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SYSTEMATIC CASE-TAKING

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A PRACTICAL GUIDE TO THE EXAMINATION AND RECORDING OF MEDICAL CASES

FOR THE USE OF MEDICAL STUDENTS

BY

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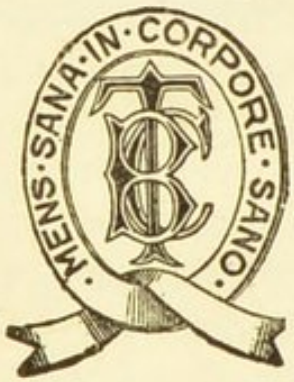

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PREFACE

WITHIN the compass of a small book I have attempted to furnish medical students with a systematic guide to the methods of examination involved in the process of case-taking. While it is certain that no mere book can take the place of personal instruction in the wards by the medical registrar or tutor, it is equally obvious that a methodical syllabus will serve to add completion and co-ordination to the student's examination of his case, and to his records of the same. Of the first importance in such a work is a description of the various means of investigation of disease ; but it is necessary, in order that the student should learn the relative importance of the facts he elicits, that some consideration should be given to the various symptoms, and to their comparative value in forming the necessary diagnosis.

I have therefore amplified the ordinary syllabus of case-taking into a short manual, which not only indicates the steps to be taken by the clinical clerk in order to obtain the information he has to record, but which also discusses, as concisely as possible, the diagnostic significance of the various symptoms discovered. There are, I am aware, many excellent books which deal specially with the symptoms of disease and the methods of examination ; but, judging from my own experience as a clinical teacher, it appears to me possible that a

small handbook dealing with the subject strictly from the case-taker's point of view may prove useful.

With few exceptions, the methods of examination referred to in this book are those which may be carried out in the ward or clinical room of a hospital ; the more elaborate bacteriological and pathological investigations cannot be described in a manual for clinical clerks, though the results of such pathological examinations must be duly noted in the case-sheet.

The scope of this book being limited to the examination and diagnosis of those affections which are usually treated in the medical wards of a general hospital, those conditions which belong to the surgical and special departments are only referred to when they have some diagnostic relation to cases of internal medicine.

H. L. McKISACK.

BELFAST,
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INTRODUCTION

In attempting the diagnosis of a medical case, one has to make a collection of all the facts bearing on the condition of the patient. The discrimination between those facts which are relevant to the case in hand and those which should not influence one's judgment upon it, is not always an easy task to the experienced, and to the novice is an impossibility. It is therefore universally the practice in medical schools to instruct the student to collect what may seem an unduly large mass of information regarding the case under examination, and to arrange and formulate his harvest of facts in a definite and methodical manner. At first, no doubt, there will be included in the case-sheet unnecessary and incorrect statements, but even from an early stage of his clinical clerkship the student, if he be possessed of ordinary powers of observation and industry, and if he follow the recognized rules of case-taking, will be able to present a fairly complete and accurate account of his cases. As his experience widens, he will be able safely to omit a certain proportion of the details which at first he must include. Systematic case-taking is the only safe way to learn thoroughly the art of diagnosis, and the time spent in this occupation is perhaps the best investment the student can make of his energy.

In the course of his examination the student may have

to put the patient to some inconvenience. This may be unavoidable, but it is one's duty to treat him with all possible consideration and patience, bearing in mind that what seems to us to be an inoffensive procedure or inquiry may to the uninstructed invalid cause apprehension or confusion.

Two means of investigation are open to the student, and both have to be taken advantage of to the fullest extent—namely, interrogation of the patient (and of his friends, if necessary) and physical examination. All that the patient has to tell must be seriously considered, but the examiner has ample scope for the exercise of his judgment as to how much is worthy of recording. Care must be taken to avoid being misled by inaccuracies of description or of fact. Statements involving a diagnosis should be recorded with some indication that it is unverified information—quotation marks serve the purpose. As far as possible, leading questions should be avoided, but at times they are necessary. The state of the patient's intellect, which will engage the attention of the student more closely when the nervous system is under special examination, should even at an early stage be carefully considered, in order to judge as to the value of the information obtained from him.

The physical examination should be comprehensive, and should be exhaustive in that system which one has reason to believe is especially involved.

SYSTEMATIC CASE-TAKING

CHAPTER I

PRELIMINARY INQUIRIES

Outline of the method recommended for case-taking—Family history—Personal history—History of the present affection—Symptoms, subjective and objective—Pain.

Outline of the Method recommended for Case-Taking.—Having collected all the information possible on the patient's family history, personal history, and the history of the complaint for which he has sought advice, a *general survey* of the present state of the individual is to be made on the lines suggested in Chapter II. The *different systems* are then to be separately examined. In each the subjective and objective symptoms are to be described, special attention being given to the organs which are obviously at fault. It is advisable to take up each system in rotation, and always in the same order. The following routine will be found convenient: (1) The *Thorax*, comprising the respiratory and circulatory systems; (2) the *Blood;* (3) the *Glandular system* (lymphatic, thyroid, spleen); (4) the *Abdomen*, including the alimentary system and the pelvic organs; (5) the *Urinary system;* (6) the *Nervous system.*

An important part of the clinical clerk's duty is to record the treatment of the case and its result, taking

notes at short intervals (not less than twice weekly) on the course of the malady. The diagnosis arrived at, and the issue, are to be recorded. In fatal cases, if a post-mortem examination be held, the condition found is to be noted on the case-sheet.

After having noted the name, age, address, occupation, and condition, whether married or single, a short preliminary statement should be made, relating what the patient complains of, or any outstanding circumstances which induced him to seek medical advice.

Family History.—It is always a difficult task to obtain accurate or reliable information on this subject, and among hospital patients it is often impossible. By the exercise of perseverance and tact it is, however, usually possible to gain some useful information concerning the hereditary tendencies of the individual. The effect of heredity is shown chiefly by the capacity of the individual to combat the innumerable disturbing influences which are constantly on the point of interfering with our tissues and organs and their functions. The man who has inherited a defective power of resistance to certain disturbing influences often proves on careful inquiry, to have had other near relatives similarly vulnerable. In many cases this susceptibility forms a widespread defect producing lesions of differing types, involving, however, structures of physiological or anatomical unity. The best example of this is seen in the group of diseases of the nervous system which may occur in a variety of forms in a family circle. Thus one member of the connection may be epileptic, another may show criminal or alcoholic tendencies, another insanity or hysteria. Again, several members of a family may show a defective resistance to the attacks of the tubercle bacillus, and various forms of tuberculosis are met with. Defects

of metabolism, renal disease, joint affections, and arterial changes, with consequent heart lesions, form another group of diseases often found occurring in members of a family in whom the so-called gouty diathesis is inherited. In some families cancer seems to occur with more than common frequency. The influence of heredity in cancerous affections is, however, denied by many authorities. A susceptibility to infection of the commoner exanthemata is at times noticed in families, while in others the resisting power is unusually effective. Of skin diseases, ichthyosis is perhaps the best instance of heredity. In psoriasis, eczema, scrofuloderma, leprosy, and syphilis, the hereditary influence has to be considered. Hæmophilia is a distinctly hereditary affection, occurring almost exclusively in males (about 90 per cent. of the cases are males), who transmit the tendency to bleed through non-bleeding daughters to male posterity. In certain organic diseases of the nervous and muscular tissues the influence of heredity is undoubted. Among these may be mentioned Friedreich's ataxia, cerebellar heredo-ataxia (Marie), progressive neural muscular atrophy, progressive muscular dystrophies, Thomsen's disease—all rare affections.

Personal History.—Under this heading the student must seek all the information bearing on the history of the individual which can in any way modify the course or issue of his malady. These, omitting family history, which has already been considered, are, shortly—(1) Age; (2) sex; (3) occupation; (4) environment; (5) habits, both personal and physiological (the latter, in the case of females, especially concerning the reproductive organs); and, lastly (6), an account of any illness from which he may have suffered before the onset of the present affection.

1. *Age.*—The age of the patient influences not only the course, but also the incidence of disease. *In infancy and childhood* the power of resistance to many disturbing factors may be defective; hence the digestive, the respiratory, and the nervous systems are frequently affected. Digestive disturbances, rickets, laryngeal and bronchial catarrhs, infantile palsies, and chorea are among the results. Imperfect resistance also gives rise to the exanthemata and to tuberculosis, which in childhood is likely to be seated in bones, joints, and lymphatic glands. In the *adolescent period* tubercular affections are still common, and especially phthisis, for which this is the favourite age ; acute rheumatism (causing greater damage to the heart in earlier than in later years) ; chlorosis ; hysteria ; gastric ulcer. By *middle age* the subject has acquired immunity from many affections, so that his liability to infections of all descriptions is diminished, though by no means abolished. On the other hand, his tissues have a tendency to undergo degenerative changes, and we find a large group of affections connected with changes in the arteries and defects of metabolism—viz., arterio-sclerosis, aneurism, cardio-sclerosis, gout, renal calculus, gall-stones, cancer, profound anæmias and blood diseases, and insanity. Tertiary syphilitic phenomena and the late parasyphilitic diseases—*e.g.*, locomotor ataxia, general paresis—now appear, and, generally speaking, the results of occupation, habits, and hardships, are met with most frequently in this period of life. With the arrival of *old age* we look for a further development of the degenerations.

2. *Sex.*—In childhood sex has but little influence on the incidence or course of disease, except in the case of hæmophilia, which commonly shows itself in boys in the first or second year. From puberty onward consider-

able difference is noticed between the sexes in respect to disease. Gastric ulcer, chlorosis, and exophthalmic goitre, are commoner in the adolescent female. In middle life women are more liable than men to gall-stones, cancer (chiefly owing to the frequency of breast and uterine disease), osteo-arthritis, neurasthenia, myxœdema, and movable kidney. At this period men suffer more than women from the effects of vicious habits, exposure, traumatism, overexertion, and dangerous occupations; hence they are especially liable to gout, arterio-sclerosis, aneurism, degenerative heart affections, cirrhosis of the liver, locomotor ataxia, and syphilitic lesions generally.

3. *Occupation.* — The broad distinction between an active and a sedentary individual often depends upon his occupation. Among hospital patients this is almost always the case, as in this class exercise is rarely taken for its own sake. The beneficial effects of an active life need not be here insisted upon; those who live an active, but not too strenuous, life are in the best state to offer an effective resistance to disease of all kinds. Certain occupations favour the development of morbid processes. Inhalation of dust, consisting of irritant particles, may cause lung affections, such as knife-grinders' phthisis or flax-dressers' bronchitis. Glanders, anthrax, lead-poisoning, result from exposure to the respective poisons. Writers' cramp, miners' nystagmus, miners' elbow, housemaids' knee, are examples of disorders directly due to occupation.

4. *Environment.*—Does the patient live in the country or in a town? If in the latter, is he a slum denizen or an inhabitant of a more healthy urban district? It would be interesting to ascertain, if it were possible, whether his house is clean, with fair sanitary arrangements, and if his bedroom is capable of ventilation; for

it is the absence of these conditions which gives rise to the surprising amount of ill-health, and particularly of tuberculosis, which one meets with in country-bred people. Damp climates, marshy districts, malarial regions, tropical countries, goitre localities, all exercise an influence on the individual and on his maladies.

5. *Habits.*—In this connection we investigate first his daily life. Is he a regular-living man, or is he of dissipated or irregular tendencies? The answer to this question is of the utmost importance in forming a prognosis, as the latter class is uninsurable. Is he a good sleeper, or is he subject to insomnia? Has he any recreations, and, if so, what form do they take? The habits of his body are to be ascertained—the regularity or otherwise of the bowels and bladder evacuation. In females the menstrual function and the reproductive history, if any, are to be recorded.

6. *Previous Illnesses.*—The occurrence of certain diseases—*e.g.*, influenza, rheumatism, gout, chorea, tonsillitis, asthma, and erysipelas—suggests a recurrence of these affections. The exanthemata, on the contrary, usually protect the subject from a second attack. In some diseases it is the subsequent developments or sequelæ, rather than a recurrence of the complaint itself, which we must look for. For example, rheumatism and chorea suggest endocarditis, scarlatina suggests Bright's disease, gonorrhœa causes arthritis, syphilis is the precursor of many affections of the nervous system and elsewhere, pleurisy may be the starting-point of a later-developing phthisis.

History of the Present Affection.—We now first approach the actual disease from which the patient is suffering, and the three chief items of information which we try to elicit from him or from his friends are—

(1) *When*, (2) *How*, and (3) *Why* did the malady attack him.

1. *When* did he first notice anything amiss? A correct reply to this question is absolutely necessary, and is often difficult to obtain. The patient must be encouraged to recall when he first felt ill, or when he left off work, or when his friends noticed him to be ailing.

2. The answer to the query *How* he was affected will inform us of the mode of onset and course of the disease. Was it sudden or gradual? Had he pain or loss of power? Was he feverish?

3. *Why* he was attacked by the disease is more than we can reasonably hope to learn from the patient. We may, however, find some useful hints in his theories as to the cause of his illness. There may be a history of exposure to cold and wet, or indiscretion in diet, an injury, or exposure to infection.

The first of these three questions is the most essential. Every possible fact concerning the *dates* of commencement and of any change, relapse, or exacerbation, must be recorded.

The Present Condition.—We have now to fully inform ourselves by inquiry and by observation as to the actual state of the patient. All the phenomena produced by morbid states and processes are spoken of as symptoms. Those of which the patient is conscious, but which the case-taker cannot verify by his own observation, are termed *subjective symptoms;* those which the observer can demonstrate by the various means of examination are called *objective symptoms* or *physical signs*. The former are less valuable than the latter, as the patient's sensations are often unreliable. In many cases, however, one gains valuable information by investigating

carefully the subjective symptoms, and they must never be neglected.

There are a number of symptoms which are mainly subjective, but which may also be recognized by the observer. For example, loss of muscular power is experienced by the patient, but careful examination will usually demonstrate it. Vertigo is felt, and also noticeable to the bystander. Dyspnœa, cough, nausea, and vomiting, may also be mentioned in this connection. It is the disorders of sensation that constitute the chief subjective symptoms, and these are, briefly, pain ; exaggerated common sensibility (*hyperæsthesia*) ; defective consciousness of sensory stimuli, involving the apparatus producing pain, heat, cold, common sensation, muscular and joint sensibility, and the special senses (sight, hearing, taste, and smell) ; abnormal sensations (*paræsthesia*), such as tickling, pricking, or tingling sensations ; and various vague discomforts, as the sensation of a lump in the throat, or fulness or tension or constriction in some part of the body. Lastly, a general indescribable discomfort or " malaise " may be experienced.

Subjective Symptoms.—Before proceeding to the examination of the patient, some record of the subjective symptoms must be attempted, and the student has to exercise patience and ingenuity in arriving at a true estimate of the patient's sufferings.

Pain.—This is the commonest of the subjective symptoms, and deserves a somewhat detailed consideration. The pain which is elicited by the examiner on pressing, rubbing, moving, or knocking the region—that is, *tenderness*—is really an objective symptom, but for convenience' sake is best discussed here.

General pain, involving the greater part of the body, s found in many fevers, and is especially characteristic

of influenza and smallpox; also of acute and chronic rheumatism.

Localized pain may be an aid in identifying the affected region, but it is often referred to a region quite remote from the source of the suffering. This is particularly the case in disease of the viscera, when pain may be felt in a superficial spot or area. This is often best elicited by stroking the surface with a blunt point—*e.g.*, a pencil. Here the stimulus from the diseased organ, proceeding to the sensory centre, spreads into neighbouring sensory paths in the cord, and is referred to the region from which the latter proceed. A summary of the regional pains and of their significance is as follows:

Headache (*Cephalalgia*).—Varieties: Migraine (sick headache, bilious headache, hemicrania) usually involves the branches of the fifth nerve, often begins with visual phenomena. Neuralgia, also one-sided, darting, often periodical. Dyspepsia and constipation: dull, throbbing, chiefly frontal, supra-orbital and bilateral. Neurasthenia: tense, constricting, often vertical, worst in morning, may be stabbing (*clavus hystericus*). Anæmia, resembles that of neurasthenia. Abdominal organs, and especially female organs of generation, when diseased, often cause sharp occipital headache. Affections of eyes, nose, and naso-pharynx: supra-orbital pain, often temporal or occipital. Intracranial disease: oftenest occipital, but may be general; rarely a guide to locality of disease. Syphilis (excluding intracranial growths): nocturnal pain, often occipital. Fever: in typhoid a frontal, vertical, or general headache characterizes the first week. Toxic states: nephritis (uræmia), chronic poisoning by alcohol, nicotine, lead, mercury, or impure air. Disease of cranial bones causes severe pain. If the disease is situated near the ear, usually

tubercular; other regions, especially frontal, often syphilitic.

Pain in the Face.—In addition to those conditions just referred to under Headache, carious teeth, tonsillitis, disease of facial bones (caries, cancer, antrum disease), and of the tongue, temporo-maxillary joint (rheumatism, osteo-arthritis, or gonorrhœal arthritis), and mumps, may cause severe facial pain.

Pain in the Neck.—Front and sides usually inflammations—adenitis commonest—also tonsillitis, mumps, pharyngitis, foreign body or cancer in throat, cervical caries, myalgia. Back of neck: myalgia (muscular rheumatism) commonest, cervical caries, meningitis, myelitis, tetanus.

Pain in the Throat.—A very common seat of infection, causing painful inflammatory lesions—*e.g.*, acute and chronic pharyngitis, follicular and phlegmonous tonsillitis, scarlet fever, influenza, mumps, diphtheria, ulcer of the fauces (follicular, cancerous, tubercular, syphilitic, or traumatic). (See Appendix I.)

Pain in the Thorax.—Upper anterior part (supra- and infra-clavicular regions): pleuritic pain—often, but not always, tubercular; gastric and diaphragmatic irritation is often felt here. *Behind sternum:* Digestive disorder (gnawing soreness and weight); bronchial and tracheal catarrh. Less common are—angina pectoris (oppressive and suffocating pain, usually extending to left shoulder and arm), disease of sternum or vertebræ (caries or syphilis), aneurism (pain often in back), mediastinal tumours, pericarditis. In *female breast:* disease of that organ (mastitis, cyst, cancer, cracked nipple), during menstruation and pregnancy, hysteria, disease of ovaries and uterus. In *precordial* region: gastric neurosis, catarrh, and flatulence; ulcer; cancer

of stomach ; pericarditis (often stabbing and sharp, and tender over lower part of sternum) ; aortic disease, with which (or even without obvious cardiac disease) may be associated angina pectoris ; pseudo-angina, resembling true angina (in anæmia, hysteria, debility). In *axillary* and *infra-axillary* regions : pleurisy (sharp, stabbing, aggravated by movement or active breathing), pneumonia (pain due to pleurisy), pleurodynia (muscular rheumatism), intercostal neuralgia, herpes zoster (pain may be the first symptom, and is neuralgic in character), flatulence, constipation, stomach disorders, may cause pain here. In the *shoulders :* rheumatism (articular or muscular) ; affections of liver, stomach, bowels, aorta ; pleurisy, apical pneumonia, and phthisis. In *infra-scapular* regions : pleurisy, pneumonia, stomach, spleen, and liver affections, movable kidney (commonest on right side), gastric ulcer (usually close to left side of eleventh and twelfth dorsal vertebræ), renal calculus (one-sided and aggravated by percussion), lumbago, acute nephritis, constipation, flatulence. In *inter-scapular* region : stomach affections ; caries of the vertebræ (commonest in this region of spinal column) ; myalgia, or muscular rheumatism, is less common here than in shoulders or loins ; aneurism of aorta (boring, persistent pain).

Pain in the Vertebral Column.—Lateral curvature (scoliosis) causes an ache in the back ; hysteria causes pain or tenderness on pressure over vertebral spines ; caries of the vertebræ, commonest in middle or lower dorsal regions, painful on pressure, and may produce symptoms of pressure on spinal cord and irritation of nerve roots ; fevers—*e.g.*, influenza, smallpox ; spinal affections —*e.g.*, meningitis, myelitis, locomotor ataxia, tetanus, syringomyelia ; a variety of abdominal diseases, such as

gastric ulcer, cancer of the liver, inflammation and cancer of the pancreas, affections of the uterus and other pelvic organs ; aneurisms of the thoracic or abdominal aorta ; mediastinal tumours. Pain over the *sacrum* and *coccyx* may be due to rectal and anal irritation (cancer, piles, fissure or fistula), or to affections of other pelvic organs (prostatitis, sexual excesses, uterine disease, ovaritis, pelvic cellulitis) ; to sciatica, sacro-iliac disease, hip-joint disease, neuralgia of the coccyx (coccygodynia).

Pain over the spine may be elicited by pressure, percussion, or by the application of heat. This especially applies to inflammations of the spinal column and canal —*e.g.*, meningitis, spondylitis, myelitis.

Pain in the Abdomen.—In the *hypochondria :* movable kidney (dragging, sickening character, worse in upright posture), renal calculus (severe, colicky, radiating downwards towards the groin, testicle, or pubes) ; pleurisy (pain may be referred to epigastric, umbilical, or lumbar regions) ; on right side : liver affections (active and passive congestion, cirrhosis, cancer, abscess), gall-stones (paroxysmal, severe, in epigastric, umbilical, and lumbar regions) ; on left side : gastric catarrh, ulceration, flatulence, enlargement of spleen, and perisplenitis. In the *epigastrium :* stomach affections chiefly ; a sharp, cutting pain, in one definite spot, felt soon after food, with or without a corresponding painful spot in the back, suggests gastric ulcer ; a more persistent pain, also aggravated by food, is felt in gastric cancer ; dyspepsia causes pain similar to gastric ulcer, but less localized and less cutting in character ; duodenal ulcer gives pain later in the digestive process, localized usually to the right and somewhat above the umbilicus ; gall-bladder and liver affections (see above) may be felt here, as also the pain of appendicitis early in the attack ; pneumonia, diaphrag-

matic irritation (pleurisy, violent coughing, or vomiting), aneurism, disease of pancreas or of vertebræ, are less frequent causes of epigastric pain. In *lumbar* and *iliac* regions: affections of large intestine—*e.g.*, fæcal accumulation (chiefly left iliac), colic, stricture of gut, volvulus (often left-sided or umbilical), colitis, appendicitis; at first pain is epigastric or general over abdomen, later restricted to right iliac fossa (McBurney's point), where the chief tenderness is located; intussusception (often umbilical pain also); typhoid fever usually causes slight pain in right iliac fossa; floating kidney is commonest on right side, causing pain in iliac or umbilical region; hernia; varicocele; renal colic (see above); pelvic disease; tubercular or other ulceration of the bowel. In the *umbilical* region: cancer or tuberculosis of omentum; umbilical hernia; strangulated inguinal or femoral hernia often causes pain to be chiefly umbilical, as is also the case at times in perforated gastric ulcer and appendicitis in the beginning of an attack; gall-stones, floating kidney, colic of intestinal irritation and of lead-poisoning, all cause umbilical pain. In the *hypogastric* region: urinary bladder affections (cystitis, stone, tubercle, cancer), diseases of female genital organs, and inflammation of pelvic tissues. In the *abdomen generally:* peritonitis (with rigidity of abdominal muscles, tenderness, and distension); most of the painful conditions already mentioned as occurring in one or other region of abdomen may at times be generalized; the pains due to irritant poisoning, cholera, gastric crises of locomotor ataxia, and Dietl's crises (in movable kidney), are usually felt all over the abdomen.

Pain in the Gluteal Region.—Sciatica (dull, boring pain, with acute attacks, one-sided, pain and tenderness over trunk and distribution of nerve), hip-joint disease,

sacro-iliac disease, lightning pains of locomotor ataxia (may be in leg, but usually worse in buttocks and back of thighs), disease of pelvic organs. Pain in *external genitals :* usually due to local disease ; in testicle may indicate stone in kidney or ureter, or may be due to neuralgia or neurasthenia ; at the point of the penis, pain may result from stone in the bladder, but more commonly from local disease. Pain in the *anus :* piles cause a dull, throbbing, heavy pain, while that from fissure is usually sharp and acute. Pain in the *groin* may be due to renal colic, intestinal colic, hernia, varicocele, enlarged inguinal glands, neuralgia, pelvic disease, etc.

Pain in the Arms and Legs, when not the result of local disease (phlebitis, bone and joint affections, etc.), may be a portion of general medical disorders : rheumatism ; locomotor ataxia (usually in legs and trunk) ; gout and osteo-arthritis (commonly in hands) ; peripheral neuritis, etc.

The remaining disorders of sensation, as well as other subjective symptoms referred to above, will be more conveniently discussed later, when the examination of the nervous, respiratory, and other systems, is under consideration.

CHAPTER II

GENERAL EXAMINATION

Aspect—Eruptions—Posture—Gait—Shape and size of the body —The tongue—Temperature—Respiration—The pulse.

General Examination.—We have now come to the actual examination of the patient, and to the study of the signs of disease thereby disclosed. A general survey of the condition, noting carefully any departure from a state of health, is to be made ; then a systematic investigation, by all the means at our disposal, of the various regions, systems, and organs, is to be undertaken. While one is obliged to examine with more minuteness that organ which is obviously abnormal, as in many cases the history and general examination discloses, it is none the less necessary to investigate all the organs possible, so that no obscure defect may be overlooked.

In examining a case we must make use of our best powers of observation, utilizing and educating our different senses, and availing ourselves of any clinical apparatus which is found suitable, supplementing these, where necessary, with more specialized laboratory methods.

In the general examination of the individual, observe and record any departure from what you believe to be healthy in colour, aspect, posture, gait, shape, and condition of nutrition ; the state of the teeth and mouth ;

all obvious disorders of movement, speech, manner, or intelligence. Any general evidence of departure from health should be here observed, so the state of the tongue, the temperature, the frequency and character of the respirations, and the quality of the pulse, may with advantage be investigated at this early stage of the examination.

