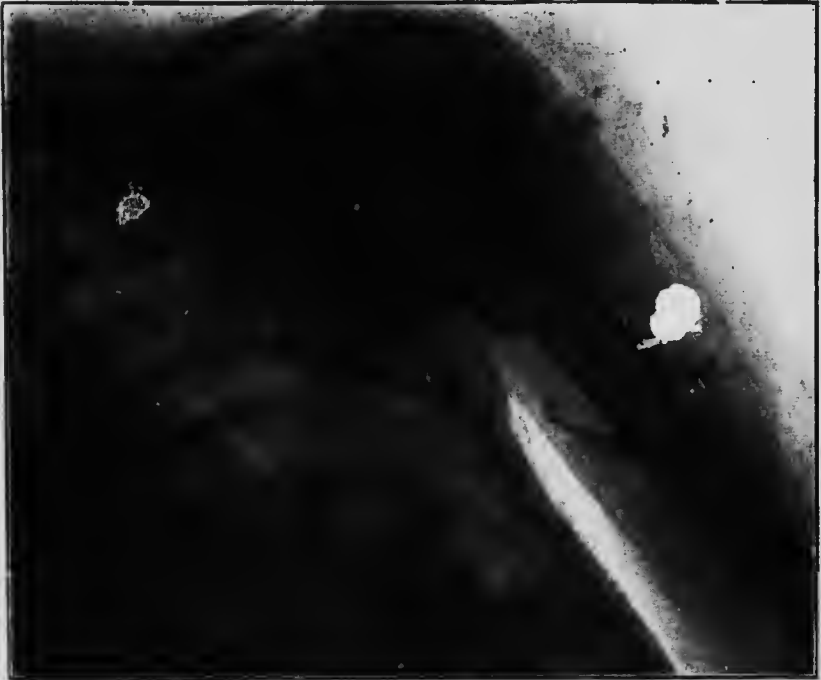


FIG. 76.—RADIOGRAM OF THE SHOULDER OF A PATIENT SUFFERING FROM MYOSITIS OSSIFICANS.

The ossified coraco-brachialis muscle had firmly fixed the arm to the side.

F. In the **Limbs** tumour growths have frequently a denser structure than the normal tissues. Erosion of bone, raising of periosteum, osteophytic growth, central abscess, and necrosis may be studied. At times old fractures and dislocations, especially about the hip-joint, in the absence of history, have not been diagnosed. Such cases have been mistaken for sciatica, the position often assumed in this disease to prevent stress on the nerve putting the physician off his guard. A dental abscess,

when not directly pressed on by the root of the tooth, has been treated as neuralgia. A central abscess of bone, where there is no redness or swelling, has been diagnosed tarsalgia, rheumatism, etc. About the joints osteo-arthritis gives a characteristic picture. The rarefication of bones with osteophytes is clearly seen.

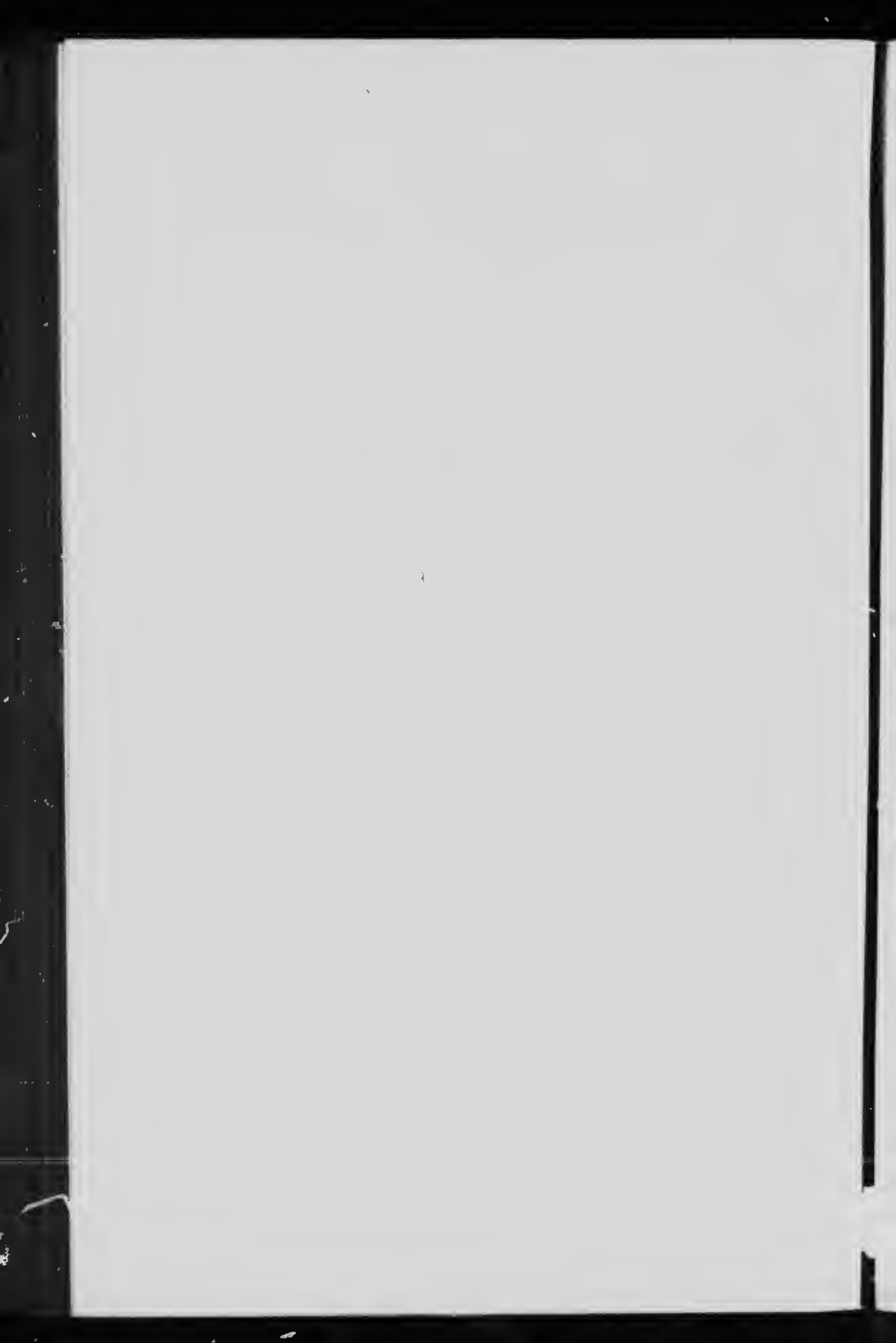
G. The **Vertebral Column** may be radiographed, and a picture of the spine obtained which gives a much truer idea of the extent of lateral curvature in scoliosis than examination of the tips of the spinous processes. Caries of the spinal column and cervical ribs may be demonstrated by radiography, and are at times the cause of obscure nervous symptoms.

H. Acromegaly, due to tumour of the hypophysis cerebri causes destruction of the sella turcica, which may be shown on the radiogram.

J. C. RANKIN.

### **YELLOW VISION (Xanthopsia).**

A yellow discoloration of all objects is often perceived by persons suffering from jaundice, and by those who have been taking santonin internally.



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