The Aspect of the patient may convey some information as to his health. Emaciation or the contrary condition of obesity may indicate merely personal habit, or may be signs of disturbed metabolism ; a pale or a florid complexion may be characteristic of the individual, or may equally point to some departure from health. The important point is to ascertain, if possible, whether there has been any change in these conditions since the onset of the malady. The expression of the patient's face in some instances is of help. (1) A dull, apathetic aspect, with a flushed, heavy appearance, raised temperature, and in severe cases delirium, dry tongue, and sordes on lips and gums, is found in typhoid fever and the so-called *typhoid state* from any cause ; a dull, apathetic expression, without accompanying signs of serious illness, is seen in paralysis agitans (*Parkinson's mask*), in myxœdema, and in children suffering from naso-pharyngeal obstruction. (2) An alert, active aspect is characteristic of most fevers, where the illness has not reached the state of prostration referred to above, as in pneumonia, phthisis, the early stage of typhoid fever, etc. (3) An anxious expression is seen in many acute inflammatory states, particularly in those of the abdominal organs, and in difficulty of respiration. (4) The nervous, self-conscious aspect is seen in hysterical or neurotic patients. (5) Facial paralysis, ptosis, exophthalmic goitre, or mumps, give characteristic aspects.

(6) The sunken eyes, deeply-lined face, sharp nose, pale, livid, or cyanosed skin, known as the Hippocratic facies, is seen in moribund cases, and in serious, but not necessarily fatal, diseases of the abdominal organs, especially in intestinal obstruction, peritonitis, and cholera.

More definite changes of the skin are to be noted. Thus a *yellow colour* of the skin, if accompanied by a similar tinge in the conjunctiva and the presence of bile in the urine, indicates jaundice, caused by the presence of bile-pigment in the blood. A yellowish colour, without, however, the conjunctival tinge, may be found in profound anæmias, and especially in pernicious anæmia. A greenish-yellow is seen in the skin of young women suffering from chlorosis, and the pallor of malignant disease has often a muddy yellowish shade. The skin may be a shade of blue, ranging from leaden white to purple (*cyanosis*), due to imperfect oxidation of the blood, as seen in respiratory and circulatory disorders. Vasomotor changes (the cyanosis of paralyzed limbs, of cold, of hysteria and Raynaud's disease), congenital heart disease, pressure of tumours interrupting venous return, may cause localized cyanosis. Abnormal pigmentation of the skin is a common disorder. It is seen in pregnancy (patches on the face, *chloasma uterinum*, and darkening of nipples and linea alba); from irritation of the skin (blisters, dirt, vermin); in Addison's disease, a bronzing of skin (especially in the regions where pigment is normally deposited) and of mucous membranes. A somewhat similar bronze colour may occur in Hodgkin's disease, arsenic-poisoning, cirrhosis of the liver, diabetes, and phthisis. Melanotic sarcoma causes a grey or black discoloration; a bluish or grey colour results from prolonged administration of silver salts; syphilitic skin affections and ulcerations are apt to be pigmented

to a brownish shade ; nævi, warts, and other skin affections, may show considerable deposits of pigment.

Eruptions.—An immense variety of skin eruptions may be observed. In many cases these are evidence of a local disease of the skin, while in others they may indicate an affection of other organs or some toxic condition of the blood. No useful description of diseases of the skin can be attempted here, and the only skin eruptions to be referred to are those which assist in the diagnosis of general medical affections. The following types of eruption may be noticed : Erythema, roseola, macules, papules, vesicles, pustules, hæmorrhage, wheals, squamæ, dermatitis.

1. *Erythema*, red discoloration, usually in large patches or areas. It is seen in the following : Scarlatina, appears about the second day of the disease on chest or abdomen, neck or face. Rötheln (German measles), red spots appearing on the first or second day of illness on the face ; on the body the spots join to form red areas, while on the face they often remain discrete. Smallpox and typhoid fever often show a preliminary erythema before the characteristic rash appears. Erysipelas, a bright red rash with sharply-defined edge, appears within twenty-four hours after the onset of the illness. Septic poisoning may cause an erythematous spotted or patchy rash ; a similar eruption may occur as a result of intestinal disorders, and occasionally after a soap-and-water enema. Infants the subjects of inherited syphilis often present a red rash on the buttocks, while acquired syphilis has an erythematous or macular rash (more commonly the latter) on the abdomen and thorax, rarely on the face or hands, occurring about six weeks after the appearance of the primary sore. Erythema nodosum, raised, painful, red areas, chiefly occurring on

the front of the leg, below the knee, and on the extensor surface of the arms. This is often associated with rheumatism, which, however, may be itself a cause of various erythematous eruptions on the body. Certain drugs cause red rashes—*e.g.*, antipyrine, sulphonal, iodine and the iodides, iodoform, bromine and the bromides, opium, belladonna. Cubebs and copaiba may cause erythema, but more often macules. Quinine, chloral, arsenic, mercury, salicylate of soda, chlorate of potash, turpentine, boric acid, benzoic acid, strychnine, may be mentioned as chief among the drugs which occasionally produce a red eruption. The injection of antidiphtheritic and other serums may be followed by erythema or a spotted rash.

2. *Roseola or Rose-Red Spots.*—The eruption of typhoid fever is a good example. Small, round, slightly raised spots, disappearing on pressure, commencing to appear on abdomen and lower part of thorax about the beginning of the second week of the disease. The secondary rash of acquired syphilis may be of this description, but there is usually a brownish tinge. In relapsing fever similar spots may be seen.

3. *Papules* and *Macules—i.e.*, spots or blotches, raised somewhat, and red from capillary injection, usually disappearing on pressure. Seen in measles, usually on the fourth day of the illness, in clusters, first on the face, spreading downwards, and lasting four or five days. The eruption is also seen inside the mouth (on palate and cheeks). Inside the lips and cheeks whitish or bluish spots surrounded by a red areola (Koplik's or Filatow's spots) may often be found before the cutaneous rash is well established. Rötheln (German measles) and syphilis, see above under Erythema. In smallpox, shotty papules, at first on the wrists and forehead, appear about the

third day of the disease. The drugs mentioned above as causes of erythematous rashes will at times cause also papular and macular eruptions. Injections of diphtheritic and other antitoxic sera may produce the same effect.

4. *Vesicles or Blisters.*—In smallpox, the shotty papules mentioned above become vesicles about the sixth day of the disease. Chicken-pox shows papules on the second day of the illness. These quickly become vesicles, soon to turn into pustules. They appear first on the face and trunk, and only occur on the arms and hands after they have freely invaded the trunk (in which respect they differ from the spots of smallpox). Herpes may arise in the course of general disease. The best example is herpes facialis, occurring near the mouth or nostrils in pneumonia, and also in catarrhal affections of the respiratory tract. It may also be seen in typhoid fever and in epidemic cerebro-spinal meningitis. Herpes zoster occurs as groups of vesicles along the course of one or more cutaneous nerves. Sudamina are minute blisters occurring where sweating is profuse—*e.g.*, rheumatism, phthisis. As an eruption due to drugs, vesicles are less common than those already mentioned. They may be due to iodides, bromides, arsenic, salol, or copaiba. All these vesicular eruptions tend to become pustular in the course of their development.

5. *Wheals* (urticarial eruptions) most frequently arise as a result of disturbance of the digestive functions; also from septic absorption from the digestive tract or other surface, serum injections, soap-and-water enemata. Many of the drugs already mentioned cause eruptions of this character.

6. *Pustules.*—The papular and vesicular eruptions often become pustular (chicken-pox, smallpox, herpes).

The pustular drug eruptions are common in iodine, bromine, and arsenic poisoning.

7. *Hæmorrhages.*—Small spots, red or dark in colour, which do not disappear on pressure (*petechiæ*), streaks of similar character (*vibices*), and larger patches of discoloration (*ecchymoses*), result from the escape of blood from the vessels into and under the skin. A good example is seen in purpura, with petechiæ, vibices, and ecchymoses all over the body, but usually best marked on the legs. This occurs in a variety of severe toxic states of the blood—*e.g.*, pyæmia, septicæmia, gonorrhœal infection, ulcerative endocarditis—or it may be a so-called idiopathic or primary disease (purpura simplex), of which a severe form is purpura hæmorrhagica or morbus maculosus of Werlhof, in which there is not only bleeding into the skin, but also in many other regions and organs. Purpura associated with articular rheumatism is known as peliosis rheumatica, or Schönlein's disease. Signs of gastro-intestinal irritation may be added to this condition, chiefly among children, the affection being then known as Henoch's purpura. Scurvy, hæmophilia, leukæmia, and splenic anæmia, may be mentioned as conditions associated with cutaneous hæmorrhage. In typhus fever the eruption is petechial, occurring first over the upper region of the thorax and abdomen about the fifth day of the disease. In measles and smallpox, when severe, the eruption may show signs of cutaneous hæmorrhage. The pustules of iodide or bromide eruption may be filled with blood, or petechiæ may occur. Petechiæ may also be seen in cases of antipyrine and sulphonal poisoning.

8. *Inflammation of the Skin* (dermatitis) is more commonly found in local affections of the skin. It may occur, however, in gout, syphilis, arsenic and iodine poisoning.

9. *Desquamation* occurs as a fine branny peeling of the superficial skin layers after most cases of inflammation or hyperæmia of the skin (measles, rötheln, erysipelas). In scarlet fever the skin peels off in larger flakes, especially where it is thick, as on the palms of the hands and the soles of the feet.

The Posture is often suggestive. The patient is unable to lie down in bed, owing to difficulty in breathing (*orthopnœa*), as observed in serious heart affections, asthma, emphysema, mediastinal tumour. He lies on his back—in a comfortable posture when he is not seriously ill ; in an uncomfortable attitude, slipping downwards in the bed, in states of prostration ; with one leg drawn up and abdominal muscles of that side rigid, in inflammatory affections of one side of the abdomen ; with both legs drawn up, the inflammation is more widely spread in the peritoneal cavity. He lies on his face : painful and non-inflammatory abdominal disorders—*e.g.*, renal, intestinal, or gall-stone colic. He lies on his side : he may be in a condition of *cerebral irritation*, with limbs flexed at every joint, resenting any attempt to move him—a condition seen in irritative intracranial lesions—*e.g.*, meningitis, hæmorrhage, pressure. On his side with head retracted suggests meningitis ; lying on his side, with obvious difficulty in breathing, may indicate pleurisy or pneumonia of the side upon which he is lying. A rigidity of posture occurs in tetanus and strychnia-poisoning. The dorsal muscles may prevail, causing an arching of the back, so that the body may rest upon the head and heels (*opisthotonos*) ; a forward curve may occur (*emprosthotonos*) ; the curve may be to one side (*pleurosthotonos*) ; the trunk muscles may antagonize each other, so that the body is straight and rigid (*orthotonos*).

Gait.—The mode of progression is to be noted, and

the following different types of movement may be distinguished: The *spastic gait*, characterized by stiffness in the motion, due to exaggerated muscular tone, especially of the extensor muscles. It occurs in affections of the central nervous system in which the leg reflexes are intensified (see Chapter IX. and Appendix XI.). The *ataxic gait*, due to defective co-ordination of muscular action. The movements of the limb are inaccurate and uncertain, and particularly so if he removes his eyes from the ground at his feet—a condition best seen in locomotor ataxia. The ataxic gait of tabes may be of a *stamping* character—when he raises his limb with unnecessary force, bringing it down again with a stamp. A greater degree of muscular inco-ordination is seen in the *reeling gait* of cerebellar disease, affections of the middle ear, alcoholic intoxication, etc. *Pseudo-ataxic* or *paretic gait* is seen in cases of muscular weakness, and particularly in peripheral neuritis affecting the legs. Here the foot tends to hang ("dropped foot"), and the knee is unduly raised in order that the toe may clear the ground (*steppage gait*). As a rule, some inco-ordination due to defective muscular sense is also found in these cases. A *festinating gait* is occasionally seen in paralysis agitans, where the patient has a tendency to fall forward, and in order to preserve his balance he gradually increases the speed of his progression. A *waddling, rolling gait* is seen in cases of abdominal tumours, ascites, pregnancy, obesity, and in pseudo-hypertrophic paralysis. Every variety of *lameness* results from lesions of one or both legs.

The Shape and Size of the Body.—A general enlargement may be dropsy (*anasarca*), but is more commonly excessive fat deposit. The thorax may be generally enlarged in emphysema, or present local enlargement

from tumour or aneurism. Abdominal swelling may be due to obesity, tumour, pregnancy, ascites, flatulent distension. Enlargement of the head is seen in rickets, hydrocephalus, and in acromegaly (head and face). The limbs are enlarged in œdema and in surgical conditions (tumours, inflammations). Medical diseases of the limbs are rheumatism, erythema nodosum, osteo-arthritis, arthropathies of trophic nervous origin, and pseudo-hypertrophic muscular paralysis.

Diminution in size due to wasting disease may be mere atrophy of disuse; it may be the emaciation of imperfect nutrition, or of one of the rarer muscular dystrophies, or it may be the result of disease of certain regions of the nervous system.

Disorders of movement, speech, and intelligence, being in most cases indications of disturbances of the nervous system, are at this stage to be only generally surveyed, their detailed examination being taken again when the nervous system is reached (Chapter IX.).

The Tongue may now be examined as an index to disease in the body generally. Observe the colour, coating (if any), tone, size, and movements. Its *colour* is pale in anæmic conditions; red in tubercular disease, in the early stage of scarlet fever, in the typhoid state (when it is also dry); bluish in cyanosis; dark in Addison's disease; yellow in jaundice (especially on its under-surface). A *coating* of thin whitish "fur" may occur in health, in digestive disorders, and in most feverish states. If thick, yellow, or brown, it may result from the same, but more severe, causes, or from the abuse of alcohol or tobacco. In typhoid fever the coating is often thick over the dorsum, but thin or absent on the edges and tip and anterior part of the middle line. A dry, brown, or dark fur indicates prostration and fever,

and is seen in the "typhoid state" from any severe disease. A whitish coating, through which the bright red papillæ project (the *strawberry tongue*), occurs in fevers, and especially in the early stages of scarlet fever. Inflammations of the mouth (carious teeth, tonsillitis, etc.) cause a thick coating. The fur may be coloured by drugs (black by bismuth, iron, or charcoal; white by carbolic acid) or by food. A defective *tone* or flabby state of the tongue, showing indented teeth-marks, indicates debility and anæmia. Increase in *size* of the tongue is usually due to inflammation (glossitis); it is seen also in cretinism and myxœdema. Decrease in size is found in serious disease with prostration—*e.g.*, typhus fever, advanced typhoid fever, profuse hæmorrhage, cholera. Lesions of the hypoglossal nerves or of their nuclei cause marked atrophy of the tongue. *Tremor* of the tongue occurs in healthy persons of a neurotic temperament; also in many disorders—*e.g.*, acute and chronic alcoholism, prostration, multiple sclerosis, general paralysis of the insane, bulbar paralysis. *Paralysis* of the tongue is shown by inability to protrude the organ properly. If unilateral, the tongue is deviated toward the affected side when protruded; if the lesion causing the paralysis be nuclear or infranuclear, the tongue will be atrophied and wrinkled; if, on the other hand, the lesion be above the hypoglossal nucleus, it is usually one-sided, and there is no marked atrophy of the affected part. A bilateral atrophic lingual paralysis occurs in bulbar paralysis; a similar condition without atrophy is produced by a two-sided lesion above the nuclei—a rare condition termed pseudo-bulbar paralysis.

Tumours, ulcers, and other lesions of the tongue, are usually local affections, without general diagnostic interest. As exceptions to this may be mentioned the

lacerations of the tongue by the teeth clenched in an epileptic fit, and the small ulcer of the frenum caused by the lower incisors in whooping-cough.

Temperature.—The temperature of the body is most suitably taken in the mouth ; it is more accurate, but less convenient, when taken in the rectum ; the least accurate, but most convenient, method is the axilla, where the temperature may be 1° lower than in the mouth. In the case of children the groin is a very suitable place to make the observation. A temperature between 98° and 99° F. may be regarded as normal. Acute inflammatory affections are almost always accompanied by a rise of temperature above normal (*pyrexia*). The range of the temperature, its mode of onset, duration, and defervescence, are often characteristic of the disease of which it is an important sign.

1. The *invasion* or *onset* of the fever may be abrupt, in which case it is often accompanied or preceded by a rigor, or in children sometimes by a convulsion. Sudden onset occurs in croupous pneumonia, scarlet fever, tonsillitis, influenza, erysipelas, digestive disorders in children. 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.

2. The *height of the fever*, or *fastigium*, may be *continued* without much fall till the end of the feverish period is near, as in pneumonia, typhoid fever. A *remittent* temperature has a daily fall, approaching, but not reaching, normal. Examples: phthisis, suppuration, pyelitis, septic infection, typhoid fever in the third week. *Intermittent* fever has periods free from fever. It may occur in hectic conditions and suppuration, in sepsis, and typically in malaria. If the intermission lasts more than a day, it is termed *relapsing* fever. Influenza, typhoid fever,

relapsing fever, and malaria, may show this type of pyrexia.

3. The *termination* of the high temperature may be sudden, falling to, or nearly to, normal in about twenty-four hours. This is known as a *crisis*, and may be seen in croupous pneumonia, measles, chicken-pox, tonsillitis, relapsing fever, malaria. A sudden fall of the temperature below normal may be an unfavourable sign (see below). A defervescence by *lysis* or gradual fall of the temperature is the termination of most fevers—*e.g.*, rheumatism, typhoid fever, broncho-pneumonia, scarlet fever, pleurisy, septic infections. A termination by lysis in diseases which usually end by crisis often indicates the supervention of some complication.

Hyperpyrexia, or a temperature above 106° F., may occur in the course of some of the fevers, especially in acute rheumatism, typhoid fever, malaria, and sunstroke. It is commonly seen as the termination in fatal cases of injuries of the brain and cervical portion of the spinal cord, in typhoid fever, scarlet fever, tetanus, etc.

Subnormal Temperature.—In certain chronic diseases the temperature is commonly below 98° F. A sudden fall from fever height to below normal is one of the signs of collapse. The pulse is more frequent, becomes small, of low tension, and often irregular ; there may be difficulty in breathing, with sighing, frequent, or slow respirations. In all these respects the sudden fall of temperature differs from that of a favourable crisis. Collapse occurs in a variety of serious conditions, of which may be mentioned heart failure in acute diseases, such as pneumonia or scarlet fever ; bleeding from typhoid, gastric, or duodenal ulcers, from an extra-uterine pregnancy, or from the lungs ; perforation of viscera, as in typhoid fever, gastric or duodenal ulcer.

Respiration.—The frequency of this act is to be noted, and any difficulty experienced by the patient may be here recorded. The more detailed examination of the respiration may be deferred till the special examination of the respiratory system is undertaken (see Chapter IV.).

The Pulse.—The pulsation of the radial artery is to be examined, and information sought on the following points: (1) The tension; (2) the condition of the arterial walls; (3) the frequency and rhythm; (4) the volume; (5) the duration of the pulse.

Methods of Examining the Pulse.—By inspection one can at times observe the pulse. Pulsations of more than ordinary amplitude may be visible in the radial artery, as commonly occurs in aortic incompetence. Here the vessel is often tortuous, and may be seen to move with each beat (*movable or locomotive pulse*). Digital examination is best accomplished by placing three fingers on the artery at the wrist, the distal and proximal fingers controlling the pulsations, and the middle finger observing the character of the pulse. By rolling the vessel under the fingers the condition of its walls may be ascertained.

The *sphygmograph* gives a graphic record of the beats. The tracing made by the instrument represents the wave which has reached the radial artery one-tenth of a second after it was created by the contraction of the left ventricle. The up-stroke of the lever is due to the *primary* or *percussion wave*. Owing to the momentum acquired by the lever, it rises too high, and in its fall again it meets the termination of the wave, which forms a little elevation, generally known as the *secondary, tidal,* or *predicrotic wave*. This is followed by an elevation of the lever causing the *dicrotic wave,* which immediately follows on the closure of the semilunar cusps, and is the

result of recoil from the closed aortic valve. The depression immediately preceding the dicrotic wave is termed the *dicrotic* or *aortic notch*. Other small secondary waves may be seen in the down-stroke of the tracing, the significance of which is uncertain. In disease of the heart, kidneys, and other organs, one finds considerable departures from the character of pulse-tracing just described.

The *sphygmomanometer* is an instrument devised to determine, in terms of the height of a column of mercury, the degree of tension of the systemic arteries. There are now many varieties of instrument for this purpose. That of Riva Rocci, modified by Martin, Oliver, Hill, and others, is probably the best for clinical use. It consists of a rubber sleeve, about 5 inches wide, forming an air-tight bag, and covered by an inextensible leather or canvas band. The rubber bag is connected with an air-pump and also with a manometer. The bag is fastened round the patient's arm just above the elbow, air is pumped in until the pressure is sufficient to obliterate the pulsation at the radial, and the manometer shows the pressure attained in millimetres of mercury ; or an excessive amount of pressure (more than enough to obliterate the pulsations) having been introduced into the bag, the air is allowed to escape slowly, and as soon as the pulsations are felt to return in the radial, the manometer reading is recorded. This is taken as the systolic tension, and normally the mercurial column reaches the height of from 100 to 140 millimetres. During all these procedures the mercury in the manometer is seen to oscillate with each pulsation. At a certain point of pressure, lower than that taken to indicate the systolic tension, the pulsating excursions of the mercury are at their maximum amplitude. This

point, which is difficult to determine, is to be noted as the *diastolic tension.*

Other forms of sphygmomanometer compress the radial artery just above the wrist (von Basch's) or measure the pressure which is sufficient to prevent the blood returning to a finger from which the blood had been previously pressed (Gärtner's).

1. *Tension* of the arteries may be investigated by any of the methods above mentioned; by education the fingers can help us to decide if the pulsations are more or less easily abolished by pressure than normal. In cases of high tension the sphygmograph forms a characteristic curve. The up-stroke is somewhat sloping; the line is sustained 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. 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. In some cases of low tension—*e.g.*, fevers—the dicrotic wave may be easily felt by the examining finger—the so-called *dicrotic pulse.*

The degree of tension depends on—(1) the strength of the ventricular contractions; (2) the volume of blood in the arteries; (3) the peripheral resistance; and (4) the elasticity of the arterial walls. The removal of blood from the arteries 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. Diminished elasticity occurs in arterio-sclerosis, with a consequent rise of arterial pressure. In kidney affections, and especially in the small red kidney, the increased peripheral resistance resulting from contracted, and sometimes from sclerotic, arteries, as well as cardiac hypertrophy, causes in most cases an increase in the arterial tension. Cardiac disease commonly causes low tension; but when arterio-sclerosis is present, a fairly high tension may be maintained. In the case of aortic incompetence the powerfully contracting left ventricle causes a momentary systolic high tension, followed by mean and diastolic low tension, the so-called *collapsing, water-hammer,* or *Corrigan's pulse* (*pulsus celer*). In mitral obstruction or regurgitation the amount of blood which reaches the arteries with each beat is diminished, and the pulse is, in consequence, of low tension.

2. *The Condition of the Walls of the Artery.*—If the walls of the vessel can be recognized by the finger while the pulsations are abolished by compression higher up the arm, then we may conclude that the vessel walls are unduly thickened. In old age this may be looked on as the normal state—senile degeneration. In younger subjects it is usually an indication of arterio-sclerosis, often associated with chronic sclerotic and degenerative changes in the kidneys. Another cause of thickened arteries is syphilitic arteritis, occurring, as a rule, in young subjects.

3. *The Frequency and Rhythm of the Pulse.*—Considerable differences in the rate of the heart-beat occur in healthy individuals, but in most cases the adult frequency may be stated as 70 to 75 per minute. In infancy the rate commences at about 120 to 140, decreasing gradually till adult age is reached. In old age the frequency again increases somewhat. Emotional and muscular activity, even in slight degree, increase the pulse-rate. It is a little quicker on the average in women than in men, in short people than in tall, during action than in rest of the digestive organs.

A rapid pulse is seen in *fevers;* the increased pulse-rate is usually in proportion to the rise in temperature. Some exceptions to this rule may be noted. In scarlatina the increased frequency of the pulse is a marked feature, while the rise in temperature is only moderate; in typhoid fever it is the reverse which obtains. A slow pulse with raised temperature is seen in intracranial disease in which there is inflammation combined with intracranial pressure, as is often the case in tuberculous meningitis and cerebral abscess; the opposite condition of low temperature with rapid pulse is found in collapse.

A frequent pulse without a corresponding rise of temperature in cases of fever is of more serious import than a moderate pulse-rate with a high temperature.

The ratio of the pulse-rate to that of respiration remains in most cases fairly constant at about 4 to 1. The most notable exception to this is found in pneumonia, where the pulse-respiration ratio may be 2 to 1, or even nearly equal.

A rapid pulse without fever occurs in exophthalmic goitre, endocarditis and its resulting valvular disease, pericarditis, chlorosis and other anæmias, hysteria, general debility, abuse of tobacco, alcohol, and tea.

Certain drugs, of which atropine is the best example, quicken the pulse.

Attacks of frequent pulse-beats, lasting perhaps an hour or more, and occurring at irregular intervals and often on slight provocation, may go on for long periods, often for years. The condition is known as *paroxysmal tachycardia*. By some writers the term " tachycardia " is used for all cases in which the pulse-rate exceeds 120 per minute, while others restrict the use of the word to those paroxysmal and recurring cases just mentioned.

The opposite condition, in which the pulse is unduly infrequent (*bradycardia*), occurs in cachectic states ; in high arterial tension ; in aortic stenosis (unlike most other valvular diseases) ; in conditions producing intracranial pressure, especially when the latter has rapidly developed. In other affections of the nervous system bradycardia is at times observed—*e.g.*, melancholia, mania, epilepsy, sunstroke ; in myxœdema ; certain poisons in the blood—*e.g.*, digitalis, opium, carbon monoxide, lead, bile, urea.

Disturbance of the rhythmical recurrence of the heart-beat has two sources of origin : disturbing influences acting upon the nervous connections between the heart and the central nervous system—that is, the vagus and the sympathetic nerves ; and some interference affecting injuriously those qualities which healthy heart muscle must possess in order to insure regularly recurring contractions—those qualities being rhythmicity, excitability, contractility, conductivity, and tonicity.

In most cases the irregularity does not follow any definite type ; some, however, are only partially irregular. A common form of the latter is the *intermittent pulse* (*pulsus intercidens*). Here a beat is apparently missed at intervals more or less irregular. Whilst the

radial pulse is being digitally examined, the stethoscope is placed over the heart ; the beat immediately preceding the missed beat in the radial is noticed by auscultation to be apparently doubled, or the doubling may be at times observed by the fingers on the radial artery. This doubled beat is due to the occurrence of a *premature* or *extra systole* of the ventricles at a moment when the excitability of the muscle has not reached its most effective period—that is, it occurred too early in the diastole—and hence the resulting contraction was so weak that the pulse-wave barely reached the wrist. This irregularity is often produced in hearts which have no other signs of disease, and may be transient and without pathological significance ; on the other hand, it is commonly a sign of failing compensation.

At times one observes two beats followed by a pause (the *bigeminal pulse*), or three beats similarly grouped (the *trigeminal pulse*). This is probably the result of extra systoles occurring at regular intervals.

4. *The Volume of the Pulse.*—The pulsations may be of small amplitude—*small pulse* (*pulsus parvus*)—as in high tension, which interferes with the expansile movements of the vessel wall. In low tension the volume may also be small, as in heart failure from any cause. With a hypertrophied left ventricle and efficient mitral valve there may still be a small pulse when an obstruction exists between the ventricle and the radial artery, as in the case of thoracic or brachial aneurism or stenosed aortic orifice.

The *large pulse* (*pulsus magnus*) is found in those conditions which favour the transmission of a large wave from the heart to the periphery—viz., a strongly acting heart, low arterial tension, elasticity of the arterial walls, and an ample supply of blood passing without obstruc-

tion to the periphery. Should the heart muscle weaken, the pulse will then become small and thready, in spite of the lowered tension. A markedly large pulse (*pulsus celer*) is found in aortic incompetence, as mentioned above.

When a strong and a weak beat alternate, the pulse is known as *pulsus alternans*. If the intervals between the beats be equal, this form of arrhythmia often indicates serious impairment of the contractile power of the heart muscle ; if the smaller beat be followed by a longer interval than that which precedes it, the rhythm is probably not *pulsus alternans*, but *pulsus bigeminus*, the result of extra systoles. Diminution or even disappearance of the pulse during the act of inspiration (*pulsus paradoxus*) may be observed in some cases of adherent pericardium. Occasionally this rhythm has been noticed in cases of pleurisy, pneumonia, obstruction of the air-passages, and valvular heart disease.

5. *The Duration of the Pulse.*—The pulse-wave may be felt as if quickly filled and as quickly emptied in the *pulsus celer* referred to above, while the slow pulse (*pulsus tardus*) consists of a wave of longer duration.

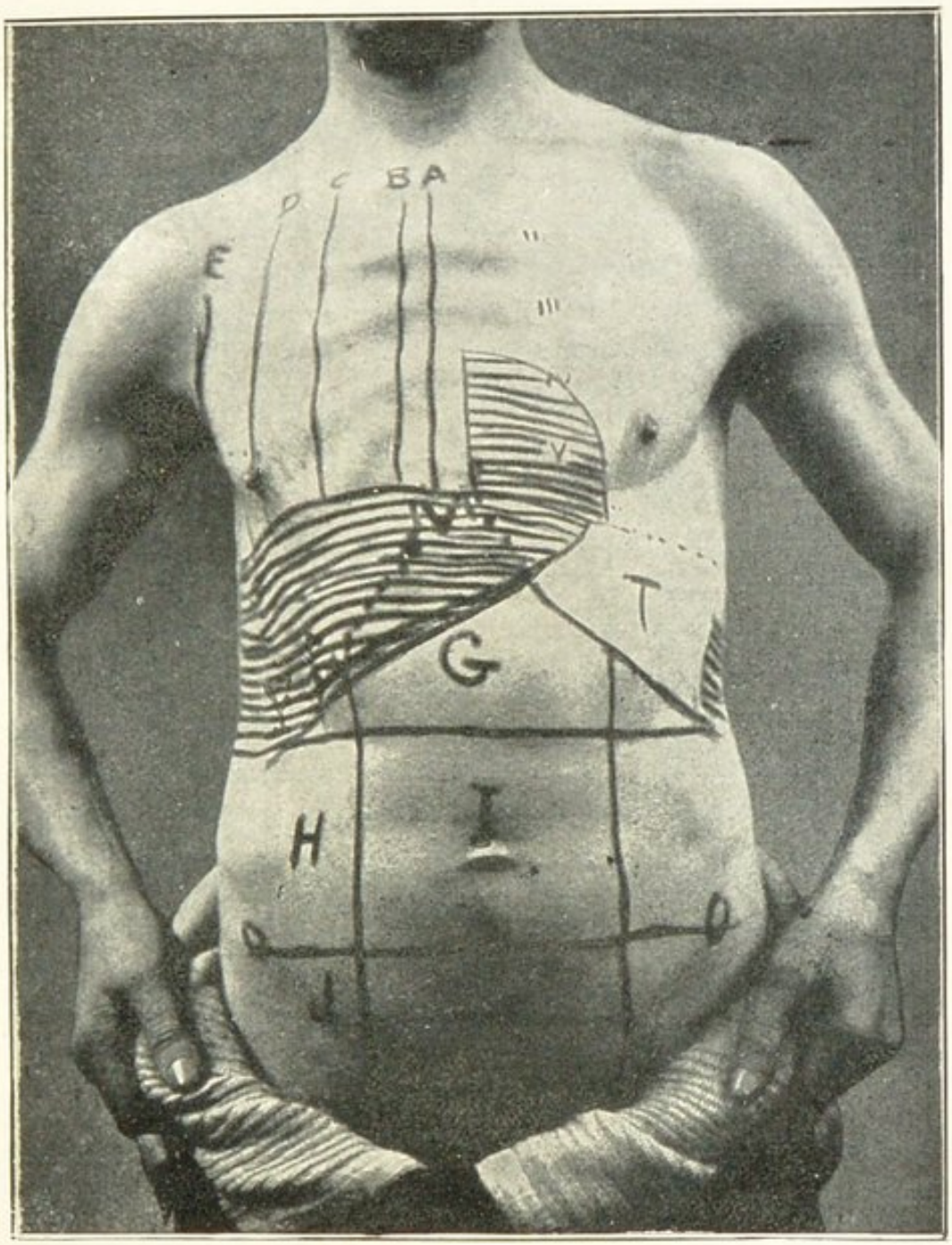
CHAPTER III

THE THORAX

Topography—Methods of examination—Shape of the chest.

Topography.—For facility in recording the result of examinations, the surface of the chest is mapped out into regions by means of natural landmarks and by the following artificial lines, all of which run vertically: (1) *Midsternal line,* the median line in front; (2) *side-sternal line,* over each border of the sternum; (3) *mammillary or nipple line,* a line through the nipple or, in the case of females, through the middle of the clavicle; (4) *parasternal line,* midway between (2) and (3); (5) *anterior axillary line,* through the spot where the anterior axillary fold joins the thorax, the arm being held out horizontally; (6) *posterior axillary line,* correspondingly through the posterior axillary fold; (7) *mid-axillary line,* midway between (5) and (6); (8) *scapular line,* through the inferior angle of the scapula; (9) *spinal line,* the median line behind.

The natural landmarks are the ribs, clavicles, nipples, spines of the scapulæ, and vertebral spines, of which the seventh cervical is usually easily recognized. The junction of the upper and middle portions of the sternum (*angle of Ludwig*) marks the level of the second costal cartilages.



TOPOGRAPHY OF THE THORAX AND ABDOMEN (FROM "DICTIONARY OF MEDICAL DIAGNOSIS").

A, midsternal 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.

To face page 36.

The thoracic regions mentioned above are twenty-six in number, and are the following :

In the middle line, in front (four regions) : lateral boundaries, the side-sternal lines ; *upper sternal*, above the angle of Ludwig ; *midsternal*, from the second to the fourth costal cartilage ; *lower sternal*, from the mid-sternal to the point of the xiphoid cartilage ; *epigastric*, filling the angle between the two hypochondriacs (relates chiefly to abdominal organs).

On each side, in front (ten regions) : lateral boundaries, the side-sternal and anterior axillary lines ; *supra-clavicular ; clavicular ; infraclavicular*, down to the level of the third costal cartilage ; *mammary*, down to the level of the sixth rib in the nipple line ; *hypochondriac*, down to the costal margin (chiefly abdominal).

At the sides (four regions) : between the anterior and posterior axillary lines—*axillary*, above the level of the sixth rib in the mid-axillary line ; *infra-axillary*, below the axillary.

Behind (eight regions) : between the posterior axillary line and the spinal line—*suprascapular ; scapular* (includes the supra- and infraspinous) ; *infrascapular ; interscapular*.

Examination of the Thorax.—The means to be employed are—inspection, palpation, mensuration, percussion, auscultation, and in some cases radiography. Pay attention first to the shape, size, and movements of the chest, using the first three of these methods. Observe the chest in a good light, looking at it from the front, back, sides, and from above and below. In palpating the chest, stand to the right of the patient, who is first to be in the recumbent position ; direct him to breathe deeply, to cough, to speak, while the hand is in contact with the chest. If possible, he is to be

examined also in the upright posture. In some cases it is desirable to measure the circumference of the chest, or to make an outlined tracing of its shape by means of the *cyrtometer*, an instrument composed of two strips of lead, which can be moulded to the shape of the chest, (usually at the level of the nipples), and the figure transferred to paper.

Abnormally Shaped Chests do not necessarily indicate disease ; they may result from past ill-health and faulty development, or may show a tendency to disease in the future.

Diseases in childhood causing a diminution of intrathoracic pressure give rise to characteristic deformities later in life. The chief affections of this nature are adenoids, enlarged tonsils, catarrh of the nasal, faucial, and bronchial passages, all of which cause difficulty of respiration in childhood, and so prolong the period of inspiration when intrathoracic pressure is at its lowest, and the thoracic walls tend to bend inwards at their least resisting parts. The results are: *Harrison's sulcus*, or the transversely grooved chest, a fairly horizontal depression just above the level of the liver ; or it may take the form of simple eversion of the costal margins. The *pigeon breast:* the front of the chest is narrow and keel-shaped. The *rickety chest:* a vertical depression on each side of the front of the chest, the junctions of the ribs with the costal cartilages usually lying at the base of the groove ; these joints are swollen, and feel like a string of beads (the *rickety rosary*). The *funnel chest* (*Trichterbrust*), a depression of the lower end of the sternum, which may be the result of obstructed respiration in childhood, of pressure, as sometimes occurs from the pressure of implements among shoemakers, or it may be congenital.

Developmental defects are seen in the *alar chest*, in which the ribs are too oblique, the shoulders in consequence sloping, the neck long, and the scapula projecting and wing-like ; in the *flat chest*, with the anterior portions of the ribs and costal cartilages flattened instead of convex ; and sometimes in the *funnel chest*.

Disease actually present may be the cause of thoracic deformities. The *emphysematous* or *barrel chest*, due to prolonged coughing and expiratory dyspnœa (usually chronic bronchitis or asthma) ; the chest is in the position of very full inspiration. Spinal curvature distorts the shape of the chest. The scoliotic, the kyphotic, the scolio-kyphotic chests, show the deformities due to lateral curvature and undue posterior convexity of the spinal column. Large pleural effusion or pneumothorax enlarge the affected side, obliterate the intercostal spaces, raise the shoulder, and cause the spine to curve with the convexity towards the effusion. Enlargement of abdominal organs may produce increase in size of the lower chest. Enlargement of the heart and pericardium may cause precordial bulging, especially among children. Aneurisms, mediastinal or other tumours, may produce a local swelling. Retraction of the lung occurs in chronic sclerotic and indurative processes (chronic phthisis, chronic pneumonia), in collapse of the lung, and after the absorption of a copious and long-continued pleuritic effusion. The affected side is retracted, the shoulder lowered, and the spine concave towards the side of the lesion. In phthisis the retraction is commonly less extreme than that just referred to, and usually occurs as a retraction of the supra- and infraclavicular regions of one or both lungs; this is most readily recognized by the undue prominence of the clavicles.

Bulging of intercostal spaces is sometimes seen in pleural effusion, in pneumothorax, and in asthma.

Any change in colour, any abnormal or enlarged blood-vessels, any obvious enlargement of lymphatic or other glands, any tumour or other deformity, must now be noted, as far as they relate to the condition of the thorax.

The further examination of the chest consists almost entirely of a study of the respiratory and circulatory systems, and these will as far as possible be taken separately.

CHAPTER IV

RESPIRATORY SYSTEM

Dyspnœa—Altered rhythm of respiration—Cough—Alterations in the voice—Vocal fremitus—Percussion—Auscultation—Breath sounds—Voice sounds—Adventitious sounds—Examination of the sputum.

A MORE detailed examination of the patient is now to be undertaken, with especial regard to the respiratory organs and to any derangement of their functions or structure that may be present. Further inquiry should be made from the patient as to any relevant subjective symptoms which he may have noticed, and he is to be examined in the first place by the means just described (inspection, palpation, mensuration), and later by percussion, auscultation, and, if necessary, by radiography.

Dyspnœa.—Any abnormality in the act of breathing is to be noted. Many diseased conditions in addition to those of the respiratory organs cause a difficulty in breathing (*dyspnœa*), owing in most cases to defective oxidation of the blood. In other cases irregularity or disturbance of rhythm occurs without obvious dyspnœa. It will be convenient to consider here abnormal breathing arising from any cause, pulmonary or otherwise. The dyspnœic conditions will be first described.

Subjective dyspnœa is the sensation of "shortness of breath," by which the individual is conscious of the

want of oxygen ; it is almost always associated with obvious signs of difficult breathing (objective dyspnœa). In hysterical and neurotic persons the subjective sensation is usually out of proportion to the objective signs.

Objective dyspnœa is of much more value in diagnosis. The chief features to be recognized are increased or decreased frequency of respiration, increased or decreased force, altered rhythm of respiration, exaggerated movements of respiratory muscles, active movements of the alæ nasi, stridor, cyanosis.

Normally the respirations in adults occur 16 to 20 times per minute ; in children of five years, 25 per minute ; and at birth, 45 per minute.

Increased frequency of respiration is the commonest form in which dyspnœa appears, and occurs in all those cases in which the amount of lung tissue available for aeration is diminished—*e.g.*, phthisis, pneumonia, capillary bronchitis, pleural effusion, pulmonary embolism and infarction, œdema and passive congestion of the lungs, pneumothorax, mediastinal and other tumours causing pressure on the lungs. Difficulty in the act of respiration, owing to pain in the chest, injuries to the chest walls, or obstruction of the upper air passages, cause increased frequency of respiration. The red blood cells may be insufficient in quantity or in hæmoglobin, as in chlorosis, and other forms of anæmia, and hæmorrhage. The blood may be imperfectly aerated owing to disease of the heart or lungs ; venous blood reaching the respiratory centre acts as a powerful stimulant to the centre. Blood which contains bacterial or other toxins, which has been imperfectly purified by the excretory organs, or which is of an unduly high temperature, probably stimulates excessively the respir-

atory centre—*e.g.*, pneumonia, Bright's disease, diabetes, and fevers.

The readiness with which dyspnœa of this description is produced in those whose breathing when at rest is natural, forms a fair index of the gravity of the lesion, or, in the case of heart affections, of the degree of compensation which has been established.

Decreased frequency of respiration is less commonly seen. It may occur in emphysema and asthma, in which cases it is chiefly the expiratory part that is prolonged. A slow, sighing type of breathing may be seen in the dyspnœa of shock, syncope, hæmorrhage, and in hysteria. Moribund patients have a slow, irregular, intermittent respiration ; poisoning by opium, chloral, chloroform, and aconite causes slow respiration.

Increased force of breathing is usually seen with slow breathing, but in some cases rapid breathing may be energetic and deep—*e.g.*, the dyspnœa of anæmia, diabetes, and heart disease. The slow breathing of shock, hysteria, and intracranial disease, may be unduly forcible.

Altered Rhythm of Respiration.—Normally the act of expiration lasts a little longer than inspiration, in the ratio of about 6 to 5, and a slight pause ensues after expiration, unless the breathing be hurried. The inspiratory portion especially of the act of respiration may be impeded and prolonged in conditions which obstruct the free entrance of air to the lungs—laryngeal or tracheal obstruction. Stridor, recession of the supraclavicular fossæ and of the epigastrium and lower intercostal spaces, and prolonged inspiration, are the chief features of this disturbance of respiration. On the other hand, expiration may be prolonged and difficult, with bulging of the supraclavicular fossæ and epi-

gastrium, and excessive action of the abdominal muscles. This arises from want of elasticity of the lungs and thoracic walls, and is seen in chronic bronchitis, emphysema, and asthma.

Irregularity in the rhythm of breathing as regards force and the length of intervals between separate breaths occurs in hysteria, heart failure, cerebral hæmorrhage, brain tumours, meningitis, etc. A more methodical irregularity is seen in *Cheyne-Stokes respiration:* alternating phases of weak and forcible breathing, the movements of respiration being entirely stopped during a short period, then gradually gaining strength till they become exaggerated, and then by degrees weakening till they cease again. It may be observed during sleep or unconsciousness, or the patient may lapse into stupor during the apnœic or quiet interval. While it is usually an ominous symptom, in many cases the condition passes away as the patient's health improves. It is found in serious intracranial disease, heart affections, kidney disease, opium-poisoning, and other toxic states threatening life. A somewhat similar type of breathing, in which, at intervals of variable duration, the breathing may stop for perhaps half a minute, is termed *Biot's respiration;* it may occur in cerebral meningitis and other grave disorders.

Stertorous dyspnœa may be mentioned; it is caused by noisy vibrations of the soft palate while breathing through the mouth, and is heard during unconsciousness, either of sleep or of coma—*e.g.*, cerebral apoplexy, uræmic, diabetic, alcoholic, narcotic, and ante-mortem coma.

Decreased activity of respiration is rarely a form of dyspnœa. The extent of the movements of respiration may be limited locally or generally by painful conditions,

by mechanical obstruction, by nerve lesions, by developmental defects, etc. Thus we find, on careful observation of the chest that the movements as a whole, or possibly of one side only, may be defective in cases of pleurisy (with or without effusion), emphysema, pericarditis, peritonitis, fracture of the ribs, pleurodynia, paralytic conditions (spinal disease, peripheral neuritis), and ankylosis of the costal articulations in arthritis deformans. In debility and collapse the breathing is shallow. Unilateral or localized immobility is seen in affections which prevent the proper expansion of the lung, as phthisis, pleural adhesions and effusions, pneumothorax, collapse of the lung, pneumonia, obstruction of a bronchus, etc.

Cough.—The sudden explosive expiration is a reflex act arising from some irritation applied to sensory nerve endings, usually, but not invariably, in the mucous membrane of the respiratory tract, some portion of which —*e.g.*, interarytenoid mucous membrane and that at the bifurcation of the trachea—are more sensitive than others. The stimulus may, however, arise from other sensory regions, which have apparently little connection with the respiratory system, as the external auditory meatus, the stomach, the ovaries. In coughs arising from these more distant regions it is possible that the general nervous system is unusually sensitive. This so-called nervous cough is a short, dry, oft-repeated effort, which, apparently, becomes a habit. A similar, but more pronounced and insistent, bark is the hysterical cough, which, though largely of emotional origin, has usually some peripheral irritation—possibly a very trivial one—for its cause.

The 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.

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.

Alterations in the Voice are caused by disease of the larynx or of its nervous supply, by disorders of the respiratory passages and mouth, and by general disturbances of health. In examination the laryngoscope is to be used.

Hoarseness and Loss of Voice (*Aphonia*) arise from any interference with the proper vibrations of the vocal cords, the commonest cause being laryngeal catarrh. It may occur in diphtheria, syphilis, tuberculosis, tumours of the larynx, cicatricial contractions, foreign bodies, œdema of the glottis, inflammatory or malignant disease of the œsophagus or pharynx, paralysis of the vocal cords from disease or injury of the laryngeal nerves (superior and inferior, branches of the vagus) or of their central connections. 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.

Nasal Voice.—Obstructions in the nasal passages interfere with the proper resonance of the voice, or give a nasal quality to the word spoken—the so-called closed nasal voice. On the other hand, inability to

shut off the pharyngeal from the nasal cavity, as occurs in paralysis of the soft palate, causes the open nasal voice—that is, talking through the nose.

Debility from any cause is often indicated by *weakness of the voice.* Hysteria has been already mentioned as a cause of aphonia.

Vocal Fremitus.—A trembling movement of the chest surface produced by the act of speaking. With the hand laid flat upon the chest, the patient is instructed to say the words " nine " or " ninety-nine," which, by imparting a nasal quality to the sound, favour the production of vibrations. Normally these vibrations are conveyed from the larynx through the air channels in the lungs (bronchi) and the spongy texture of these organs, thence to the chest wall. The distinctness with which these movements are felt is favoured by—(1) a low-pitched, loud note ; (2) the presence of good conducting material between the larynx and the chest wall. Columns of air, as found in the larger bronchi, are excellent conductors of sound vibrations. The spongy mass of air cells in the lung is a poor conductor, but its conducting quality is improved either by relaxation of the lung tissue or by its replacement by a more solid mass, such as is found in consolidation of the lung from inflammation, particularly if the consolidated mass is traversed by open air tubes.

On the other hand, a high-pitched and weak voice, or the presence of non-conducting material between the source of the vibrations (larynx) and the chest wall, such as pleural effusion, fatty chest wall, œdema, interfere with vocal fremitus.

Litten's Sign.—Place the patient on his back with his feet toward the light, other side-lights being removed ; a linear shadow at right angles to the mid-

axillary line is then seen moving downward with inspiration, and, less distinctly, upward with expiration. The distance traversed by the shadow may be 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.

Percussion.—The sounds elicited by a stroke of the fingers or of a specially designed hammer (*plessor*) acquire a musical quality or *resonance,* owing to the presence of enclosed air. The vibrations caused by the blow acquire a regularity of rhythm, owing to the resounding qualities of the air channels. The result is, as in many musical instruments, a musical note of resonance instead of a noise. The degree of resonance varies, and depends mainly upon the resounding qualities of the objects struck, collections of air forming the best resounding medium. Bone is a moderately good resounding body, but soft tissues have no resonant quality. Practically, resonance depends upon the presence of collections of air in the immediate vicinity of the spot struck. Percussion is practised almost exclusively by the so-called mediate method—that is, the hammer (usually the middle finger of the right hand) does not strike the part immediately, but an intermediate instrument called the "pleximeter." The latter may be composed of bone, vulcanite, cork, or other material; but most examiners prefer to use a finger of the left hand, which is placed, palmar surface downward, firmly, but not too forcibly, upon the portion of the body under examination. One or two fingers of the right hand

bent at a suitable angle form the hammer or plessor, sharp taps being delivered from the wrist with a staccato movement and with varying force. The use of the finger as a pleximeter has the additional advantage of conveying to the observer information as to the condition of *resistance* or *tone* of the part. In determining the boundaries of the organs by means of percussion, it is best to percuss first the more resonant regions, advancing the pleximeter finger in a line at right angles to the boundary which is to be determined. Careful comparison of similar regions on each side of the body, due consideration being given to the anatomical relations of the organs, is of the utmost importance.

Generally speaking, a forcible blow, a large collection of air under moderate pressure, and a smooth wall cavity, all favour a high degree of resonance or musical quality, which, being of a drum-like character, is spoken of as a *tympanitic note*, or *hyper-resonance*. This is the sound produced by percussing over the stomach. In the lung the air chambers are small and divided by innumerable membranous septa, which, being in a high state of tension, are imperfect resounding media ; the percussion note of the lung is therefore less resonant than that just mentioned, and is generally spoken of as a *subtympanitic note*.

If the stroke is delivered over a solid organ, such as the liver or heart, there is an absence of the resounding quality, and a dull note or noise results (*dulness*).

That portion of the chest wall which is in contact with the lung is spoken of as the *pulmonary region*. It extends from about 1½ inches above the clavicles to the level of the base of the lung on each side, which during quiet breathing is at the sixth rib in the nipple line, eighth in the mid-axillary line, tenth in the scapular

line. On the left side in front, the anterior edge of the left lung deviates from the middle line at the level of the fourth costal cartilage, curving out to the apex of the heart about 3½ inches from the middle line, leaving a triangular area, called the *area of superficial cardiac dulness* (A.S.C.D.), over which the percussion note is dull.

That portion of the thorax in contact with the stomach is called *Traube's semilunar space.* It is bounded above by the base of the left lung, the heart's apex, and the left lobe of the liver ; below and internally by the left costal margin ; and externally by the splenic dulness.

The sounds obtained by percussion may be altered by disease in various ways :

Increased Resonance, due to excessive activity and force of the resonant vibrations. This is found in— (1) Emphysema of the lung. The resonance in this condition is drum-like, giving a sound like that obtained by striking an empty cardboard box (*box note*). (2) Relaxation of the lung. The healthy lung is in a state of extreme tension. Any condition which reduces the capacity of the thorax permits the lung to relax, causing a more resonant note. Thus in cases of pleural effusion, above the level of the fluid, the percussion note has a somewhat high-pitched, clear quality—the so-called *skodaic resonance.* (3) Pulmonary cavities, if large, superficial, and recently emptied by expectoration, become hyper-resonant. Other modifications of the note produced by pulmonary cavities are referred to below. (4) Pneumothorax, a large collection of air in the pleural cavity, causes a loud and resonant note, unless the air should be under high pressure, as is often the case, owing to a valve-like communication between the pleural cavity and a bronchus. In this case the percussion note is only of moderate resonance.

Diminished Resonance.—1. A reduction in the quantity of air in the lung lessens the resonance of the stroke. This condition may be caused by infiltration of the lung with the products of inflammation ; hence pneumonia (commonest at the base) and phthisis (oftenest at the apex) cause diminished resonance. Passive congestion of the lung may give rise to dulness, and may be found in prolonged debilitating diseases or in heart disease. Other conditions, less frequently met with, producing this abnormal dulness are infarction, œdema, collapse, or cirrhosis of the lungs. Compression of the lung by pleural effusion or by tumours of the thorax, and the presence of the tumours themselves, cause a dulled percussion note.

2. A decrease in the actual quantity of lung tissue from disease, of which some instances have just been mentioned, is another cause of diminished resonance.

3. The interposition of airless material between the lung and the surface is a common cause of dulness—*e.g.*, thickened pleura and pleural effusion.

In cases of pleurisy with effusion it is noticed that the fluid does not strictly follow the lines of gravity. The fluid is found, even in patients who remain in the upright position, to rise 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 position of the fluid results mainly from the tendency of the liquid to follow the line of least resistance, which, in this case, means the replacing or compression of the most voluminous part of the lung by the fluid at the back and sides of the lower part of the lung. The curved line, representing the upper level of the fluid, is known as *Ellis's* or *Garland's line.* This line can best be demonstrated when the chest is moderately full of fluid.

In hydrothorax, where the fluid is not the result of pleuritis, it obeys more closely the laws of gravitation ; hence we may partly ascribe Ellis's line to pleuritic adhesions.

The dulness due to an effusion of blood in the pleural cavity (hæmothorax) resembles that of hydrothorax. It does not usually coagulate for several days, and unless fixed by adhesions it is quite movable.

An unusually thick chest wall (muscle, fat, œdema) deadens the resonant note. Alterations in the percussion note due to affections or displacements of the heart are referred to in Chapter V.

Changes in Quality or Tone.—The presence of a smooth-walled, fairly large cavity—*e.g.*, pneumothorax or a pulmonary cavity—gives a reverberating quality to the percussion note, known as *amphoric* or *metallic resonance.*

On percussing over a pulmonary cavity connected with the bronchus, the resulting tympanitic or amphoric note is raised in pitch if the patient open his mouth, and the pitch is lowered when the mouth is closed (*Wintrich's sign*). The same phenomena may be observed at times in percussing the apex of the lung in consolidation of that region (*Williamson's tracheal resonance*).

Bell Sound.—If the chest be percussed, using a coin as pleximeter and another as plessor, a sound is normally heard by the stethoscope of a metallic character. In conditions where a large collection of air is found in the chest, and particularly in pneumothorax, the sound acquires a clear, bell-like quality.

Cracked-Pot Sound.—A percussion sound which is usually an indication of a cavity in the lung. The percussion stroke, driving the air from the cavity into a bronchus, causes a squeaking or chinking sound, which,

once heard, can easily be recognized. A similar quality of percussion sound can be produced by percussing the chest of a healthy infant, particularly during forcible expiration or crying. In this case it is the air escaping at high pressure from the glottis which causes the sound. Its resemblance to the note produced by striking a cracked vessel or bell is the origin of the name.

Changes in the *extent* of the resonant area of the thorax as compared with the healthy chest must be observed. The pulmonary region may be increased in extent, as is seen in emphysema and pneumothorax; in these conditions the area of cardiac dulness may be diminished or abolished, and the liver and spleen dulness may be encroached on by the resonant areas. A diminution of the extent of resonance is another mode of expressing the occurrence of dulness where resonance ought to be found. The conditions which give rise to it are those enumerated above in discussing diminished resonance.

Auscultation.—It is possible to obtain most of the evidence required by the unaided ear, but for various reasons the use of an instrument (the stethoscope) is desirable. Many clinicians still prefer the single stethoscope. There can, however, be no doubt that the binaural instrument offers advantages in almost every case.

Care must be taken, when placing the bell of the stethoscope upon the body, that it press evenly and firmly, but lightly, upon the surface. Movement 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 skin with vaseline or other lubricant.

There are three groups of audible symptoms resulting

from respiration which have to be noted: (1) The sounds produced by the act of respiration in diseased conditions; (2) the voice sounds as modified by disease; (3) the various new or adventitious sounds produced in connection with disease of the lungs.

1. **Breath Sounds.**—The normal sound produced by breathing when one listens over a region of the lung distant from the larger air channels may be easily recognized as a sighing, whispering rustle (*vesicular breathing*), coinciding almost entirely with the act of inspiration. During expiration this sound is only heard in the earliest portion, and usually lasts about one-third or less of the time occupied by the inspiratory breath sound; the short expiratory portion is softer and somewhat lower pitched than the inspiratory. Often a faint, soft sound persists during the whole of expiration.

When one places the stethoscope nearer the trachea and larger bronchi—*e.g.*, near the sternum, in the upper part of the chest—and behind, close to the upper three or four dorsal vertebræ, the sound has a harsh to-and-fro quality—the so-called tracheal or bronchial breathing. The farther from the larynx one places the stethoscope, the less distinct is this sound, as a larger quantity of imperfectly conducting lung tissue is interposed, until, on listening over the sides or bases of the lung, one hears the characteristic vesicular breath sounds.

Increased Breath Sounds.—The sounds may be louder than normal, either owing to the increased production of the sound or to an improved conduction of the vibrations. Excessive action such as occurs when one lung sustains most of the respiration, owing to disease of the other lung, causes increased breath sounds—the so-called *puerile breathing*. The sounds are more in-

tense in forced breathing, in dyspnœa, in narrowing of the rima glottidis, in external or internal obstruction of the trachea or larger bronchi.

Improved conduction increases the loudness of breath sounds when a portion of the spongy lung tissue is replaced by a more homogeneous texture, as occurs in pneumonic or tubercular infiltrations, in excavation, in tumours, in compression, and, to a less degree, in relaxation of the lung. Unduly thin walls similarly transmit intensified breath sounds.

Broncho-Vesicular Breathing.—Increased breath sound generally takes the form of bronchial rather than louder vesicular breathing. When the increased intensity is only moderate, it may consist in inspiratory vesicular breathing, with either prolonged expiration or an actual bronchial quality of the expiratory portion. This is known as "transitional," "indeterminate," or "mixed" breathing. Prolongation of the expiratory portion may be the only evidence of increased intensity of breathing. At the right apex breathing is normally harsher than at the left, owing to the anatomical relations of the bronchi. Bronchial breathing heard at the lower part of the chest, and especially at the sides and back, may indicate pleural effusion, as the fluid causes relaxation of the lung. 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 open bronchi is probable. If the breath sounds be not only bronchial, but also cavernous (see below), a cavity may be diagnosed if the evidence obtained by percussion corroborates.

Decreased Breath Sounds result from either deficient production or imperfect conduction. The former occurs in debility, pneumothorax, emphysema, a flattened or

badly-developed chest, pleurisy, peritonitis, intercostal rheumatism, neuralgia, or fractured ribs. Defective conduction is found in conditions where the air passages are completely blocked, as in *massive pneumonia*, or where an inferior conducting material lies between the bronchi and the surface of the thorax, such as pleural effusion, thickened pleura, tumour, and thick chest walls.

A variety of abnormal qualities of the breathing, independent of the degree of loudness, have to be noted :

Tubular Breathing, a clear, high-pitched quality of breath sound, caused partly by the good conducting qualities of consolidated lung, and partly produced in the affected tissue by the passage of air over the open mouths of bronchi.

Cavernous Breathing, a curious reverberating quality of breathing heard over a large air cavity. A somewhat similar sound, resembling that produced by blowing across the opening of a narrow-mouthed vase or wide-mouthed bottle, is termed *amphoric breathing*.

Cog-Wheel Breathing, an intermittent, vesicular breath sound, heard chiefly in inspiration. If localized, it may be the result of obstruction in the bronchioles, and is found in bronchial catarrh and in late phthisis.

Metamorphosed Breath Sounds.—A change in the breath sounds may be observed at times ; inspiration may begin as a harsh or bronchial murmur, becoming softer as it proceeds, or it may acquire a cavernous character. This change is due to the partial distension during inspiration of a cavity in the lung. Other changes in the breath sound may be observed; thus Lænnec's *veiled puff* (*souffle voilé*) is a sudden change in the intensity or quality of the sound, owing to the temporary removal of a plug or curtain of mucus from a partially blocked bronchus.

Stridor, a noisy breath sound, caused by an obstruction in the bronchi, trachea, or larynx, and most frequently heard during inspiration. The respiratory difficulty is obvious, as seen by the exaggerated movements of respiration (see Dyspnœa, above).

2. **Voice Sounds.**—On listening over the chest whilst the patient speaks, a droning, buzzing quality of voice is heard, in which the articulation of words and syllables can hardly be distinguished. This is known as *Vocal Resonance*, and over those regions of the chest in which loud or bronchial breathing is normally heard the voice sound is loud, and gives one the impression of being generated at the surface practically under the stethoscope.

Increased Vocal Resonance usually indicates consolidation of the lung, and is termed *bronchophony*.

Decreased Vocal Resonance is caused by diminished conductivity of the tissues of the chest—*e.g.*, pleural effusion, thickened pleura, unusually thick chest walls, an obstructed bronchus, and emphysema.

Pectoriloquy, an unusually distinct and articulate voice sound, and *whispering pectoriloquy*, a similar distinctness of whispered speech, observed in conditions in which the conduction is exceptionally good. A pulmonary cavity communicating freely with a bronchus, and surrounded by an area of consolidation, is likely to produce this sign.

Ægophony.—A nasal or whining quality of voice, often heard over the situation of pleural effusion, especially at its upper limits. Occasionally it is heard over pulmonary consolidation.

3. **Adventitious Sounds.**—On listening over the respiratory organs, certain sounds may be noticed which cannot be described as mere modifications of the voice

or breath sounds. They may be classified as follows: (1) Rhonchi; (2) râles; (3) friction sound; (4) bell sound; (5) metallic tinkling; (6) succussion sound.

(1) *Rhonchi* are musical sounds heard during the act of breathing. They result from localized narrowing of the calibre of the bronchi by the deposition in the tubes of mucus, by thickening of their lining membrane, or by spasm of their muscular coat. Thus, sounds produced in the larger bronchi are low-pitched, and are termed *sonorous rhonchi;* whilst those originating in the smaller bronchi have a higher pitch, and are termed *sibilant rhonchi.* When heard over large areas of the chest, they are a sign of bronchitis, asthma, or more rarely of phthisis. In asthma the sounds are mostly sibilant, chiefly heard in the prolonged expiratory act. When localized, and particularly at the apex of a lobe, rhonchi suggest early phthisis.

(2) *Râles.*—Crackling, rattling, or bubbling sounds may be heard. The term "râle" (French, "rattling noise") may be used to include many different varieties of these sounds. *Crepitant râles,* fine crackling sounds, heard chiefly during inspiration. They may be imitated by rolling a small lock of hair between the fingers near the ear, or by gently tearing paper. They are heard in the earliest stages of croupous pneumonia, before consolidation has been established (*crepitatio indux*). A similar but coarser râle may be heard during resolution of pneumonia (*crepitatio redux*). Crepitant râles are also heard in catarrhal pneumonia, in hæmorrhagic infarction, and in œdema of the lung. This râle is by some observers regarded as an ill-defined pleural friction sound. *Subcrepitant râles*—rather coarser than the foregoing. They indicate a more copious or fluid secretion in the small bronchi. They are found in

bronchitis, inflammatory consolidations of the lungs, pulmonary œdema, hæmorrhage, or hypostatic congestion. *Mucous râles*—a larger and coarser class of râle, originating in the larger bronchi or in pulmonary cavities. They may be observed in bronchitis, bronchiectasis, and phthisis. When very coarse, they are termed *gurgling râles. Consonating râles*—a clear, crackling, bright, resonating character is given to the râles by the presence of solidified lung, through which the sounds are well conducted to the stethoscope. *Metallic râles*—an extreme degree of this consonating quality, often heard when consolidation surrounds large open bronchi or cavities. *Cavernous râles*—in which the reverberating quality is still more marked. The amount of reverberation will depend on the size of the cavity and the condition of its walls.

In many cases râles are only produced when the patient draws a deep breath, or after he coughs. 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.

(3) *Friction Sound.*—If the pleural or pericardial membranes have become roughened or dried with disease, their friction during respiration (chiefly inspiratory) may be enough to set up audible vibrations. The sounds vary from a faint brushing to a harsh, grating, scraping, or creaking noise. The fainter varieties resemble, and are by some regarded as, crepitant râles. A fairly accurate idea of the commoner type of friction sound is gained by completely covering the ear with the palm of the hand, and gently rubbing the back of that hand with the finger of the other hand.

A friction sound restricted to the pulmonary region

indicates pleurisy (including that accompanying pneumonia or phthisis) and tumour. Precordial friction usually denotes pericarditis, but may be caused by pleurisy affecting the 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 *pleuropericardial friction.*

Friction at the apex of a lobe often indicates phthisis. The commoner situations are the infra-axillary, mammary, and infrascapular regions, where it may denote simple or tubercular pleurisy or pneumonia. Friction sounds disappear when the pleural surfaces are separated by effusion. Reappearance of the rub, especially when it is found in the upper portions of the area of dulness, implies the removal of fluid at that region. Adhesion of the surfaces or resolution of the pleurisy also cause the rub to disappear.

(4) *Bell Sound.* See above, under *Percussion.*

(5) *Metallic Tinkling,* a faint but clear musical note, believed to be due 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 mucous râles (*amphoric râles*).

(6) *Succussion Sounds.*—When air and fluid are present in the pleural cavity, they give rise to a splashing sound on shaking the patient.

There are a few adventitious sounds to be heard over the pulmonary region which are unconnected with respiratory affections—*e.g.*, creaking sounds caused by movements of the scapula upon the thorax or by grating in the shoulder-joint; the muscular sound produced by contraction of the chest muscles may become evident; noises arising from accidental friction of the stethoscope

or skin may simulate friction sounds. Care in examination, and observation of the effect upon these sounds of respiration, will in most cases obviate error.

Examination of the Sputum.—A thorough investigation of the expectoration has to be made, both macro- and microscopically. The colour and character are to be noted, whether purulent, mucous, serous, or bloody. Microscopic examination discloses the constituents of the sputum, both in its cellular and non-cellular elements —viz., red and white blood cells, epithelial cells, elastic fibres, crystals, Curschmann's spirals, fragments of lung tissue, actinomycosis.

Micro-organisms are recognized by examination of a stained film. Other methods, such as inoculation of animals by carefully prepared specimen or cultivation of a specimen, are carried out in the laboratory when necessary. A smear is made by placing a small morsel of pus from the sputum between two cover-glasses, which are pressed together, then drawn apart ; this is repeated several times till the film is quite thin. If *tubercle bacilli* are suspected, the film is to be stained by the Ziehl-Neelsen method. Further examination for pneumococcus, staphylococcus, streptococcus, etc., is to be made by Gram's method of staining (see Appendix II.).

CHAPTER V

CIRCULATORY SYSTEM

Movements of the chest wall—Area of cardiac dulness—Displacements of the apex-beat—Thrill—The sounds of the heart—Adventitious sounds—Murmurs—Friction sounds—Examination of the arteries, veins, and capillaries.

THE means of examination described in discussing the respiratory system are also to be employed for this system.

Movements of the Chest Wall due to circulation are observed by inspection and palpation. In health they are confined to a spot not greater than an inch in diameter in the fifth left interspace just inside the nipple line and outside the parasternal. This is known as the *apex-beat*, and represents, with sufficient accuracy for clinical purposes, the situation of the apex of the heart in the thorax. *Diminution* of this movement is of small diagnostic importance, as its disappearance is not inconsistent with health. Weakening of its force may, however, be due to pericardial effusion, pulmonary emphysema, feeble ventricular contraction from dilatation, muscular degeneration of the heart, or from general debility. An unduly thick chest wall is a common cause (fat, œdema). *Increase* of the force and extent of the apex-beat, which has a slow, heaving character, and is displaced downwards and outwards, indicates hypertrophy of the left ventricle. Increased force and

extent of the impulse, with a knocking rather than a heaving character, but without displacement, is seen in cases of palpitation from exertion, from emotions, and as an effect of tea, alcohol, etc.

Changes in the position and extent of the precordial pulsation are found in many diseased conditions, and before considering them it will be convenient to examine the region of the heart by means of percussion.

Area of Cardiac Dulness.—The portion of the chest wall in contact with the pericardial sac can be determined, being absolutely dull on percussion. It is known as the area of superficial cardiac dulness, and may be defined by light percussion in the manner already described (Chapter IV.). Normally it extends from the level of the fourth costal cartilage above down to the liver dulness ; to the left for a distance of about $3\frac{1}{2}$ inches from the middle line to a point in the fifth interspace corresponding to the apex-beat. On the right, the area of dulness is bounded by the right side-sternal line. On forcible percussion, a comparative dulness is found, extending about a finger's breadth outside the above limits, forming the area of deep cardiac dulness. Alterations in the position and extent of this area and of the pulsations of the heart have to be noted.

Displacements of the Apex-Beat may be due to disease of the heart itself, of the lungs, pleuræ, mediastinum, abdominal organs, etc. If downward and to the left, it is usually caused by hypertrophy and dilatation of the heart, particularly of the left ventricle. Upwards and to the left : contractions of the left lung and pleura (fibroid phthisis, removed pleural exudations, with retraction of the lung) pull the heart across, while it may be pushed in the same direction by right pleural effusions, by mediastinal tumours, or by abdominal pressure. With

the exception of pericardial effusion, this displacement is rarely produced by cardiac affections. Directly outward to the left: by the causes last mentioned; by dilatation of the right ventricle. Directly downward: aneurism of the arch of the aorta, mediastinal tumours, emphysema of the lungs. To the right: left pleural effusion, tumour of left lung, contraction of right lung, dextrocardia. Changes of posture only slightly modify the position and force of the apex-beat.

Hypertrophy of the right ventricle causes pulsation in the lower region near the sternum. Retraction of the lung from phthisis may make evident the pulsation of the heart. In the left second intercostal space the pulsation of the pulmonary artery may be visible near the sternum; on the right side a corresponding pulsation may be due to the aorta. Pulsation in the episternal notch, if very forcible, may result from dilatation or aneurism of the aorta, but may be an unusually active innominate artery.

Pulsation behind the upper half of the sternum or immediately to either side is seen in aneurism of the aorta. Epigastric pulsation, if systolic, may be from a strongly-acting right ventricle or a displaced apex-beat; or the normal heart movement may be transmitted to the surface at this spot through the liver or tumour in the epigastrium. It is often due to the so-called irritable aorta, observed in nervous subjects. Rarely the epigastric pulsation is due to aneurism of the abdominal aorta. In this position a pulsating liver may be observed in cases of serious incompetency of the tricuspid orifice.

Instead of pulsation a *retraction* of the thorax at places may occur. This is seen in the lower intercostal spaces behind, as the result of pericardial ad-

hesions involving the diaphragm and lateral chest walls (*Broadbent's sign*). It may also be observed in the precordial region from the same cause, and is seen commonly in the epigastrium with the contraction of the right ventricle.

The *extent* of the area of cardiac dulness varies greatly in disease. It is diminished or abolished in emphysema ; it is increased in dilatation and hypertrophy of the heart, in pericardial effusion, and in retraction of the lung. In pericardial effusion the shape of the dull area differs from that due to heart enlargement. The chief increase is laterally, the right border of the dull area sloping outwards toward the right, thus forming a pyramidal-shaped area of dulness, with its base on the diaphragm. The area of cardiac dulness may also be apparently enlarged by the presence of mediastinal tumour or aneurism.

Pulsations in the neck may be more obvious than normal. Excessive arterial movements are seen in aortic incompetence and in a strongly-acting heart without disease. The venous pulse, when well marked, usually indicates dilatation of the right side of the heart.

Thrill.—Vibrations originating at one of the orifices may in some cases be felt as a quivering movement. It is most likely to be felt in obstruction of an orifice ; thus a thrill is most commonly felt at the apex of the heart, occurring in the period of auricular contraction—*i.e.*, presystolic—and caused by mitral stenosis. A systolic thrill at the base indicates in most cases contraction or roughening of the aortic orifice. Thrills due to regurgitation through the aortic or mitral valves are occasionally met with, but are of rarer occurrence. In the larger arteries thrills may sometimes be felt as

the result of constriction from some cause at a more proximal point.

The Heart Sounds.—The patient is to be examined with the stethoscope, both in the upright and in the recumbent position. Any departure from the quality of the normal sounds has to be noted.

The sounds are *intensified* by increased activity of the heart from bodily exertion, emotional excitement, Graves' disease, cardiac hypertrophy, Bright's disease, and sometimes from valvular heart disease. In other and less common instances increased loudness is due to improved conduction of sound, as may occur in pulmonary consolidation, in pneumothorax, in cavities of the lung, and in retraction of the lung from the precordial area.

The second sound is frequently accentuated ; it most commonly occurs in mitral disease, when the right ventricle is acting forcibly, and it is best heard over the second left costal cartilage. In Bright's disease, arterio-sclerosis, and aortic aneurism, the second sound is most accentuated near the second right costal cartilage, owing to increased tension in the aorta.

Increased loudness of the first sound is heard at the apex in cases of mitral stenosis ; here the accentuated first sound follows an immediately preceding murmur.

The normal heart sounds are *weakened* by all conditions which decrease the power of ventricular contraction, or which reduce tension in the aorta and pulmonary artery—*e.g.*, debility following acute disease, collapse, failure of the heart from degeneration of its muscle fibres. The sounds are also enfeebled by imperfect conduction of the sounds—*e.g.*, a thick chest wall, pericardial effusion, pulmonary emphysema. Incompetency of the aortic valves, owing to their imperfect

closure, causes weakness or loss of the second sound, which here may be replaced by a murmur. Weakening of the loud first sound of mitral stenosis or of the intensified second sound of mitral regurgitation or stenosis indicates failure of the left and right ventricle respectively. Tricuspid incompetence (usually from dilatation of the right ventricle) reduces tension in the pulmonary artery, with consequent weakening of the pulmonic second sound.

Irregularities in the *rhythm of the sounds* are to be noted. *Pendulum rhythm* 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. It is found in some cases of high arterial tension, and is probably due to prolongation of the closure-time of the heart, whereby the second sound is delayed. *Embryocardia,* or fœtal-heart rhythm, is similar but quicker, and is usually a bad sign. It occurs in heart exhaustion, as in fevers, in diphtheritic or other paralyses affecting the heart, and in the terminal stages of heart disease.

Prolongation of the first sound usually means hypertrophy of the ventricles; if *shortened*, dilatation.

Reduplication of the Heart Sounds.—Doubling of the second sound results from want of synchronism in the closure of the semilunar valves. Any condition which hastens the diastolic fall of pressure in the ventricle will hasten the second sound. In mitral stenosis the diastolic fall in tension in the left ventricle is hastened by the poor supply of blood from the left auricle, so that the aortic cusps close sooner than usual and before the pulmonary. In mitral regurgitation, on the contrary, the distended left auricle will rapidly fill the left ventricle, and so delay the fall in left intraventricular

tension ; hence the aortic cusps are the last to close. Another and more commonly accepted explanation of the doubled second sound is that the increased tension in the pulmonary circulation in mitral disease causes the pulmonary cusps to close before the aortic ; normally, however, the aortic tension is much higher than the pulmonary, and yet the two orifices are simultaneously closed.

The second sound may be apparently reduplicated by the presence of a murmur in the early part of the diastole, due to mitral stenosis or to aortic regurgitation transmitted to the apex.

Reduplication of the first sound is usually in reality an indistinct presystolic or late systolic murmur.

Gallop Rhythm.—There are three sounds with each beat, the third being usually accentuated. This is often observed when the heart muscle is failing, as in the feeble heart of broken-down compensation, of fatty degeneration, or of advanced Bright's disease. It may also occur with the excited cardiac action of emotions, of excessive exercise, or of exophthalmic goitre. The cause is uncertain, the first of the three elements being, perhaps, an abnormal sound or murmur, due to passive tension of the weakened ventricle or to auricular contraction.

Altered Quality of Sounds.—A metallic or ringing quality is heard when the heart is acting forcibly from any cause, or in the condition known as "arteriosclerosis," where the vessels and valve cusps have become hardened through disease. The proximity of resounding air cavities may add a metallic or reverberating quality to the normal heart sounds.

Slight impurity of the sounds may be due to some unusual degree of rigidity or tension of the cardiac

tissues, without lesion of the valves, but in some cases such impurities can be changed into a definite murmur by increasing the force of the heart's action with exercise.

Adventitious Sounds.—Abnormal sounds produced by the organs of circulation in the thorax are—(1) Endocardial and vascular murmurs ; (2) exocardial sounds.

1. **Endocardial Murmurs.**—The term "murmur" is usually restricted to sounds produced by a blood current set in vibration by some abnormal condition of the blood channels. Normally the blood current is noiseless, but in disease the so-called *fluid vein* is often produced, usually by the sudden alteration in the sectional area of the channel, audible vibrations being thus produced in the fluid. Similar vibrations occur as a result of the blood stream passing over roughened or irregular surfaces, even when the lumen is not altered in size. Some authors include in the term "murmur" friction sounds caused by the rubbing of roughened pericardial surfaces, but it seems better to restrict the use of the word to those noises produced by the blood current alone.

In some cases they do not depend upon anatomical changes in the heart, and are termed "functional," "hæmic," or "accidental." The following points are in favour of a murmur being accidental : A soft, blowing systolic murmur, best heard near the second left costal cartilage ; anæmia or fever may be present ; there may be a venous hum ; other evidence of cardiac affection may be wanting—*e.g.*, no history of rheumatism, no enlargement of the heart, no accentuation of the second sound, no changes in the pulse, nor signs of impaired circulation in the various organs and tissues of the body.

In the heart the lesion causing the murmur is almost invariably at one of the orifices, and consists in either a leaking (incompetence), a narrowing (stenosis), or a

roughening of the orifice in question. Incompetence is produced either by damage to the protecting cusps of the orifice (usually by endocarditis) or by dilatation of the chamber, whereby proper apposition of the cusps is prevented. Dilatation commonly occurs as a result of degenerative changes in the heart muscle. Stenosis is caused by cicatricial contraction or by adhesion of valve cusps following inflammation.

In studying a precordial murmur, one must determine (1) its point of maximum intensity ; (2) its time ; (3) the direction in which the murmur seems best propagated ; and (4) the character or quality of the murmur.

(1) *Point of Maximum Intensity.*—One can usually distinguish a spot on the front of the chest where a murmur is more distinctly heard than elsewhere. This point does not necessarily correspond to the 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 near the apex-beat (*mitral area*) nearly always originate at the mitral orifice. If at the lower end of the sternum or close to either side of its lower part (*tricuspid area*), the tricuspid is probably at fault. The immediate vicinity of the second right costal cartilage, known as the *aortic area,* is the situation where murmurs produced at the aortic orifice are often best heard, though diastolic murmurs of aortic origin are often heard better down the sternum and to the left (*secondary aortic area*). At the *pulmonary area,* as the neighbourhood of the second left costal cartilage is termed, murmurs generated at the pulmonary orifice and in the pulmonary artery are best heard.

If more than one point of maximum intensity can be demonstrated, more than one lesion is present.

(2) *Time of the Murmur.*—Ascertain at what precise period of the cardiac cycle the murmur occurs. The moment of ventricular contraction may be identified by placing the finger over the apex or on the carotid artery, or by recognizing the first sound of the heart by the stethoscope. Those murmurs which occur simultaneously with the ventricular contraction are called *systolic* murmurs. Those occurring immediately before the ventricle contracts are *presystolic* murmurs, and the term *diastolic* applies to all murmurs occurring after the second sound and before the first (including the presystolic murmur).

Systolic Murmurs originating in one of the four orifices of the ventricles—*i.e.*, excluding accidental murmurs—are obviously due to the passing of blood from the ventricles, which are at that moment in the act of contracting. If we have reason to believe that the lesion is at the mitral or tricuspid orifices, it must be of the nature of a leakage permitting the blood to leave the ventricle in a backward direction (*regurgitation*). If, on the other hand, the aortic or pulmonary orifice is at fault, it is caused by the blood passing onward in the natural direction, and is termed an *onward* or *obstructive* murmur.

Systolic murmurs heard best at the apex (mitral area) are therefore due to mitral incompetence. At the tricuspid area a similar murmur indicates leakage through the tricuspid orifice, almost invariably the result of dilatation of the right ventricle. At the base systolic murmurs are less definite in diagnostic significance. On the left side, at the pulmonary region, as already stated, they are commonly functional or accidental in character. At the aortic area over the right second costal cartilage systolic murmurs indicate (*a*) roughening or irregularity

of the inner surface of the aorta or the aortic orifice ; (*b*) dilatation of the aorta ; or (*c*) contraction (*stenosis*) of the aortic orifice.

A systolic murmur at the base may in rare instances be due to aortic aneurism, congenital heart defects, or the pressure of a tumour upon the large vessels in the mediastinum.

In some cases of mitral incompetence the valve only leaks towards the end of ventricular contraction, owing to some anomaly of the papillary muscles. Here the murmur occurs in the late portion of the diastole, and is known as a *late systolic* or *prediastolic* murmur.

Diastolic Murmurs—i.e., those occurring at any period of the cardiac cycle, except that of ventricular contraction—are produced by the entrance into the ventricles of blood through one or other of the orifices. When best heard at the aortic or secondary aortic, or pulmonary area, a murmur replacing or accompanying the second sound is due to reflux through a leaking aortic valve, and very rarely to regurgitation through the pulmonary orifice.

A murmur occurring a little later in the period of rest —the so-called *early diastolic murmur*—may be heard at the apex in some cases of mitral stenosis. It is heard at the moment when the relaxed ventricles are being filled by a stream from the auricles. Owing to constriction of the mitral orifice, the tension is raised in the left auricle, so that the current through the orifice may be brisk enough to produce a murmur. This occurs soon after the closure of the semilunar valves, and before the contraction of the auricles. A murmur heard immediately before the first sound (*presystolic murmur*) is a more common and characteristic result of mitral stenosis.

Another diastolic murmur best heard at the apex is that known as *Flint's* murmur. It is observed in cases where the aortic orifice is incompetent and the left ventricle is dilated. The exact mode of its production is uncertain, but it is probably due to vibrations set in motion by the reflux current through the aortic orifice striking the anterior cusp of the mitral valve. It is distinguished from the murmur due to mitral stenosis by the presence of signs of aortic disease, and by the absence of accentuation of the second sound.

A rare cause of presystolic murmur is tricuspid stenosis, which resembles that due to mitral stenosis, but is best heard in the tricuspid area.

(3) *Transmission of the Murmur.*—When the point of maximum intensity of a murmur is determined, the bell of the stethoscope should be moved along lines radiating from the spot where it is best heard. It is often found that the murmur can be heard farthest along one or other of these radiating lines. This line is termed the "line of transmission or direction" of the murmur, and is influenced not only by the conductivity of the tissues, but also by the direction of the blood current.

The apical systolic murmur of mitral incompetence is directed towards the left axilla, and may often be heard below the left scapula. A similar murmur resulting from tricuspid incompetence is better heard towards and to the right of the sternum. The diastolic murmur resulting from aortic regurgitation is transmitted best downward toward the ensiform cartilage.

The systolic murmur of aortic disease is usually transmitted upward into the vessels of the neck, and may be extensively transmitted in all directions.

The presystolic murmur of mitral stenosis is trans-

mitted, if at all, towards the sternum ; but is usually fairly localized.

(4) *Character of the Murmur.*—Loudness or faintness does not constitute any measure of the amount of injury present, as a serious lesion may only produce a feeble murmur, and *vice versa.* If the murmur be loud, it may be assumed that the blood current through the affected orifice is at any rate energetic. In cases of mitral stenosis, the greater the contraction the louder the murmur, as a rule ; but in mitral regurgitation an unmportant lesion may give rise to a much louder murmur than a serious leaking.

Generally speaking, obstructive or onward murmurs are harsh, while the murmurs of regurgitation are often soft and blowing. The harsh murmur of mitral stenosis has also the quality of ingravescence—that is, it grows louder as it progresses, and ends suddenly in the first sound.

Certain changes in the character of a murmur may be dependent upon circumstances unconnected with the circulatory organs. An increased loudness or resonating quality may be the result of consolidation of the lung, a pulmonary cavity, or other source of improved conduction of the murmur.

Vascular Murmurs.—See below.

2. **Exocardial Sounds.**—The following sounds, not produced in the heart or bloodvessels, but caused by the heart's action, are to be noted :

(1) *Pericardial Friction Sounds,* resulting from the rubbing together of roughened pericardial surfaces, usually heard over a very limited area of the chest, but if loud they are more extensively distributed. They have a to-and-fro rhythm, independent of the normal heart sounds, and are usually superficial—that is, the

sounds seem to be immediately below the bell of the stethoscope. They may be soft or harsh, according to the conditions of the surfaces involved.

(2) *Pleuro-Pericardial Friction*—that is, the rubbing of roughened surfaces external to the pericardial sac, synchronously with the heart-beat.

(3) *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.

(4) *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; from the presence of surgical emphysema in the mediastinum; from diaphragmatic pleurisy; or from subdiaphragmatic inflammation. A consideration of the general symptoms and condition will usually distinguish these different affections.

Examination of the Bloodvessels—*Arteries.*—In Chapter II. are some general considerations regarding the pulse. As a rule, the pulsations of the arteries cannot be seen, but in many cases they can be easily felt. When visible, the pulsations are usually more ample than those in health, the excess being due to (*a*) excited circulation, as in emotions, hysteria, exercise, exophthalmic goitre; (*b*) the pulsus celer, or bounding pulse, seen typically in aortic incompetence and, to a less degree, in fevers; (*c*) aneurismal dilatation of the vessels—a rare cause of visible pulsation.

In some cases the larger arteries should be auscul-

tated ; the two heart sounds can usually be heard in the large vessels. By pressure of the stethoscope over a large artery, a systolic bruit is normally elicited. The systolic and diastolic murmurs of aortic disease are easily heard in the carotid, and often in the subclavian. Sometimes they are heard in the femoral. Place the stethoscope gently over the femoral artery without pressure, and either one or two sounds resembling the normal heart sounds may be heard. A little pressure now gives the usual systolic arterial murmur ; press still more, and, with careful graduation of the pressure, one often succeeds in hearing not only a systolic, but also a diastolic murmur, which is not heard in health. This is known as *Duroziez's double murmur*.

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

Veins.—Varicose enlargement of the veins is usually a surgical affection. An unusually distended condition of the veins is, however, in some medical cases, of diagnostic importance. In the head, neck, and upper part of the thorax, enlarged veins frequently indicate some serious obstruction in the course of the superior vena cava, such as mediastinal tumour or aneurism, or severe disease of the right heart. Enlargement of the veins on the lower part of the thorax and over the abdomen are usually due to disease of the abdominal organs (see Chapter VII.).

Pulsation of the veins in the neck is sometimes found in health, but is usually the result of some disturbed action of the right side of the heart. The patient should be in the recumbent position, if possible, and an effort must be made to place accurately the venous movements in their proper position in the cardiac cycle. It is

usually impossible to do this satisfactorily by the unaided eye, and a graphic record of the movements is necessary if full advantage is to be taken of the information afforded by pulsating veins. This may be obtained by means of recording tambours, and Mackenzie's ink polygraph supplies all the data required in clinical examination.

The pulsations thus recorded may be a combination of venous and arterial pulses, but careful study of these tracings and of those obtained from the apex-beat, radial artery, etc., enables one to analyze in many cases the constituent factors producing the movements. By this means the condition of the heart muscle can be investigated, and much has been thus learned of the nature of disturbed rhythm.

Auscultation of the veins of the neck in cases of anæmia discloses a continuous murmur, often loud, snoring, or musical in quality, known as the *venous hum*, *bruit de diable*, or *Nun's murmur*.

Capillaries.—Excessive pulsation of the arteries transmitted to the capillaries (almost always an indication of aortic regurgitation) is shown by rhythmical paling and deepening in the colour of the skin. This is best seen on causing red patches to appear by rubbing the skin. Similar capillary pulsation may be seen in the finger-nails.

CHAPTER VI

BLOOD

Blood examination—The glandular system.

Blood Examination.—In certain cases an examination of the blood is to be made. The following is a brief description of the methods recommended as suitable for clinical purposes :

The chief points to be investigated are—(1) the number of red and white corpuscles per cubic millimetre of the blood ; (2) the percentage of hæmoglobin present as compared with normal blood ; (3) the character of the red and white cells and their proportionate count ; (4) *Widal's* reaction ; (5) *Wassermann's* reaction.

1. *Enumeration of the Red Cells.*—In calculating the percentage of red cells present, 5,000,000 per cubic millimetre may be taken as the normal count. Thoma's or Gowers' hæmocytometer gives reliable results. A known quantity of diluted blood is placed by means of a suitable pipette on a microscope slide divided by engraved lines into squares $\frac{1}{20}$ millimetre across. The number of red or white cells lying in a given number of squares is counted, and the quantity of blood which supplied the cells can then be calculated without difficulty.

2. The hæmoglobin value of the blood may be de-

termined by the hæmocytometer of Gowers, Sahli, or Haldane. The estimation of the percentage of hæmoglobin is made by comparing the colour of diluted blood with a standard colour.

Colour Index.—A convenient means of expressing the relation of the percentage of hæmoglobin to that of the red cells in the blood is by determining the "colour index." Thus, a normal specimen of blood has 100 per cent. of both hæmoglobin and red cells, and is expressed as $\frac{100}{100}=1$. Suppose the blood showed 60 per cent. hæmoglobin and 80 per cent. red cells, the colour index would be $\frac{60}{80}=0{\cdot}75$. Should the hæmoglobin be, say, 50 per cent. and the red cells 40 per cent., the colour index would be $\frac{50}{40}=1{\cdot}25$. In the diagnosis of different types of anæmia this relationship is of help. Thus, the colour index of pernicious anæmia is high, being usually 1 or more ; while in chlorosis it is invariably below unity, and often as low as 0·5. Other secondary anæmias nearly always show a colour index below 1.

3. A film is to be prepared and stained. A small drop of blood taken from the finger or lobe of the ear is spread thinly on the cover-glass by means of another glass or piece of cigarette-paper. A little practice enables one to spread such a film sufficiently thin and even. It is then treated with a staining fluid, of which Leishman's offers many advantages.

On examining with a microscope a normal blood film stained in this manner, the red cells are seen coloured a pinkish-red, their discs for the most part lying flat on the surface of the glass, and measuring 7 μ to 8 μ in diameter ($\mu=\frac{1}{25000}$ inch). The white cells are found

in greater variety, the following being an average proportionate count :

Polymorphonuclear leucocytes	..	70 per cent.
Small lymphocytes	..	23 ,,
Large mononuclear leucocytes	..	3 ,,
Transitional leucocytes ..	..	2 ,,
Eosinophile leucocytes ..	..	2

In diseased conditions changes are found in both red and white cells—viz. : *Poikilocytes :* red cells distorted in shape. *Megalo-* and *Microcytes :* cells unusually large and unusually small, but otherwise resembling the normal red cells. *Polychromatophilia :* red cells which stain a bluish-red or violet with Leishman's stain. *Basophile Granulation :* cells with granules which stain well with basic stains, and are therefore blue with all staining methods. *Nucleated Red Cells* occur in two forms—*normoblasts,* the size of a normal red cell, with a deeply staining homogeneous nucleus ; and *megaloblasts,* red cells of a larger size than the normal. Various nucleated red cells, transitional between normoblasts and megaloblasts, may be found. *Neutrophile Myelocytes :* large granular cells with a single divided nucleus. These constitute the majority of the cells of the bone-marrow, but do not normally find their way into the general circulation. *Eosinophile Myelocytes* have a somewhat smaller nucleus than the neutrophile myelocytes. They occur normally in the bone-marrow, but not in the general circulation. *Non-Granular Marrow Cells :* large cells with a homogeneous protoplasm and faintly staining nucleus—probably transitional forms of myelocytes.

In any severe anæmia poikilocytes, megalocytes, and microcytes, can usually be recognized, and are particularly characteristic of pernicious anæmia. Under

similar conditions polychromatophilia and basophile granulation are also likely to occur. Nucleated red cells are usually an indication of an attempt at regeneration of the blood. If they are of large size (megaloblasts), the case is probably one of pernicious anæmia.

Normally the white cells number from 8,000 to 10,000 per cubic millimetre. When this quantity is materially increased, the condition is termed *leucocytosis*. Should the polymorphonuclear cells constitute the chief element in the increased count, which is the most common type of leucocytosis, we may suspect acute inflammations, such as abscesses, etc. Acute infectious diseases, as erysipelas, pneumonia, diphtheria, scarlatina, and occasionally debilitating diseases, such as cancer, may show a similar leucocytosis. Also, a temporary leucocytosis, lasting several days, follows copious hæmorrhage. It is important to remember that this form of leucocytosis rarely occurs in tuberculosis, typhoid fever, or influenza, unless complications arise.

An increased number of white cells, of which the majority are lymphocytes, is termed *lymphocytosis*, and is best seen in lymphatic leukæmia. A relative increase of lymphocytes, the total number of white cells remaining about normal, is often found in anæmias and in enlargement of the lymphatic glands from various causes. It is also a feature of typhoid fever, smallpox, and pernicious anæmia.

Increase in the percentage of eosinophile leucocytes (*eosinophilia*) occurs in many diseases of the skin, nervous system, bones, and from intestinal parasites.

The presence of myelocytes in large numbers in the blood is an indication of spleno-medullary leukæmia.

In small numbers they occur in other forms of anæmia and in diphtheria.

4. *Widal's Reaction* is almost exclusively used as a test for typhoid fever, though it may be employed in the investigation of cholera, Malta fever, and other microbic diseases (see Appendix III.).

5. *Wassermann's Reaction* is practically never found in health, and in very few diseases except syphilis (see Appendix IV.).

A further examination of the blood must in some cases be made, with the object of obtaining information on the following points: the opsonic power; the saline concentration of the serum; the specific gravity; viscosity; freezing-point; alkalinity; coagulability; volume; amount of calcium salts; presence of micro-organisms; presence of parasites (*Plasmodium malariæ*, spirochæte of relapsing fever, varieties of filaria, the trypanosomata). The details of the procedures and their significance must be studied by practical work in the laboratory, and by reference to special treatises on the subject.*

The Glandular System.—In the neck enlargement of glands has to be noted. That of the cervical lymphatic glands may be the result of inflammations of the mouth and throat or tubercular disease. Visible enlargement of the thyroid gland may be part of the symptom-complex known as "exophthalmic goitre." A swelling just below or in front of the ear on either or both sides is produced by inflammation of the parotid gland, and usually indicates mumps. Enlargement of lymphatic glands in other regions may be due to tuberculosis or syphilis, but if the swellings occur over regions such as

* See the article on "Blood Examination," by Dr. T. Houston, in the author's "Dictionary of Medical Diagnosis."

the neck, armpits, groins, a more widespread affection may be suspected, such as Hodgkin's disease or lymphatic leukæmia. In the last-named affection the blood examination gives the necessary clue to the nature of the case. Affections of other abdominal glands will be referred to later.

CHAPTER VII

THE ABDOMEN

Topography—Aspect and surface markings—Palpation—Percussion—Auscultation—The stomach and its contents—Intestines—Liver—Spleen—Kidneys—Pelvic organs.

Topography.—In describing the condition of the abdomen, the clinical clerk makes use of natural landmarks and of imaginary lines which divide the abdomen into nine regions. The landmarks are—the ensiform cartilage ; the lower border of the ribs (the costal margin) ; the crests and anterior superior spines of the ilia ; the symphysis pubis ; the umbilicus ; the linea alba ; the lineæ semilunares forming the outer border of the recti muscles. Four lines may be drawn on the skin—two vertical, through the middle of Poupart's ligament ; two horizontal, one joining the lowest points of the tenth ribs, the other connecting the two anterior superior spines of the ilia. The regions thus formed are the *hypochondriac, epigastric, lumbar, umbilical, iliac,* and *hypogastric* (see figure, Chapter III.).

Aspect and Surface Markings.—*Colour changes* may be observed (see Chapter II.). Note *enlarged veins* due to obstruction to venous return. If arranged somewhat radially from the umbilicus as a centre (*caput medusæ*), the obstruction is in the outflow from the portal circulation, and may be the result of cirrhosis of the liver or

thrombosis of the portal vein. If the enlarged veins are chiefly at the sides of the abdomen, the cause is more likely to be obstruction of the inferior vena cava. The *umbilicus* is flattened and stretched in ascites, projecting in hernia and pregnancy, depressed in fatty abdominal walls and œdema. *Lineæ albicantes*, whitish streaks (reddish when recent), indicate considerable and prolonged stretching of skin (pregnancy, tumours, ascites, fat). *Eruptions* (Chapter II.), with the exception of that of typhoid fever, are not specially liable to appear on the abdomen.

Palpation.—The patient lies on his back in an unconstrained position, so that the abdominal muscles may be as much relaxed as possible. The whole hand should be employed, not the tips of the fingers alone, steady but carefully regulated pressure being maintained. Any abnormal mass is thus explored, and tender regions investigated. Pain thus elicited is characteristic of various inflammatory affections, while that due to spasmodic contractions of the bowel or other hollow viscus is usually relieved by pressure. Should there be free fluid in the abdominal cavity (*ascites*), sudden pressure of the abdominal walls with the tips of the fingers may reveal a solid mass floating in the fluid. Inflammatory affections of the abdomen are sometimes to be recognized by rigidity, which is an involuntary defensive procedure of the abdominal muscles. While palpating the flanks, it is usually of service to place one hand behind the patient and the other hand in front. By this means structures in the hypochondria (spleen, kidneys, liver) can be readily examined. In some cases it is advisable to change the posture of the patient and examine him in a sitting position, or lying on one side or on the face.

Shape of the Abdomen.—Retraction of the median line (scaphoid or boat-shaped abdomen) occurs in meningitis, cerebral tumour, colic. A flattening of the abdomen, with the flanks somewhat bulging, is found in cases of moderate ascites. A larger collection of the fluid causes the abdomen to be generally rounded and swollen.

The size of the abdomen is diminished in wasting from any cause (starvation, disease of the digestive organs, cancer). Enlargement of the abdomen may result from gaseous distension of the bowels and stomach. When extreme it is termed *meteorism*, as seen in peritonitis or obstruction of the bowels. Free gas in the peritoneal cavity occurs as the result of rupture of one of the gas-containing viscera. It is to be recognized by a consideration of the history of the case—the sudden onset of distension, pain, probably dyspnœa, and collapse. The absence of liver dulness on percussion, with dulness in the flanks, indicating fluid in the peritoneal cavity, strongly supports this diagnosis.

Certain abnormalities in the *movements* of the abdominal wall may be seen :

Movements of Respiration.—Excessive abdominal respiration occurs when thoracic respiration is impeded—*e.g.*, fractured ribs, pleurisy, pericarditis ; in weakness of the chest muscles (rare nervous lesions affecting the thoracic muscles) ; in pleural effusion or pneumothorax. Dyspnœa from any cause exaggerates both abdominal and thoracic respiratory movements. *Diminished* abdominal breathing is seen when the diaphragmatic movements are interfered with from any cause—*e.g.*, peritonitis, diaphragmatic pleurisy, copious pericardial effusion, weakness of the diaphragm (phrenic-nerve paralysis), abdominal distension from any of the causes named above.

Pulsating Movements in the abdomen are chiefly those of the epigastrium, already referred to in Chapter V. The liver may at times be found to pulsate ; this can usually be demonstrated by bimanual palpation, or, if the abdominal walls are lax, by insinuating the fingers below the anterior edge of the liver. A pulsating liver is an evidence of serious valvular heart disease.

Muscular Contractions of the viscera may be visible or palpable. A peristaltic movement of the stomach may be seen to traverse the upper part of the abdomen with a wavelike movement, passing from left to right. These movements are only perceptible when the abdominal wall is thin and the visceral movements unusually active. They almost always indicate obstruction of the pylorus (cicatricial contraction following ulceration or cancer). Vigorous intestinal movements also give reason to suspect obstruction in the gut, but the healthy intestinal wall may at times be seen to contract if the abdominal walls are thin.

Mobility of a tumour, on causing the patient to change his position or to draw a deep breath, gives useful information as to its relations. Enlarged glands, inflammatory exudation or abscess, tumour of the pancreas, aneurism of the abdominal aorta, are fixed.

Movements caused by the Presence of Fluids.—Large collections of fluid (ascites, ovarian cysts, hydronephrosis) give rise to a definite fluctuating wave. One hand being placed flat over the swelling at one side of the abdomen, a sharp tap or fillip is given at the opposite side, when a wave is felt to strike the palm of the hand placed upon the abdomen.

Movements produced by the fœtus in a pregnant uterus may be felt, and are easily recognized.

Percussion.—The note elicited by percussing the abdomen varies in resonance and pitch. Over the stomach and large intestine the note is more drumlike and deeper in pitch than over the small intestine. The whole surface of the abdomen is normally resonant, with the exception of the regions covering the liver and spleen. Hyper-resonance occurs in meteorism, where the gaseous distension of the viscera may be sufficient to displace upwards the liver, spleen, and heart, and, indeed, may cause diminution, or even disappearance, of the liver dulness. A higher degree of resonance is due to escape of gas into the peritoneal cavity, as a result of perforation of one of the air-containing viscera. The anterior area of liver dulness may then be replaced by a clear resonance.

Diminished Resonance indicates a diminished quantity of gases in the abdominal organs. Dulness in the flanks or iliac fossæ, with clear resonance in the higher regions suggests free fluid in the abdomen (*ascites*); change of posture of the patient, as by turning him on his side or raising him in bed, alters the relative position of the clear and dull areas of the abdomen. In cases of cyst, abscesses, or collections of ascitic fluid restrained by peritoneal adhesions, the change of posture does not effect to any extent a change in the situation of the dull area. Ovarian cysts, enlarged spleen, hydronephrosis, or other enlargement of the kidney, cause a dull area to appear in the situation of the swelling, which, by pressing aside the bowel, removes the resonant area to one side or to the flanks.

A dull note immediately above the symphysis pubis is caused by the urinary bladder if it contain 10 to 15 ounces in the case of men, or 15 to 20 ounces in women. Other non-resonant objects in the abdomen

are enlargements of solid organs, new growths, inflammatory exudation, abscess.

Auscultation.—The stethoscope is of but small assistance in examining the abdomen. In peritonitis friction sounds may occasionally be heard. A systolic murmur is to be sought where aneurism of the abdomen is suspected. The fœtal heart may be heard beating in pregnancy. One may also hear the entrance of fluid into the stomach from the œsophagus and of fæces into the cæcum (see below).

The Stomach.—The movements and shape of the stomach may at times be recognized by examination, to facilitate which it is usually desirable to inflate the stomach. A simple method is to administer separately draughts of sodium bicarbonate and tartaric acid in solution. The stomach, distended by the gas thus generated, forms a rounded swelling in the epigastrium, the lower border of which (the greater curvature) extends downwards to within an inch of the umbilicus. The lesser curvature lies under the liver, and close up to the ensiform cartilage. When the greater curvature lies below the level of the umbilicus, the lesser curvature remaining in its normal position, the stomach is dilated.

The stomach may be in a lower position than normal without being dilated (*gastroptosis*). This is best observed with the patient in the erect posture, as a projection in the umbilical and hypogastric regions and a recession in the epigastric region. A pyloric tumour may easily be mistaken for a gall-bladder. The latter is, however, more movable with respiration, while the pyloric tumour readily moves on handling. Should a tumour be situated at the cardiac end of the stomach, it is recognized with difficulty. Splashing sounds can be produced by pressing or striking sharply over the

stomach. Should they be heard at a time when digestion ought to be complete—that is, six hours after the last meal—it is an evidence of dilatation of the stomach.

Percussion.—The stomach note of percussion is sometimes definite enough to enable one to recognize the dimensions of the organ, especially when it has been inflated. After a considerable quantity of fluid has been drunk, a crescent-shaped dull area, corresponding to the greater curvature of the stomach, may be observed on percussion, the patient being in the upright posture.

A combination of auscultation with percussion (*auscultatory percussion*) is at times of service, and particularly in the examination of the stomach. Whilst listening with the stethoscope placed over the organ, the surface of the abdomen is tapped or rubbed. As soon as a spot which lies over the organ under examination is struck, the sound suddenly becomes intensified. It is thus possible in many cases to delineate the outline of the stomach or other organ.

Auscultation of the stomach is only of use in cases where the cardiac orifice is obstructed. The stethoscope is placed over the stomach, or an inch to the left of the spine at the level of the eighth dorsal vertebra. The patient is directed to swallow fluid, and in normal cases a gurgling sound is heard six or seven seconds later. When obstruction exists, the sound is either lost or delayed. Direct inspection of the interior of the stomach (*gastroscopy*) is rarely necessary. The same may be said for *gastrodiaphany*, which involves the introduction into the stomach of a small electric lamp.

Radiography has already proved of decided service in examining the condition of the stomach.

Vomiting.—Two classes of vomiting may be distinguished—(1) central vomiting, the result of direct

stimulation of the vomiting centre in the medulla—*e.g.*, intracranial lesions, emotions, uræmia, and other toxic states of the blood ; (2) reflex vomiting, due to nervous impulses reaching the vomiting centre from the periphery—*e.g.*, from the fauces, stomach, kidneys, testicles, ovaries, ears, etc.

The following points concerning vomiting have to be considered :

1. Vomiting readily occurs in *infants* and *young children.* It is frequently an early sign of acute diseases —*e.g.*, the exanthemata.

2. *The Relation of Vomiting to Meals.*—A stricture at the lower end of the œsophagus does not permit the food to rest in the dilated gullet for any length of time before being regurgitated. True vomiting may occur immediately after eating, when the stomach is in a state of irritability, as in some cases of acute gastritis, gastric ulcer, and gastric cancer. In chronic gastritis the interval between eating and vomiting is usually considerable (one or two hours), and here the vomited food is found in a still undigested condition. In all cases of central or reflex vomiting, except those arising from the stomach, vomiting occurs without relation to meals.

3. *The Time at which Vomiting Occurs.*—Morning vomiting occurs during pregnancy ; change of posture sets up nervous impulses from the uterus to the vomiting centre. In chronic alcoholism morning retching and vomiting occur, 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, and also in atonic dyspepsia, vomiting may only appear at longer intervals —perhaps every two or three days—and then copiously.

4. *The Relation of Pain to Vomiting.*—The pain of

gastric cancer, chronic gastritis, acute dyspepsia, and gastric ulcer, is usually relieved by vomiting. It does not relieve the pain from the passage of gall-stones and kidney-stones, from peritonitis and from appendicitis.

5. *The Relation of Nausea to Vomiting.*—In almost all cases vomiting is accompanied or preceded by nausea. Its absence is important, as this often indicates a lesion in the cranial cavity.

6. *Projectile Vomiting* is the sudden expulsion of the stomach contents without preliminary retching, often observed in intracranial lesions.

Examination of the Stomach Contents.—As a rule, the contents of the stomach must be withdrawn for examination by means of the stomach-tube. This procedure is contra-indicated in the following conditions: In extreme weakness, fevers, defective compensation of heart disease, arterio-sclerosis, aneurism of the aorta, pregnancy, and hæmorrhage from the stomach or lungs.

In order that the contents of the stomach may be known before their examination, it is usual to give a light meal after a fast (*test breakfast*). A couple of slices of bread, with one or two cupfuls of tea (Ewald's test meal), is suitable; but occasionally the examination is made after an ordinary mixed meal. The following points are to be investigated in examining the stomach contents:

1. The *motility* of the stomach. If an ordinary mixed meal, containing a fair proportion of proteids, has been selected as a test meal, there will be little or no solid matter found on washing out the normal stomach after seven hours. Should the movements be defective (atony or dilatation), solid masses may be found remaining from meals swallowed many hours before. Ewald's test meal should have left the stomach in two hours if the motility is normal.

2. The *quantity* and *quality* of the gastric juice may be estimated by determining the presence and the quantity of free hydrochloric acid in the fluid obtained by filtering the stomach contents. Place a few drops of *Günzburg's reagent* (phloroglucin 2, vanillin 1, absolute alcohol 30) with the same quantity of filtered gastric contents in a white porcelain capsule, and evaporate to dryness with gentle heat. A rose-red colour indicates free hydrochloric acid. The total acidity of the stomach contents and the quantity of free hydrochloric acid present have at times to be determined. For details, see Appendices V. and VI.

Free hydrochloric acid may be absent or diminished (*hypochlorhydria*) in cancer of the stomach, atonic or catarrhal dilatation of the stomach, and severe anæmia. Excess of free hydrochloric acid (*hyperchlorhydria*) is found in gastric ulcer, in acute and chronic gastritis, and sometimes in nervous dyspepsia. In the latter affection, however, the opposite condition of diminished acid may occur. An excessive quantity of the gastric fluids generally (*supersecretion*) is the rule in gastric neuroses, gastric catarrh, in the gastric crises of locomotor ataxia, and sometimes in gastric ulcer.

3. *Abnormal constituents* may be found in the stomach —*e.g.*, organic acids, bile, fæcal matter, or blood.

Organic acids may be demonstrated by *Uffelmann's test:* To a test-tubeful of a 1 per cent. solution of carbolic acid add 1 drop of liquor ferri perchloridi. Dilute till the solution becomes an amethyst-blue colour. The addition of organic acids changes the blue colour to yellow. Free lactic acid is often found in cases of cancer of the stomach. It is present (often in company with acetic or butyric acid) when fermentative changes are proceeding in the stomach.

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.

Fæcal vomiting is a further stage of the reverse peristalsis which causes bilious vomiting. It occurs in obstruction of the bowels and in peritonitis, and is said to have been seen in hysteria.

Blood may be vomited (*hæmatemesis*). It may be shed in the stomach or duodenum, or may come from the respiratory passages or œsophagus, to be afterwards ejected from the stomach. The following causes may be mentioned: (1) Ulcer of the stomach or duodenum. Here the blood is usually vomited at considerable intervals, and may be copious. (2) Cancer of the stomach. The blood is usually less copious, and only occurs at a late stage of the disease. (3) Gastric catarrh may infrequently cause hæmatemesis, but streaks of blood may be found after any prolonged attack of vomiting. (4) Congestion and varicosities of the capillaries and venules in the portal circuit often give rise to fairly free hæmorrhage into the stomach, causing hæmatemesis. This may occur in cirrhosis of the liver, the "nutmeg liver" (cardiac), and cancer of the liver. (5) Diseases of the spleen. (6) Corrosive poisons and other injuries to the stomach or œsophagus. (7) Aneurism of the aorta opening into these organs. (8) Blood states giving rise to hæmorrhages—*e.g.*, purpura, scurvy, septic inflammations. As a rule, there is no difficulty in detecting blood when present in the stomach contents. It may, however, be simulated by drugs, chiefly bismuth

or iron. It is best recognized by means of the hæmin test, described in Chapter VIII.

Food, mucus, and saliva, are found in all conditions causing vomiting, and are of no diagnostic value except as to the motility of the stomach, as mentioned above.

The Intestines. — Normally the bowel cannot be readily palpated, but masses of scybala or tumours of the gut may be felt as movable and often painless swellings. Inflammatory exudations in connection with the bowel are painful, fixed, and often dull on percussion ; but if covered by air-containing bowel, the percussion note may be resonant. Auscultation of the bowel is rarely of practical value, but the time of the arrival of food at the ileo-cæcal valve may often be determined by means of the stethoscope. In health a period of from four to five hours elapses from the meal-time (naturally, breakfast is the only suitable meal) till the first arrival of the stomach contents at the cæcal orifice, and in many cases the fluid fæces can be heard entering the large intestine as a series of squirting noises when the stethoscope is placed over the ileo-cæcal valve (Hertz). Delay in this period of the passage of the intestinal contents is much less common than in the large bowel.

An examination of the rectum has in many cases to be undertaken. In all cases of abnormal intestinal discharge—*e.g.*, blood, pus—and in many cases of chronic constipation, digital examination of the rectum should be made. By this procedure not only can the condition of the rectal contents, if any, be noted, but the adjoining structures can be investigated—*e.g.*, any tumour in the lower abdomen, the uterus and ovaries in females, and the prostate in males.

Inquiry as to the condition of the fæces has to be made, and, if necessary, they should be examined. By inspection of the evacuation one may observe 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 must be washed thoroughly through a sieve. 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.

Liver.—Normally the lower edge of the liver can just be felt in the right nipple line during inspiration, as it is depressed just below the costal margin. A line drawn from this point upwards and to the left to a point a little below the apex-beat corresponds to the lower border of the organ. Above, a horizontal line about the level of the sixth rib in the right nipple line, the eighth rib in the right mid-axillary line, and the tenth rib in the right scapular line, marks the level of absolute liver dulness, due to the contact of that organ with the thoracic wall. An area of relative dulness can be traced about an inch above this level.

Enlargements of the liver are usually observed in the direction of least resistance—that is, downwards. The enlarged liver is sometimes visible, and nearly always palpable.

Percussion of an enlarged liver is not always satisfactory, as the presence of adjacent air-containing organs gives resonance even over the liver, unless great care be

taken to percuss gently. Free fluid in the abdomen also renders percussion of the liver difficult.

The shape and character of the surface of the liver are to be determined by palpation. Note if the surface and edge of the liver are smooth, or, on the contrary, nodular, fissured, or irregular.

Diminution, or even disappearance, of the area of liver dulness is to be expected in intestinal distension. Complete disappearance of the dulness, however, is characteristic of the escape into the peritoneal cavity of gas from a ruptured viscus.

The gall-bladder, if enlarged, may be felt as a rounded or pear-shaped tumour, proceeding downwards from the right costal margin between the nipple and median lines.

Spleen.—The normal spleen remains covered by the lower left ribs, even during forcible inspiration. Its area of dulness in the left mid-axillary line extends from the ninth to the eleventh ribs, its long axis being parallel with the eleventh rib. When enlarged, the spleen may be recognized by its situation, by its mobility with respiration, and by the fact that it usually retains its shape and the characteristic notch on its anterior border, even when much enlarged.

Kidneys.—The chief source of information as to the condition of these organs is derived from an examination of the urine (see Chapter VIII.). By abdominal examination the lower end of the normal kidney can often be felt during deep inspiration, especially on the right side. Undue mobility of one or both kidneys (most commonly the right) is readily detected by palpation. In extreme enlargements of the organ (hydronephrosis, sarcoma, cystic disease, etc.) the tumour may be dull on percussion, except for a band of resonance where the

colon comes in front of the mass. In cases of moderate enlargement the kidney region is usually quite resonant, owing to overlying bowel.

Pelvic Organs.—Enlargement of the pelvic organs may be detected by abdominal examination. The pregnant uterus, after the third month of gestation, rises into the abdomen, by the sixth month reaching the umbilicus. Fibroid or other tumours of the uterus, ovarian tumours, solid or cystic, may attain a large size and become abdominal. The urinary bladder, if it contain over 10 ounces of urine, gives rise to a swelling, dull on percussion, immediately above the pubes.

CHAPTER VIII

EXAMINATION OF THE URINE

Method of examination—Naked-eye examination (colour, translucency, odour, reaction, density, quantity)—Chemical examination (albumin, sugar, diacetic acid, urea, bile, blood, uric acid, indican, chlorides)—Microscopical examination (pus, casts, epithelium, urates, phosphates, oxalates, micro-organisms, etc.).

Method of Examination.—It is recommended that the following procedure be adopted in examining a specimen of urine :

The sample should, if possible, be taken from the accumulated excretion of twenty-four hours. Observe its *colonr, translucency,* and *odour ;* test its *reaction* and *specific gravity*. Find out, if possible, the *quantity* of urine passed in twenty-four hours. Should the urine be turbid, proceed as described below under *Translucency*. Test for *albumin* and *sugar*. Should the latter be present, a further examination for *acetone* and *diacetic acid* (and possibly *pentose*) is to be made. The quantity of *urea* present is next to be determined. *Bile, blood, uric acid,* and *indican,* may be sought.

Should any abnormality be discovered or any deposit be present, examine microscopically for *casts, blood, pus, epithelial cells, crystals,* etc. In some cases a *bacteriological examination* is to be made. In this event care must be exercised to obtain a specimen free from accidental contamination.

In the following pages details of the examination on the above lines are furnished. Those tests which have been found most serviceable for hospital work are alone described.

1. **Naked-Eye Examination**—*Colour*.—Urine of high specific gravity is dark in colour, except that of diabetes mellitus. Smoky urine usually contains a small quantity of blood ; when dark, like porter, more copious blood. Various shades of brown indicate blood, carbolic acid, bile, or melanin. Greenish : bile, salol, carbolic acid. Whitish or yellowish : phosphates, pus, oxalates. Bright yellow after the administration of santonin. Aniline dyes taken by the mouth give their colour in many cases to the urine.

Translucency.—Normal urine is clear. After standing, a semitransparent mass of mucus collects at the bottom of the vessel. A brick-red or pink deposit on cooling indicates urates. If turbid, use the following tests : Place about 2 drachms of turbid urine in a test-tube, add about 5 drops of dilute acetic acid. If the urine clears, phosphates are present ; if not, boil. If it clears, 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 it clears, it is oxalate of lime ; if not, it is to be examined microscopically (see below).

Odour is increased in urine containing a larger amount of urea than normal (specific gravity high). It becomes ammoniacal when putrefactive changes have occurred in the bladder. It is sweet-smelling in diabetes. Garlic, copaiba, sandalwood, give their odour to the urine.

Reaction.—Test with litmus-paper. If the same urine turns blue paper red and red blue, it is said to be *amphoteric*. A quantitative estimation of acidity is rarely required (gout, diabetes). See Appendix VI.

Normal urine is acid, owing to the presence of acid phosphates. It is rendered alkaline by prolonged cold baths, by dyspepsia, anæmia, debility, and by the administration of alkaline drugs.

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. Acidity of the urine is increased by exercise, hot baths, fevers, and by all conditions in which the concentration of the urine is increased. Gout, acute and chronic rheumatism, diabetes, and fevers, also a diet chiefly of animal food, cause an increase in acidity.

Specific Gravity.—Average healthy limits: 1015 to 1025. The ordinary urinometer is the best means of observing the density. 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, the urine may be diluted, and the correct density is easily calculated. The specific gravity varies with the temperature. In this country the urinometer is usually graduated for a temperature of 60° F. If the temperature of the urine be much over or under that figure, add 1 to or subtract 1 from the reading for every 5° F.

Alterations in the density depend chiefly on variations in (1) the quantity of urea excreted by the kidneys, and (2) the bulk of water which has passed through them. Albumin, if present, has only a slight effect in raising the density. The specific gravity is increased when the urine is scanty, as in fevers and profuse sweating. In diabetes mellitus, however, where the quantity is invariably increased, the specific gravity is raised by the sugar in solution. Diminished density is observed

in many conditions of cachexia, in neuroses, in diabetes insipidus, in the small red kidney, in the small white kidney, in hydronephrosis, and in cystic disease of the kidneys.

Quantity.—The normal adult passes about 50 ounces daily.

The quantity is *increased* (polyuria) by cold (inhibiting perspiration); recent copious draughts of any fluid; hysteria, epilepsy, or simple emotional excitement; diuretic drugs—*e.g.*, digitalis, broom, nitrate of potash, etc.; absorption of dropsical effusions; diabetes; granular or contracted kidneys (the small red and small white kidney); the lardaceous kidney.

The quantity is *decreased* by excessive perspiration; diminished absorption of fluid from the stomach; excessive loss of fluids, as in diarrhœa, severe hæmorrhage, vomiting, cholera; fevers; shock; active or passive congestion of the kidneys; acute and chronic tubular nephritis; advancing dropsy.

2. **Chemical Examination.—Albumin** usually occurs in the urine in the form of serum-albumin, which is often accompanied by globulin.

Tests—Heat.—Filter the urine if turbid; add 4 or 5 drops of dilute acetic acid, unless the reaction is distinctly acid (excessive acidity interferes with the test); boil. A white precipitate is serum-albumin or globulin.

Nitric Acid (*Heller's Test*).—A drachm of strong nitric acid is placed in a test-tube; on its surface run carefully by means of a pipette about 2 drachms of urine. If albumin be present, a precipitate will be formed at the junction of the two fluids. The test-tube should be allowed to stand for a couple of minutes if no precipitate appears, as it may be delayed in forming when the quantity of albumin present is very small.

Fallacies.—A haze, found chiefly towards the upper part of the layer of urine, if unaffected by boiling, is *mucin* or *nucleo-albumin ;* if it dissolves on heating, it is *hetero-albumose.* If the urine is of high specific gravity, a precipitate of nitrate of urea is formed. Dilute the urine and test again, and the deposit is not formed. Balsams—*e.g.*, turpentine or copaiba—in the urine give a cloud with nitric acid, dissolving in ether or alcohol.

Picric Acid.—Place equal parts of urine and of a saturated solution of picric acid in a test-tube. A whitish-yellow cloud forms if there be albumin, albumoses, nucleo-albumin, antipyrine, or quinine. All these precipitates, except that of albumin, disappear on heating.

Other Tests.—Ferrocyanide of Potash with acetic acid : a turbidity produced by albumin and albumoses.

Salicyl-sulphonic acid causes a white precipitate with albumin and albumoses.

Biuret Test.—Copper sulphate and caustic soda give rose-pink colour with albumoses, violet with albumin.

If, as rarely happens, it is considered necessary to distinguish the different varieties of proteids in the urine, the picric acid and biuret tests suffice. Albumosuria has been observed in cases of myeloid sarcoma, acute yellow atrophy of the liver, phosphorus-poisoning, in abscesses, in resolving pneumonia, and in the puerperal period.

Note the *quantity* of albumin passed. Esbach's albuminometer is a simple and sufficiently accurate apparatus. Measured quantities of urine (which is to be filtered if turbid) and a solution of picric and citric acids are placed in a graduated tube. The precipitate from proteids is allowed to settle for twelve hours, and the level to which it has reached read off. The figures

represent the number of grammes of dried albumin in a litre of urine—*i.e.*, 1 per 1,000, or $\frac{1}{10}$ per cent.

Albuminuria may be renal or extrarenal. The latter group of conditions are of surgical interest, and are usually associated with the presence of pus (*pyuria*) or blood (*hæmaturia*) in the urine. The source of the albuminous and cellular addition to the urine may be the pelvis of the kidney, the ureters, bladder, urethra, vagina, or prepuce. When the pus, blood, and other cells, have been removed by filtration, the amount of albumin present is found to be small, except in cases where the kidneys also are affected.

Renal albuminuria is found in three groups of conditions: (1) Functional or physiological; (2) pathological, without definite kidney disease; (3) due to disease of the kidneys.

(1) *Functional albuminuria* is of a transitory character, and it is believed that in most cases there is no anatomical change in the kidneys. There is, however, a very general opinion held that even the transitory affections causing albuminuria leave the kidneys in some slight degree damaged, and that such functional albuminuria eventually in many cases develops into true nephritis.

The following varieties of functional albuminuria may be enumerated: *Periodic, intermittent, cyclical,* or *paroxysmal.* Here the albumin appears at more or less regular intervals. *Albuminuria of adolescents:* chiefly among boys, usually increased by exercise. *Postural or orthostatic:* it is found on assuming the erect posture. *Dietetic* occurs during digestion. *Thermal:* heat or cold may cause it.

These conditions are distinguished from the albuminuria of nephritis by the absence of casts, except, perhaps, some hyaline forms; by absence of the cardio-

vascular changes of nephritis ; and by the fact that the albumin in the urine is of temporary occurrence.

(2) *Albuminuria due to Disease elsewhere than in the Kidneys.*—Slight temporary changes in the kidney epithelium may be found in this group, which includes *febrile, toxic* (syphilis, gout, lead and mercury poisoning), and *nervous* albuminuria (epilepsy, tetanus, brain injuries).

(3) Albuminuria with definite kidney lesion is found in the following conditions : (*a*) *Congestion* of the kidneys, either active (early nephritis, kidney irritants—*e.g.*, turpentine, cantharides, alcohol, etc.) or passive (heart disease, abdominal tumours). (*b*) *Acute and chronic nephritis :* here the albumin may be abundant, and casts are usually present in the urine in considerable numbers and variety. (*c*) *Granular* or *small red kidney :* the albumin is scanty or may at times be absent, casts are few, and the quantity of urine passed is large. (*d*) *Lardaceous* or *amyloid kidney :* albumin often abundant, and a large quantity of urine is excreted.

Sugar.—Glucose is the only sugar of clinical importance, and is recognized by the following tests :

Fehling's Test.—The reagent is composed of two solutions : (1) Sulphate of copper, 34·64 grammes to 500 c.c. water ; and (2) Rochelle salts, 180 grammes ; caustic soda, 70 grammes ; water, 500 c.c. Take equal parts—say 1 drachm—of the solutions in a test-tube ; boil. If the fluid remains clear, add urine (free from albumin, and filtered if turbid) drop by drop till not more than a quantity equal to that of the reagent has been added. If sugar be present, the blue colour is discharged and a yellowish-orange precipitate of cuprous oxide forms.

Fallacies.—A similar reduction of the copper salt is produced by uric acid, excess of urates, and glycuronic

acid. Add to the urine one-fourth of its bulk of a hot 10 per cent. solution of acetate of lead, which precipitates these bodies, but not sugar. Glycuronic acid is not fermented by yeast.

Phenylhydrazine Test (von Jaksch).—Add to a couple of drachms of urine in a test-tube as much phenylhydrazine hydrochloride as will lie on the point of a penknife (7 or 8 grains), and twice as much sodium acetate. Keep in a beaker of boiling water for half an hour. If glucose be present, a yellowish precipitate of glucosazone forms (sheaves and bundles of needle-like microscopical crystals).

Fermentation Test.—It is convenient to have a specially-formed tube for this test, and when suitably graduated it forms a quantitative test also. An ordinary Doremus ureometer tube does very well. Dissolve a piece of fresh yeast about the size of a pea in enough acidulated urine to fill the Doremus tube, and set it aside in a warm place for twelve hours. If glucose be present, gas is generated (the volume of gas produced is a measure of the quantity of sugar present).

Fallacies.—Urine may contain fermenting bacteria; the yeast may be inert or may be contaminated with starch. Test the yeast for starch with tinct. iodine (turns starch blue), and perform control experiments with cane-sugar solution and yeast, and with urine without yeast.

Quantitative Tests for Sugar—Fehling's Solution.—0·005 gramme of glucose reduces all the cupric salt in 1 c.c. of Fehling's solution (combined Nos. 1 and 2). Place 10 c.c. of urine diluted to 100 c.c. in a burette, and run in drop by drop into a porcelain capsule containing 10 c.c. of Fehling's solution diluted, and kept gently boiling. The moment the colour is discharged and the precipitate begins to form, note the quantity of

urine expended. Suppose this is 20 c.c., the percentage of glucose is calculated thus:

$$\frac{0{\cdot}05 \times 100}{2} = \frac{5}{2} = 2{\cdot}5 \text{ per cent.}$$

A modification of the above method by Pavy is more convenient. By the addition of ammonia the cuprous oxide is kept dissolved, and the reaction is the disappearance of the blue colour. This can be more accurately determined than the point at which Fehling's reaction takes place. Pavy's solution is one-tenth the strength of Fehling's.

Fermentation.—A rough quantitative method is to compare the specific gravity of the urine before and after fermentation. For each degree of density lost by the urine there is 1 grain of sugar to the ounce of fluid.

Sugar may be found temporarily in the urine in a variety of affections—viz., dyspepsia, gout, asthma, epilepsy; and certain drugs and poisons—*e.g.*, chloroform, ether, antipyrine, carbonic oxide. Should the glycosuria persist, and be accompanied by thirst, polyuria, and wasting, the patient is suffering from diabetes mellitus. The diagnosis of this disease is obvious as a rule, but in some cases there is no loss of flesh, and it is only by examination of the urine or by the discovery of some of the usual diabetic sequelæ (cataract, carbuncle, peripheral neuritis) that the condition is recognized.

Acetone (*Legal's Test*).—Alkalinize the urine with liq. potassæ, and add a solution (0·1 gramme to 15 c.c. water) of nitroprusside of soda. Acetone gives a ruby-red colour, becoming violet on acidifying with acetic acid.

Aceto-Acetic Acid (*Diacetic Acid*).—Continue adding a few drops of dilute liquor ferri perchloridi till the pre-

cipitate of ferri phosphate ceases. Filter, and add a few more drops of iron. Aceto-acetic acid gives a violet-red colour.

Fallacies.—If the urine has been previously boiled, this reaction fails. The same reaction is given by antipyrine, salicylates, and carbolic acid.

Acetone and diacetic acid are derivatives of hydroxybutyric acid, and when present they signify grave metabolic disturbance. They occur in severe diabetes, and the diminished alkalinity of the blood found in this disease is due to hydroxybutyric acid.

Pentose reduces copper like glucose, and forms pentosazones with phenylhydrazine, but does not ferment.

Orcin Test (*Bial's*).—Reagent consists of hydrochloric acid (30 per cent.), 500 c.c. ; orcin, 1 gramme ; solution of ferric chloride (10 per cent.), 25 drops. Boil 5 c.c. of this, add a few drops of urine ; a green colour indicates pentoses.

The occasional appearance of this form of sugar in the urine may give rise to an erroneous diagnosis of diabetes. The nature of its occurrence is uncertain.

Urea.—The sample should, if possible, be taken from a mixture of twenty-four hours' urine.

The presence of urea is ascertained by evaporating a few drops of urine with a drop of nitric acid on a slide. Nitrate of urea is found as hexagonal or rhombic crystals.

The quantity of urea is to be determined by setting free and measuring its nitrogen, of which there are 372 c.c. to each gramme of urea. This is effected by the action of alkaline sodium hypobromite (2 c.c. of bromine in 23 c.c. of a 40 per cent. solution of caustic soda). The most convenient apparatus for the measurement of the gas is Hind's modification of Doremus's

ureometer. Larger and more accurate instruments are Gerrard's and Dupré's ureometers.

The average quantity of urea excreted in health is 2 per cent. of the total urine passed. Increase in this amount (*azoturia*) results from excessive nitrogenous diet or excessive destruction of nitrogenous tissues. A diminished output of urea is the result of deficiency of nitrogen in the food, disease of the liver (cirrhosis, cancer, acute yellow atrophy), and renal inefficiency (Bright's disease, cystic kidney).

Bile.—The urine is coloured dark yellow to brown or green, with yellow froth, and stains linen yellow.

Gmelin's Test.—Fuming nitric acid oxidizes the bile-pigment, producing layers or rings of colours: green, blue, violet, red, and yellow. Pass the urine through white filter-paper ; a drop of fuming nitric acid is placed on the bile-stained paper.

The presence of bile-pigment in the urine indicates in most cases obstruction to the outflow of bile.

Blood, if scanty, imparts a smoky appearance to the urine ; if copious, it is brownish-black.

Guaiacum Test.—Moisten a small piece of lint with the urine, add a drop of freshly-prepared tincture of guaiacum, and on the same spot a drop of ozonic ether. If blood is present, a blue colour appears. The same test may be done by the contact method. A drachm of ozonic ether with a few drops of tincture of guaiacum are placed in a test-tube ; a drachm or so of urine is placed on the surface of this fluid by means of a pipette. A blue layer forms at the junction of the two fluids.

Fallacies.—Iodides and saliva give a similar reaction.

Heller's Test.—Alkalinize the urine strongly with liquor potassæ ; boil. A brownish-red deposit consists

of earthy phosphates and hæmatin. The supernatant fluid is greenish-coloured.

Fallacies.—Rhubarb, senna, and santonin, give a similar result. If the urine was originally alkaline, it gives no such reaction until a little lime-water has been added.

Hæmin Test.—Place a little of the urinary sediment on a glass slide, add a minute crystal or two of common salt, place a cover-glass on it, run in a drop of glacial acetic acid, warm gently, allow it to cool, and mahogany-red rhombic crystals of hæmin will be found by the microscope.

Spectroscope.—A convenient pocket spectroscope can be obtained which will show the absorption bands of hæmoglobin.

Often the blood-corpuscles can be recognized by the microscope (*hæmaturia*). They do not lie in rouleaux, and may be misshapen. If the chemical tests show blood, but the red corpuscles are not found microscopically, the condition is termed *hæmoglobinuria.*

Blood may come from any portion of the urinary tract, from the meatus to the kidney—viz., urethral ulcers or injuries; bladder lesions—*e.g.*, prostatitis, varicose veins, villous growths, tubercular or malignant ulceration, stone ; in the ureter and pelvis of the kidney tubercular disease, malignant tumours, and renal calculus, cause bleeding ; hæmorrhage from the kidney occurs especially in acute and subacute nephritis, but also in chronic nephritis and granular kidney, in hæmophilia, with some acute fevers, and from certain tropical parasites.

Hæmoglobinuria, the result of destruction or hæmolysis of the red corpuscles, occurs as a result of certain poisons in the blood—*e.g.*, carbolic acid, chlorate of

potash in large doses, carbon monoxide, quinine, toxins of fevers (scarlet, yellow, typhoid). A paroxysmal hæmoglobinuria sometimes occurs, apparently caused by some exposure or excessive exercise in susceptible individuals.

Uric Acid.—Normally uric acid is not found free in the urine, but as urates of sodium, potassium, and calcium. If an acid urine stands for some time, a scanty reddish deposit of uric acid comes down. It is composed of microscopical pink-coloured crystals of a variety of forms. Chemically it is detected by the *murexide test*, which also indicates urates. A little of the deposit is placed in a porcelain capsule with a few drops of dilute nitric acid, and evaporated to dryness. Add a drop or two of ammonia to the yellowish residue, and a violet colour appears ; add a drop or two of caustic potash, and the colour becomes more blue.

An excessive excretion of uric acid signifies an unduly active proteid metabolism, as seen in a variety of wasting and acute diseases.

Indican, the indoxyl-sulphate of potash, is normally present in the urine in small quantity. It is increased in constipation, intestinal obstruction, suppuration, and excessive proportion of animal food.

Jaffé's Test.—Take about 2 drachms each of urine and strong hydrochloric acid ; add a few drops of a 5 per cent. solution of calcium hypochlorite till a blue colour appears ; add a little chloroform, and shake well. On standing, the chloroform coloured by indican settles to the bottom as a blue layer of fluid.

Chlorides may at times be estimated. Normally they exist (chiefly in the form of chloride of sodium) in the urine, 12 to 14 grammes being excreted daily by a healthy adult.

Mohr's Test.—To 10 c.c. urine add 30 to 50 c.c. distilled water and 2 or 3 drops of a 10 per cent. solution of potassium chromate. From a burette run in nitrate of silver solution (29·042 grammes nitrate of silver to 1 litre of water) until a permanent red colour is obtained, and note the quantity of silver solution expended. For every cubic centimetre of silver solution there is present 0·01 gramme of sodium chloride. The result is a little too high, as other substances are present which also unite with the silver solution ; deduct, therefore, 1 c.c. from the amount of silver solution used.

Two urinary reactions having reference to the diagnosis of typhoid fever may be here referred to—viz., *Ehrlich's diazo reaction* (see Appendix VII.) and *Russo's methylene-blue reaction* (see Appendix VIII.).

3. **Microscopical Examination.**—The deposit should be centrifugalized.

Blood.—See above.

Pus.—The deposit resembles phosphates, but if the urine is, or is rendered, alkaline, it has a slimy, ropy consistency. Microscopically the pus cells are seen to be larger than red blood cells, with a divided nucleus. Add acetic acid to the slide, and the bodies of the cells become transparent, while their nuclei remain visible.

If from the bladder (tuberculosis, cystitis, calculus, prostatic enlargement, or stricture), the reaction is usually alkaline ; but if due to *Bacillus coli*, it is acid. It is also usually acid if the seat of the disease is the ureters or kidneys.

After filtering pus cells from the urine, a little albumin is found in the filtrate. Should, however, a considerable quantity of albumin be present, it is probably due to renal albuminuria.

Tube-casts are moulds of the renal tubules formed of

albuminous material, to which have been added a variety of substances giving the respective characters to the casts. The following varieties may be enumerated: *Hyaline* casts: clear, semitransparent. *Epithelial* casts: more or less completely composed of epithelial cells. If studded with granules, the detritus of broken-up cells, they are *granular* casts. These granules may be largely composed of fat droplets, which, if abundant, give their name to *fatty* casts. *Blood* casts have a number of red corpuscles incorporated in them. If the cells are white corpuscles, the casts are termed *leucocytic* casts. *Waxy* casts are probably epithelial or hyaline casts which have undergone degeneration owing to their retention in the tubules for a considerable time. They are broad, pale, often yellowish, and highly refractive, with sharp outline.

Casts are almost invariably an indication of disease of the kidneys, the chief exception being the fact that hyaline and occasionally granular casts are sometimes found in the urine of cases of jaundice, where the kidney is not apparently affected; also, hyaline casts may be found in some cases of functional albuminuria.

Any or all of the varieties of casts named may be found in cases of nephritis, but by observing which type occurs most frequently in any case some help may be gained in the diagnosis. Thus, blood casts are common in acute nephritis, and at times in chronic nephritis. Epithelial, granular, and fatty casts are characteristic of tubal nephritis, and are less often seen in the contracted forms (small white and small red kidney). Waxy casts are not often found in cases of amyloid kidney, but are an evidence of chronicity. Hyaline casts are of little or no diagnostic value.

Epithelial cells of many forms may be found in the

urine—viz., *squamous* cells from the superficial layers of bladder mucous membrane, similar shaped but larger flattened cells from the vagina in females, *cubical* or *tailed* cells from the deeper layers in the bladder, and the somewhat smaller *columnar* cells (or rounded with large undivided nucleus) from the kidney tubules.

Urates form the ordinary brick-red deposit in the urine. The commonest are the amorphous urates of sodium, potassium, and ammonium; but crystalline forms may be observed—viz., urate of sodium, urate of ammonium (more or less spherical bodies with spines).

Phosphates are precipitated in alkaline urine, and are commonly found as an amorphous deposit of calcium, or more rarely magnesium phosphate. Crystallized phosphates in smaller quantity are usually found with the amorphous. The "triple phosphate," or ammonio-magnesium phosphate, is common, and is seen in the well-known "knife-rest" or "coffin-lid" and feathery star crystals. The so-called "stellar phosphates" are often found in acid urine.

The presence of phosphates is usually merely a result of alkaline reaction of the fluid, and is common in cases of nervous dyspepsia. A definite increase, however, in the quantity of phosphates (phosphaturia) is seen in wasting diseases, in rickets, in severe anæmia, and in some affections of the nervous system.

Oxalate of lime occurs in urine as octahedral or "envelope," or more rarely as "dumb-bell" crystals. It may be found after indiscretions in diet. Certain vegetables (rhubarb, tomatoes, onions), want of exercise, emotional disturbance, all tend to its appearance. It may cause pain and irritation in the urinary tract, and, in common with uric acid, phosphates, and other crystals, may go to form calculi.

Carbonate of lime rarely occurs in urine, and is usually an amorphous deposit dissolving with effervescence on the addition of an acid.

Cystin, cholesterin, xanthin, leucin, and *tyrosin,* are rare constituents of urine. The last two occur in serious disease of the liver, in pernicious anæmia, and in phosphorus-poisoning. *Fat* is found in droplets free in the urine in some cases of fatty degeneration of the urine. Spermatozoa, " cylindroids," " prostatic threads," parasites, masses of bacteria, and foreign bodies of all description, may be recognized in the urine.

Micro-organisms.—See Appendix II.

CHAPTER IX

NERVOUS SYSTEM

Routine method of examination—Defects of movement—Loss of power—Increased muscular action—Disorderly movements—Reflexes—Sensory disturbances—Psychical functions.

Routine Method of Examination.—The condition of the nervous system is to be investigated by a methodical examination. The following routine is recommended :

1. Observe if any *defect of movement* exists. Exclude by careful examination any lesion of the joints, bones, or muscles, which might affect the motor functions of the part. In connection with movements, the *tone* and *nutrition* of the muscles are to be examined.

2. The condition of the *reflexes* is to be observed, including cutaneous, deep, vascular, and visceral reflex functions.

3. Next, the *sensory* functions are to be examined, including common sensation, muscular sense, pain, heat, cold, abnormal sensations, and the special senses.

4. Lastly, the higher or *intellectual faculties* are to be studied, including consciousness, the state of the memory, power of concentration, attention and understanding, the capabilities of speech and writing.

1. **Defects of Movement.**—In order to inquire into the state of any muscle or group of muscles, the patient is directed to perform various acts which would bring the

muscles into play. He must, first, as far as possible, use the limbs as a whole and the trunk muscles. Thus, he is directed to turn over on either side in bed, to sit up, to draw up and cross his legs, raise his arms in different directions. He should stand, if possible, and walk. His mode of walking and turning, rising and sitting, are to be observed. The separate groups of muscles, and even individual muscles, may be investigated by testing their ability to execute the various actions which they should normally perform. During this part of the examination the state of nutrition or tone of the muscles is to be observed. The firm consistency of normal muscles, as felt by the fingers, may be lost ; or, on the other hand, even in a paralyzed limb, it may be equal to or greater than that of healthy muscles. A further investigation of muscular nutrition by electrical stimulation has at times to be carried out (see Appendix IX.). In this connection the condition of the reflexes has also to be taken into consideration (see below). Efficiency of movement depends on the integrity of the *motor tract*, which is the pathway of nerve impulses proceeding from the motor area of the cortex to the efferent nerve endings in the muscles. Clinically we recognize two divisions or segments of the motor tract. The upper segment (*central neurons*) extends from the cortex to the lower motor centres or nuclei from which the respective cranial and spinal efferent nerves arise. These nuclei in the case of the cranial nerves lie in the crura, pons, and medulla, below the decussation of the fibres of the upper segment, and for the spinal nerves the nuclei are situated in the anterior horns of the grey matter of the cord. In addition to the function of directing efferent impulses, the nuclei exercise a trophic influence on the nerves and muscles in

connection with them. Destruction of the nuclei or of the motor nerves proceeding from them causes atrophy of the muscle fibres supplied by the affected nerves. The portions of the motor tract from, and including, the ganglion cells forming the nuclei to the peripheral ends of the nerve fibres are known as the *peripheral neurons*, or lower segment of the motor tract.

Three disturbances of the normal movements may be observed: (1) Weakened or abolished; (2) increased or exaggerated; and (3) perverted or disorderly movements.

(1) **Weakened or Abolished Movements,** the result of disease of the central or peripheral nervous system, are described as *paralysis* when the loss of power in any lesion is complete, and *paresis* when partial.

Two main groups of paralysis may be recognized: (i.) *Spastic* or *tonic paralysis;* and (ii.) *flaccid* or *atrophic paralysis*.

(i.) *Spastic paralysis* results from a lesion of the central or upper neurons; there is, therefore, no interference with the nutrition of the affected muscles, and their tone is equal to or greater than that of normal muscles. On making the patient perform some movement, his affected limbs are noticed to be not only feeble, but also stiff. The reflexes are exaggerated in most cases. Any lesion which interrupts the passage of nerve impulses from the motor area of the cortex to the nuclei may cause a paralysis of this description. It is therefore found in *transverse interruption* of the cord from any cause (compression, myelitis, hæmorrhage into the cord); *sclerotic changes* in the upper segment of the motor tract, either in the brain or spinal cord; *cerebral disease* or *injury* involving the motor tract (see Appendix XI.).

(ii.) *Flaccid paralysis* is recognized by the extreme wasting of the muscles, which are soft and flaccid when handled; the limb is loose and flail-like in its movements. The lesion which produces flaccid paralysis is situated in the lower or peripheral neuron, whereby not only the voluntary power of movement is lost, but the nutrition rapidly deteriorates, and the reflexes are diminished or abolished. The disease may be either in the central organs (brain or cord) or in the peripheral nerves. In the former case the anterior horns of the cord, or the nuclei of the cranial nerves, are injured, either by inflammatory or degenerative changes as a rule, though traumatism, tumours, hæmorrhage, and other causes, may occur. The peripheral nerve lesions are varied in their origin and in their result according to the situation and action of the affected muscles (see Appendix XI.).

The Regions of the Body affected.—The loss of power may be *bilateral*. When, as in the majority of cases, this is due to a lesion of the cord, the condition is termed *paraplegia*. If the damage is below the cervical region, the legs alone are paralyzed (*paraplegia cruralis*); if at or above the cervical enlargement of the cord, all four limbs are powerless (*paraplegia totalis*).

An injury or disease of the brain which interrupts the fibres from both sides of the brain may, less frequently, be the cause of a bilateral paralysis, which is termed *diplegia* or *cerebral paraplegia*. In some cases the limbs of one side and the facial or eye muscles of the other are paralyzed (*crossed* or *alternate paralysis*); here the lesion is likely to have damaged the nucleus of the sixth or seventh nerve in the pons, or the third or fourth nerve in the crus, together with the fibres to the limb from the other side of the brain.

A bilateral paralysis may also be caused by disease of the peripheral nerves. This is most likely to be the result of some blood poison (multiple neuritis); it is found that, while such poisons may injuriously affect any nerve, they have in many cases a selective influence which causes certain nerves to be most vulnerable to the respective poisons. Thus alcohol attacks by preference the external popliteal nerve, giving rise to inability to flex the foot dorsally, to extend the toes on the dorsum of the foot, and to raise the outer edge of the foot. This paralysis causes *foot-drop* and the so-called *steppage gait*, in which the knee has to be raised unduly high in order that the toes may clear the ground. The same nerve is especially liable to be affected by the poison of diphtheria, diabetes, beri-beri, etc. Diphtheria has also a preference for the soft palate and for the muscles of the eye, especially for the ciliary muscles which control accommodation. Lead-poisoning selects mainly the extensor muscles of the forearm (the supinator longus escapes), causing the condition known as *wrist-drop;* this is more commonly symmetrical. A one-sided wrist-drop often occurs as a result of injury to the musculo-spiral nerve; this may result from pressure of the arm over the back of a chair during sleep (*Saturday night palsy*).

A paralysis of one side of the body only is not often due to spinal cord disease, where damage from disease or injury, even if it begin at one side of the cord, is apt to affect the adjoining nerve structures controlling both sides of the body. It is in the brain or in the peripheral nerves that a one-sided paralysis (*hemiplegia*) is likely to originate.

The lesion of the brain causing paralysis is of a destructive character; it is commonly of vascular origin—

either hæmorrhage from a ruptured artery, embolism, or thrombosis. Tumours, abscesses, or degenerations, are also possible causes. A consideration of the mode of onset of the paralysis, the condition of the bloodvessels, heart, and kidneys, the habits and age of the patient, render a diagnosis of the cause of hemiplegia possible.

The seat of the lesion is in many cases the internal capsule, where the fibres of the motor tract are brought together in a small space. If lower down the motor tract, the nuclei of motor nerves are likely to be damaged, causing the crossed paralysis referred to above. When higher than the internal capsule, one limb only may be paralyzed (*monoplegia*), or the subcortical or cortical regions may be involved, giving rise to twitchings or convulsions.

A not uncommon cause of one-sided paralysis is infantile paralysis ; here the lesion is an acute inflammation involving the anterior cornua of the cord, and in many instances only a limited portion of the cord is affected, though it is also common for regions on both sides of the body to be affected. A chronic inflammation or degeneration affecting the same regions of the cord produces the form of paralysis known as progressive muscular atrophy, which is most frequently, but not exclusively, bilateral. When the cranial nerve nuclei (chiefly those in the medulla) suffer from a similar disease, the symptoms of bulbar paralysis are produced —that is, interference with the movements of the tongue, palate, pharynx, and larynx. When this form of paralysis is due to lesion of the peripheral nerves arising from the medulla, the loss of power may be one-sided ; if bilateral, it is termed "pseudo-bulbar paralysis."

Paralysis restricted to one or more groups of muscles,

or even to one muscle, is usually the result of disease of the peripheral nerves, but may be due to the selective action of morbid processes whereby the nuclei of origin of the nerves are individually affected. Less frequently a restricted paralysis is the result of a supranuclear lesion, such as a hæmorrhage, softening, or tumour, involving a limited number of fibres proceeding from the cortex to the lower centres or nuclei.

Oculo-motor paralysis is shown by loss of power in the movements of the eye and of the upper lid (third, fourth, and sixth cranial nerves). Determine in which direction and in which eye the movements are defective. Note if either upper lid droops (*ptosis*). Observe if the visual axes remain parallel, or if, on the contrary, they converge or diverge (convergent or divergent *squint* or *strabismus*). Cover the sound eye, and tell the patient to endeavour to look steadily at an object with the affected eye ; in oculo-motor paralysis the squint is increased (*secondary deviation*). In paralytic squint *double vision* is common ; in the sound eye the image falls on the macula lutea, and is therefore more distinct (*true image*) than that which falls on another portion of the retina of the paralyzed eye (*false image*). The respective positions of the true and false images as seen by the patient serve to determine which of the muscles is at fault.

Conjugate Deviation.—Both eyes are turned to one side, without squinting, and the head is often turned in the same direction. This paralysis means a lesion of the sixth nerve on one side (external rectus) and of the association fibres from the nucleus of the third on the other side, controlling the internal rectus. If the lesion be in the pons (*e.g.*, hæmorrhage), injuring the nucleus of the sixth, the eyes turn away to the side opposite

the lesion. Should the injury be supranuclear, before decussation has taken place the eyes will look toward the lesion. If the injury should be cortical, a stimulation of the opposite sixth nerve may result, instead of a paralysis, and the eyes will look away from the lesion.

Lower Jaw Paralysis (fifth cranial nerve).—The temporal, masseter, and pterygoid muscles have lost tone and power. On depressing the jaw it is deviated to the paralyzed side.

Facial Paralysis (seventh cranial nerve).—The affected side is less marked by skin folds, and is in consequence expressionless. Direct the patient to frown, raise his eyebrows, tightly close his eyes, blow out his cheeks, whistle, draw back the corners of his mouth to show his teeth. When (as is often the case) this paralysis is part of the result of a supranuclear lesion, the paralysis is less marked in the upper than in the lower half of the face. This is due to the bilateral innervation which is believed to be supplied to those regions which are habitually used in concert with their fellows of the opposite side of the body (*Broadbent's law*). In cases of peripheral seventh nerve disease the patient is unable to close his affected eye, and on endeavouring to do so the eye rolls up under the upper lid (*Bell's phenomenon*). Weakness of the cheek muscles causes difficulty in mastication, and speech may be indistinct. Lesions of the nerve in the bony canal may cause loss of the taste sense in the anterior half of the tongue and diminution of the secretion of saliva, owing to injury to the chorda tympani.

Sterno-mastoid paralysis (eleventh cranial nerve) causes difficulty in turning the head to the sound side. The upper half of the trapezius is supplied by the same nerve

and is paralyzed by its lesions ; the point of the shoulder is lowered, and the inferior angle of the scapula approaches the spine ; there is difficulty in raising the arm above the level of the head.

Diaphragm paralysis occurs as a result of disease or injury to the phrenic nerve (third and fourth cervical nerves).

During deep inspiration the epigastrium is observed (by inspection and palpation) to fall inwards when the diaphragm is paralyzed. In health it bulges forward as the ribs rise during inspiration. In Chapter VII. a variety of causes for immobility of the diaphragm are enumerated.

Paralysis of the arm, from injury to the brachial plexus, may involve all the muscles of the limb. The deltoid, biceps, brachialis anticus, and supinator longus, may be paralyzed (*Erb's paralysis*) by injury to the fifth and sixth cervical nerves. *Serratus magnus paralysis* (long thoracic nerve, fifth and sixth cervical) prevents the proper apposition of the scapula to the thorax. On raising the arm with a pushing action, the inferior angle of the shoulder-blade is rotated backward, while the bone projects like a wing. *Deltoid paralysis* results from lesions of the circumflex nerve ; the arm is raised with difficulty. *Paralysis of the hand ;* inability to oppose the thumb to the tips of the fingers (*ape-hand*), to flex the first row of interphalangeal joints, or the second row of the first and second fingers, together with imperfect pronation of the forearm and flexion of the wrist toward the radial side. This paralysis is due to lesion of the median nerve ; the usual wasting due to lower neuron lesions causes the characteristic appearance of the hand. Lesion of the ulnar nerve produces the following paralysis : Abduction of the thumb ; defective

lateral movements of the fingers, the hand being flat; extension of the metacarpo-phalangeal joints; imperfect extension of the two distal sets of phalanges, especially those of the ring and little finger; the wrist cannot be flexed toward the ulnar side. When this condition has existed for some time, the wasting and deformity produces the condition known as *claw-hand*.

Paralysis of the adductors of the thigh (inability to cross the legs), from lesion of the obturator nerve; this may occur during parturition. *Inability to extend the knee*, with loss of knee-jerks, results from lesion of the anterior crural nerve. *Paralysis of the hamstring muscles* and of the leg below the knee follows injury or disease of the sciatic nerve; this sometimes arises in the pelvis (tumours, parturition), or the nerve may be damaged at the hip or thigh.

(2) **Increased Involuntary Movements** of various descriptions are observed in certain affections of the nervous system, and are spoken of as *spasm*. They may take the form of (i.) continuous or *tonic* spasm; and (ii.) interrupted or *clonic* spasm.

The cause of increased muscular action is an irritation of the motor tract by a lesion or disorder, which may be of the same nature as those which have been already referred to as producing destruction of the motor tract. Indeed, the two conditions of paralysis and spasm may be found in the same patient for this reason; the ordinary spastic paralysis, however, is the result of a destructive lesion of the motor tract, together with exaltation of the reflexes, and not irritation of the motor centres.

Irritation of the nerve roots is less frequently a cause of spasm, and hysteria is not unusual as the chief factor in spasmodic affections.

(i.) *Tonic spasm* produces a rigidity of the limb or group of muscles affected. The following instances may be noted :

Trismus, spasm of the muscles of mastication, occurs early in tetanus and late in strychnine-poisoning. The *risus sardonicus* of these two diseases is a spasm of the facial muscles, and tonic spasms of the muscles of the trunk and limbs occur in the same affections, to which the following descriptive names have been given : *Opisthotonos*, muscles of the back, causing backward curving of the body ; *emprosthotonos*, forward curving ; *pleurosthotonos*, curving to one side ; *orthotonos*, the body remaining straight and rigid.

A tonic spasm of the arms and legs, known as *tetany*, consists in flexion of the wrist and metacarpo-phalangeal joints, extension of the finger-joints, and the thumb adducted across the palm ; elbow flexed ; leg and foot extended, and toes flexed. When tetany is present, if the facial nerve is struck just in front of the ear, the facial muscles contract (*Chvostek's sign*), and the motor nerves generally are unusually sensitive.

Torticollis, a spasm chiefly of the sterno-mastoid, tonic as a rule, but sometimes clonic.

Retraction of the head, owing to spasm of the posterior cervical and dorsal muscles, occurs as a result of intracranial irritation, commonly found in cerebral meningitis. In this disease spasm of the hamstring muscles is often found ; the hip-joint should be well flexed, and in cases of meningitis it is found impossible to straighten the leg at the knee (*Kernig's sign*).

A tonic spasm occurs in muscles which have been overworked, and is most common in the hand. It is known as *professional spasm* or *occupation neurosis*, and occurs as a cramp-like contraction of the muscles on

attempting to use the hand. Sometimes a trembling and weakness is found instead of spasm.

(ii.) *Clonic spasms* are seen as intermittent contractions of muscle groups ; the force, frequency, and regularity of recurrence of the contractions may be observed in all degrees of difference from the finest tremor to the most violent convulsion.

The finest movements of this description are *fibrillary twitchings* of very small bundles of muscle fibres, seen in wasting diseases. *Tremor* is the regular clonic spasm, which is sufficiently ample to move the limb. In some conditions (*e.g.*, paralysis agitans) it is worst while the limb is at rest. In other cases (*e.g.*, disseminate sclerosis) the trembling becomes more marked as the patient tries to control it in order to perform some purposeful movement (*intention tremor*). Tremor is a familiar symptom in functional nervous disturbances (hysteria, emotions) ; it is caused by exposure to cold ; a fine tremor is found in exophthalmic goitre. A tremor of the lips and tongue is seen in general paralysis of the insane and in alcoholism.

Rigors are a rather coarser tremor, involving most of the muscles of the body. They occur in fevers of some severity.

A regularly recurring clonic spasm of the ocular muscles is termed *nystagmus*. On causing the patient to turn his eyes strongly to one side, the head being maintained steady, movements of the eyeball, about two or three per second, are observed. They are usually in a lateral direction, but are occasionally upward or downward. It is chiefly in multiple sclerosis that nystagmus is seen, but it occurs in cases of cerebellar disease, Friedreich's ataxia, in cerebral hæmorrhage, and in some other rarer affections of the nervous system. In addition, defects

of sight from disease or injury of the eye are a common cause. Of this nature is miner's nystagmus, due to the continuous use of the eyes in a constrained position in bad light.

Irregular forcible contractions occur under a variety of circumstances, and from their resemblance to idiopathic epilepsy are grouped together as *epileptiform* attacks or *convulsive fits*. The latter name should be reserved for convulsive attacks accompanied by unconsciousness. The conditions which are likely to produce these clonic spasms belong to three classes : (1) Cortical disease or injury (Jacksonian epilepsy) ; (2) toxic conditions of the blood, causing irritation of the motor centres (uræmia, puerperal eclampsia, diabetes, asphyxia, opium-poisoning, etc.) ; (3) convulsive attacks without known lesions (hysteria, idiopathic epilepsy, infantile convulsions, chorea, and a variety of choreiform affections).

(3) **Disorderly Movements.**—Normal power of muscular contraction is of little use if the muscles do not act properly in harmony. Want of concerted contractions produces irregular and badly-directed movements of the affected limbs, the condition being known as *ataxia*, or *inco-ordination*. In health the individual is conscious, without the aid of sight, of the position of his limbs, and, owing to the information conveyed to his sensorium, he knows the amount of energy that is expended in any muscular action. It is this *strength sense*, or *innervation sense*, which is the chief factor in producing co-ordinated movements of the body.

In order to test co-ordination, the patient is directed to perform a variety of actions without the aid of sight. He is to stand, turn, walk, with his eyes open and then closed ; inability to maintain his balance while

standing with closed eyes is known as *Romberg's sign*. Want of neatness and accuracy in the various movements is to be noted. This is best marked when the eyes are closed. Thus, he may be directed to touch the tip of his nose with his forefinger, or bring the forefinger of each hand together, the eyes being closed. The legs may be tested by requesting him to describe circles or other figures in the air, or on the floor, with his great-toe, to touch certain spots with his toe or heel, to walk along a board or follow a pattern in the carpet, etc. His mode of progression, or *gait*, is to be observed; it may be *stamping*, *reeling*, or *stumbling*.

Inco-ordination is found in (1) lesions of the posterior columns of the cord, of which locomotor ataxia is the commonest example; (2) lesions of the cerebellum; (3) disease of the semicircular canals and vestibular nerves causes an ataxia similar to that of cerebellar disease (*vertigo*), and, when accompanied by vomiting, deafness, faintness, and other symptoms, is known as Ménière's disease—irritation in the external or middle ear is a common cause of vertigo; (4) other cerebral lesions, especially those of the parietal lobes, cause a form of ataxia; (5) lastly, peripheral nerve lesions may produce a condition closely resembling inco-ordination.

2. **Reflexes.**—Certain muscular contractions occur independently of volition; their normal occurrence depends on the integrity of a *reflex arc*, consisting of a sensory nerve, a motor centre (in the anterior cornua, medulla, pons, or crura), and an efferent nerve with its muscle. It is by a mechanism of this nature that the normal tone of muscles is maintained. It is found that the activity of the reflex acts is modified and controlled by nerve impulses descending to the reflex arc from higher cerebral centres. Owing to interruption in these con-

trolling or inhibiting pathways, such as occurs in supra-nuclear lesions in the brain and cord, the exaggerated reflexes occurring in spastic paralysis take place. There are other causes for increased reflex activity, such as the effects on the cord of the poison of tetanus, of hydrophobia, of strychnia, etc.; the influence of emotional stimulation is seen in the exaggerated reflexes of hysteria.

By stimulating the reflex arc, certain movements of the region are caused, and departures from the normal occurrence of these movements are to be noted. We must compare the resulting reflex movements with what we regard as normal, and also compare the movements on opposite sides of the body.

There are three groups of reflexes to be observed: (1) Deep or tendon reflexes; (2) skin reflexes; and (3) visceral reflexes.

(1) *Deep or tendon reflexes* are elicited by suddenly stretching still further a muscle which has already been put on the stretch. The most useful example is the *knee-jerk* or *patellar reflex.* The patient is seated, if possible, with his bent leg supported on the observer's hand; a sharp blow is struck on the patellar tendon, causing the extensors of the knee to contract, with brisk extension of the limb at the joint. This reaction may be rendered more obvious by distracting the patient's attention; thus, he should grip his hands and pull as if drawing them apart, his eyes meanwhile being directed to the ceiling. The knee-jerk is usually present in health; its absence is known as *Westphal's sign*, and may be an evidence of lesion of the reflex arc (locomotor ataxia, neuritis, etc.). *Patellar clonus* is an intermittent or clonic contraction of the extensors of the leg, when they have been suddenly stretched by quickly forcing

downward the patella, the leg being fully extended. It is only found when the knee-jerk is exaggerated. *Ankle clonus* is a rhythmical contraction of the calf muscles, observed in conditions of exalted reflexes; it is demonstrated by putting the muscles in question on the stretch by pressure on the sole of the foot, causing the foot to be flexed dorsally. This may be enough to produce the clonus, but it is usually necessary to give a stimulus by sudden pressure on the sole, further increasing the flexion of the foot. This phenomenon is rarely found in health. A single jerk (*ankle-jerk*) on striking the tendo Achillis is similarly a sign of increased reflexes.

Arm-jerks may be found at times in health, but are usually an evidence of excessive reflex activity. The following may be mentioned: *Wrist-jerk:* a tap on the extensor tendons just above the wrist causes the hand to be extended. *Elbow-jerk,* an extension of the bent forearm on striking the triceps tendon just above the joint; a flexion of this joint may also be obtained by striking the biceps or supinator longus tendons. *Scapulo-humeral reflex,* an external rotation of the arm on striking the spinal border of the scapula; when the reflex is exaggerated more extensive motions of the arm occur.

Jaw-jerk, not usually found in health. The patient's mouth being open, one finger of the observer is placed, pleximeter fashion, on the chin, and a downward stroke is given with the finger of the other hand; an upward jerk of the jaw follows, and if pressure be kept up clonic contractions may occur.

(2) *Skin or superficial reflexes* are of less diagnostic value, as, with the exception of the dorsal flexion of the toe, their occurrence is uncertain in health and disease. They are useful at times in estimating the level at which

spinal disease has occurred, and their occurrence on each side of the body should be tested and compared. They are obtained by stroking or gently irritating or pricking or pinching the skin, and noting if the appropriate movement takes place.

Plantar reflex is shown by involuntary drawing up of the limb ; in cases of exaggerated reflexes this movement is increased in activity, and in addition there may be dorsal flexion of the great-toe, the other toes being flexed towards the sole of the foot (*Babinski's sign*). In a normal state of the reflexes all the toes are flexed toward the sole of the foot, except in infants who have not learned to walk, and with whom dorsal flexion of the great toe is the rule. *Cremaster reflex :* drawing up of the testicle on stimulating the skin of the inner and upper part of the thigh. *Inguinal reflex :* a similar stimulus causes contraction of some of the fibres of the internal oblique muscle near Poupart's ligament. *Abdominal reflexes* are contractions of portions of the oblique and recti muscles on stroking the side of the abdomen and lower part of the thorax (epigastric, umbilical, and hypogastric reflexes). *Gluteal reflex :* contraction of the glutei following irritation of the skin over the buttocks.

Corneal reflex : closure of eyelids on gently touching the cornea. *Palatal reflex :* the soft palate rises on being touched. *Pharyngeal reflex :* retching caused by tickling the fauces.

(3) *Visceral or complex reflexes,* by which the efficiency of certain organs is assisted, may be disturbed in disease, and in some instances their disorders may be of diagnostic importance.

Pupil Reflex.—Observe the behaviour of each pupil separately when exposed to varying amounts of light. On light being suddenly admitted to a normal eye, the

pupil contracts forcibly, then slightly relaxes and again contracts ; this oscillation of the pupil, termed *hippus*, is slight in health, but in conditions of exaggerated reflexes it may be quite obvious. A reflex dilatation of the pupil to painful stimuli is also obtainable by pinching or pricking the skin of the neck, and at times by painful impressions from any part of the body. A contraction of the pupil also occurs on focussing the eyes to close vision, this being due to muscular effort of the muscles concerned in accommodation. Absence of light reflex with persistence of accommodation contraction (the *Argyll-Robertson pupil*) is found in locomotor ataxia and general paralysis of the insane.

The light reflex is absent in conditions of deep unconsciousness, in poisoning by belladonna, hyoscyamine, cocaine, etc. ; in paralysis of the third nerve ; in intracranial lesions interrupting the pupil reflex arc ; and in loss of perception of light.

Bladder and *rectum reflexes* are at times disturbed by disease. The functions of these organs are largely carried out by a series of reflexes ; the retention by a sphincter and the expulsion by detrusor muscle fibres of their contents are mainly involuntary and reflex acts. The reflex arcs have their centres in the lumbar portion of the cord ; damage to this region, or the portions of the cord higher up, may result in incontinence or obstruction of urine or fæces.

Vasomotor Reflex.—The proper supply of blood to the different organs and regions of the body, in accordance with their needs, is maintained by a reflex mechanism, acting chiefly on the muscular coats of the small arteries. The diagnostic interest of disturbances of this reflex are not great. The appearance of bright red marks, and even raised œdematous patches, where the

skin was irritated by rubbing gently (*tache cérébrale* or *spinale*, also known as *dermographism*) is sometimes found in meningitis and diseases of the brain and cord. Erection of the penis (*priapism*), a result of vasomotor disturbance, occurs in lesions of the cord and medulla. Other examples of visceral reflexes, such as coughing, vomiting, hiccough, do not require further mention here.

3. **Sensory Functions.**—Afferent nerve impulses from sensitive organs in the periphery give rise to (1) tactile and pressure sensibility ; (2) pain ; (3) thermal sensibility ; (4) sense of strength or innervation ; (5) sense of movement or position ; (6) sense of shape or form ; (7) special senses of sight, hearing, taste, and smell.

(1) *Tactile sensibility* is tested by directing the patient to say "now" the moment he feels a light touch, his eyes being closed. A piece of cotton-wool or a camel's-hair brush should be used, so that pressure is avoided. In order to test his power of locating the stimulus, he may be told to point to the spot touched. His ability to distinguish when he is touched in two places close together may be used as a measure of the acuteness of his common sensation. For this purpose the points of a pair of compasses or scissors may be applied gently to the skin, and the shortest distance between the points at which he can recognize two points of contact should be noted. An instrument (æsthesiometer) is made for this purpose. *Pressure sensibility* is a form of tactile sensibility, and is tested by placing objects of the same size but of different weight on the surface, the limb being supported so as to obviate muscular effort ; the patient is to state which object is the heaviest.

Any obstruction in the path of afferent nerve im-

pulses will result in more or less complete loss of common sensation (*anæsthesia*). This symptom is often a help in locating the situation of such an obstructing lesion. In neurotic states anæsthesia is common, but without anatomical consistency.

An undue sensitiveness of the organs concerned in the perception of tactile stimuli may give rise to excessive acuteness of the sense of touch (*hyperæsthesia*) ; abnormal sensations (*paræsthesia*) may be perceived, such as tingling, tickling, cotton-wool feeling, crawling of insects (*formication*); a single stimulus may give rise to several tactile impressions (*polyæsthesia*); the patient may be unable to localize pain or touch sense (*allocheiria*).

(2) *Pain.*—The inability to recognize painful stimulation may be diminished or lost (*analgesia*), or may be intensified (*hyperalgesia*). The former is tested by pricking or pinching the skin, noting if the patient shows any sign of suffering ; the latter by rubbing a blunt instrument (*e.g.*, the head of a pin) firmly in parallel lines over the surface, or by pressure of the hand. Should hyperalgesia be present, the patient will be able to indicate the position where the friction or pressure causes pain instead of mere tactile sensibility.

Loss of the sense of pain usually accompanies loss of tactile sensibility, but in cases of syringomyelia the painful impulses (as well as those of heat and cold) are obstructed by the disease of the central canal, while those of common sensation passing upward through the posterior columns are less interrupted. The loss of painful and thermal sensibility, with the retention of touch sense, is termed *dissociated anæsthesia.*

Increased sensibility to pain may be found in lesions of the spinal cord and its meninges, whereby the posterior nerve roots are irritated. As a result there may

be a zone of hyperalgesia, or hyperæsthesia, at the level of the injured segment of the cord (*girdle pain*). Other examples of hyperalgesia may be observed, by similar means to that mentioned above, in certain cases of internal disorders, in which, as mentioned in Chapter I., the visceral pain is referred to a spot on the surface, owing to the proximity in the cord of the afferent nerve paths from the respective regions.

(3) Sensibility to heat and cold may be tested by touching the patient's skin with test-tubes, one filled with hot and the other with cold water. In syringomyelia dissociated anæsthesia frequently occurs; the heat sense may be absent in multiple sclerosis, in locomotor ataxia, and in hysteria.

(4) *Sense of strength* or of *innervation* is tested by adding weights to or taking them from a sling suspended by a broad band from the hand; the patient is to say when he notices differences in the weight. The muscular power employed can, with practice, be accurately judged by healthy persons, but in conditions of inco-ordination the judgment is defective.

(5) *Muscle sense or sense of movement and position* is a combination of the innervation sense with tactile sensibility (skin, joint surfaces, etc.). Direct the patient to perform a number of movements of the limbs with the eyes closed; he is then to describe the position of his limbs without having looked at them.

This sense, like the strength sense, is disturbed in ataxic conditions.

(6) *Stereognosis*, or judgment of shape, form, and character of an object, is produced by a combination of the perceptions of touch, temperature, and muscular action.

(7) *The organs of special sense* are to be examined

with regard to the state of the nervous system, and not to local disease in the organ, for which the reader must consult special textbooks.

Vision.—The patient's distinctness of visual perception is first to be investigated, both for distant and for near objects. The conditions which give rise to dimness of vision (apart from local eye affections) are mainly optic neuritis and optic atrophy; the latter always, and the former sometimes, causes defective sight.

The ophthalmoscopic examination is next to be made. The following abnormalities having reference mainly to nervous diseases may be observed:

(1) *Optic Neuritis.*—The disc is swollen and red, its margins indistinct, the central vein larger than normal, while the central artery is of normal size or contracted. In extreme cases *papillitis* or *choked disc* occurs—that is, the papilla projects as a dome-shaped elevation, and white striæ or spots may surround it, while flame-shaped patches are seen near and on the papilla. The cause of optic neuritis is most frequently tumour or abscess of the brain, also meningitis and hydrocephalus. Occasionally it occurs in spinal disease—viz., tabes dorsalis and myelitis; also rarely in peripheral neuritis. It may be due to tumour or inflammation of the orbit. In certain toxic states of the blood, in which the central nervous system is not primarily at fault, optic neuritis also occurs—*e.g.*, chlorosis, rheumatism, lead-poisoning, Bright's disease, syphilis.

(2) *Optic Atrophy.*—The disc is pale, hollowed on the surface (*cupped*), the vessels shrunken, and often outlined by two white lines representing their thickened coats. It may be a primary disease of the nerve, or may be secondary to lesion in the nerve or brain. Sight is affected in proportion to the extent of the atrophy.

(i.) Primary optic atrophy occurs in locomotor ataxia most commonly, less frequently in multiple sclerosis and general paralysis of the insane. It is also believed to be due to exposure to cold, sexual excess, diabetes, lead-poisoning, and alcoholism.

(ii.) Secondary optic atrophy may result from optic neuritis ; from embolism of the central artery of the retina and from retinitis ; from intracranial pressure of tumours, inflammation and hæmorrhage.

(3) *Retinitis.*—Cloudiness of the fundus with enlarged veins ; hæmorrhagic patches, and white exudations and degenerations. If congestion of the papilla accompanies the retinitis, the condition is *neuro-retinitis.* It is found in the following conditions: Nephritis, most frequently in the contracted form, and in an advanced stage ; cardiac and vascular diseases—*e.g.*, valvular disease, arterio-sclerosis, aneurism ; diabetes, severe anæmias, lead-poisoning, infectious diseases. In pyæmia or septicæmia a purulent retinitis may occur, in which case the inflammation may soon involve the whole eye (panophthalmitis). In children with defective development from hereditary causes (*e.g.*, syphilis or consanguinity of parents) pigmented spots distributed towards the periphery of the fundus and encroaching upon the central regions may be observed.

(4) *Tumours* of the eye are only of diagnostic interest when they indicate the nature of similar growths in other parts of the body. The most important is tubercle, occurring as one or more round yellowish spots in the choroid, usually near the disc, and usually part of a general miliary tuberculosis.

Various minor disturbances of vision occur ; floating spots and beaded threads (*muscæ volitantes*) are observed by persons suffering from digestive disturbance, hysteria,

cardiac hypertrophy, etc. *Yellow discoloration* of all objects may be noticed by jaundiced patients and by those taking santonin. *Flashes of light* occur in indigestion, in migraine, and they may form the aura of epilepsy. Dark patches with bright margins (*glittering scotomata*) occur in migraine ; also in irritative lesions of the cortex.

The Field of Vision is the area within which, the eye under examination being fixed upon any point and the other eye being closed, white objects can be distinguished. On the temporal side of the eye it reaches about 90 degrees, and on the nasal side 50 degrees. At one spot, the " blind spot," 15 degrees to the outside of and a little below the point upon which the gaze is fixed (the *point of fixation*), objects are invisible. From this spot light passing through the pupil falls upon the entering optic fibres. 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 order to investigate the field of vision an instrument, the *perimeter*, is used, by means of which the area visible to the patient may be recorded on a chart.

The patient may be unaware of defects of the field of vision ; this is the condition known as *vision nulle*. *Vision obscure* is a darkened or indistinct vision, which causes the patient inconvenience. The former is usually caused by a lesion of the vision centre in the cortex, while the latter results from a lesion lower in the visual path. A defective area in the field of vision is termed a *scotoma*, which may be *peripheral*, *central*, *temporal*, or *nasal*, according to its position. The following defects in the field of vision may be found :

(1) *Hemianopsia*.—One-half of the field is blind. This

may occur in one or both eyes ; most commonly both eyes are affected. When the blind halves of the field are both on the same side of the median line, the condition is termed *homonymous hemianopsia.* A lesion of the optic path above the chiasma will cause this defect. Loss of sight in both the outer halves of the field of vision (*bitemporal hemianopsia*) is rarer, and is due to a lesion of the chiasma. Other forms of hemianopsia (*unilateral, bilateral, nasal, superior,* and *inferior*) are very rare, as they depend on damage to a portion only of the fibres of the chiasma.

The lesions causing hemianopsia are usually organic brain affections, fractures of the base of the skull, or tumour of the pituitary body. Functional disorders of the nervous system, however, are an occasional cause—*e.g.*, hysteria, migraine, fatigue, epilepsy, digestive disturbances.

(2) *Total blindness* of one eye alone is due to a lesion of the optic nerve (excluding disease of the eye).

(3) *Contracted field of vision,* the peripheral area being defective, may result from optic atrophy, glaucoma, and functional disturbances.

(4) *Central scotoma,* or *toxic central amblyopia,* is a defect of vision in the central part of the field of vision, especially for colours. It is caused by excess of tobacco and alcohol ; also by diabetes, uræmia, quinine, iodoform, etc.

Hearing.—Defects of this sense have but little diagnostic interest, and generally indicate disease of the organ of hearing. Psychical deafness—that is, inability to recall the meaning of well-known sounds—is referred to below.

Imaginary sounds are heard in some mental diseases, and at times in irritative lesions of the cortex of the

brain ; they may form the aura in cases of Jacksonian epilepsy.

Smell.—Loss of this sense (*anosmia*), or perversions of it, generally result from local affections of the nose. Occasionally it may be due to lesion of the olfactory bulb or of the intracranial connections of the nerve, to lesion of the fifth nerve, to degenerative affections of the central nervous system (*e.g.*, locomotor ataxia), or it may be a symptom of hysteria.

Excessive acuteness of the sense of smell (*hyperosmia*) is sometimes a symptom of hysteria.

Perverted sense of smell (*parosmia*) sometimes occurs in mental affections, and it is not unusual for the aura of epilepsy to assume this form.

To test the sense, direct the patient to smell savoury but non-irritating substances—*e.g.*, oil of peppermint, assafœtida, but not "head-salts" or other form of ammonia—and see if he can identify them.

Taste.—Deficiency in acuteness (*ageusia*) and perversions (*parageusia*) of the sense of taste are observed as a result of affections of the tongue or mouth, and of conditions which impair the sense of smell. They are less frequently due to disease of the trigeminal or glossopharyngeal nerves or their central connections. Facial paralysis is often accompanied by partial and one-sided ageusia, owing to the implication of the chorda tympani.

Parageusia sometimes occurs as an epileptic aura, and is not infrequent in cases of mental disease and in hysteria.

In order to test the sense of taste, particles of sugar, common salt, or magnesium sulphate, are placed on the dorsum of the tongue, first on the anterior portion, then on the posterior ; the tongue remaining protruded, the patient is to signify (in writing if possible) what is the substance employed.

4. **Psychical Functions.**—The state of the patient's *intellect* is to be investigated, so far as it concerns the diagnosis of medical diseases. Is his *memory* for recent and for remote events up to the average ? Does he give *attention* to what is going on, or does his attention wander off too readily ? Is his *understanding* or intelligence fair ? Has he any delusions or hallucinations ?

Should he be *unconscious*, note if there are any accompanying symptoms which may assist in the diagnosis (see Appendix X.).

Speech.—If he is sufficiently conscious, his command of *language* is to be carefully investigated.

Ascertain if he can hear and see sufficiently well to perceive the sound and appearance of spoken and written words respectively. When he speaks, observe if there is any defect in his voice (see Chapter IV.), in the distinctness of his articulation, or in the facility with which he can express his thoughts in words. Thus, he may speak in a clumsy, awkward manner, with imperfect pronunciation, especially of the dentals and labials (*slurring speech*) ; this is a characteristic of facial paralysis, bulbar paralysis, and, when combined with tremor of the lips and tongue, of general paralysis of the insane and of alcoholism. A slow, measured speech (*scanning speech*) is a symptom of multiple sclerosis and of the rarer condition, Friedreich's ataxia. *Stammering* is a defect of co-ordination of the muscular acts involved in speech, and a somewhat similar speech defect is observed in chorea and hysteria. Lisping and other similar defects may be due to habit or to disease of the teeth or mouth.

The imperfections of speech mentioned above are examples of inefficiency of the organs of speech rather than of the mental faculties, and are spoken of generally as *anarthria*.

Aphasia.—Intelligent speech involves the active use of the mind in recalling memories of sounds (or written signs) which have been stored up in the brain in the process of learning to understand language, to speak, and to write. Disorders of speech which are due to disturbance of the mental functions and organs are termed *aphasia*. The storing-up of impressions of sound and vision is not a haphazard process, but proceeds on definite lines. Impressions of sounds and spoken words which can be recalled as memories are stored in a "centre," situated in the first temporo-sphenoidal convolution, of the left side in right-handed persons, and of the right side in the smaller number of persons who are left-handed. A "centre" for visual memories is similarly situated in the left or right angular gyrus. In the occipital lobe on both sides are the primary centres for vision. These centres are connected by means of association fibres, and are similarly in communication with a co-ordinating centre which controls and regulates the impulses from the above centres, resulting in spoken or written language. The last-named centre is situated in the third frontal convolution (*Broca's convolution*), and possibly in the second frontal as regards writing. In right-handed persons Broca's centre is in the left frontal lobe, while in left-handed persons it is in the right. From Broca's centre association fibres convey impulses to both sides of the brain, stimulating the motor centres concerned in the complex muscular actions of speech.

In order to discover the situation of a lesion giving rise to a speech defect, a full and searching examination of the powers of speech of the patient is to be undertaken. The following outline of the method of investigation may be adopted and amplified:

(1) Ask the patient to pick up a pencil, strike a match,

put out his tongue, etc. If he hears, but fails to comprehend the words, he has *word deafness* or *auditory aphasia*. (2) Give him similar directions in writing; if he cannot understand, it is *word blindness, alexia,* or *visual aphasia*. These two groups of cases indicate a lesion of some portion of the receptive apparatus—*i.e.*, the cortical centres for storing auditory or visual memories, or the fibres connecting these centres with Broca's convolution. They are therefore known as "sensory aphasia." (3) He cannot speak voluntarily and correctly: *motor aphasia* or *aphemia*. (4) He cannot write: *agraphia*. Classes (3) and (4) result from lesion of the centre for the production of the motor impulses which produce speech (Broca's centre), or of the association fibres connected with it; it is therefore *motor aphasia* and *motor agraphia*. Should he be able to write to dictation, but not voluntarily, it is *sensory agraphia*. (5) He can speak and write voluntarily, but makes mistakes in the use of words, calling objects or actions by wrong names: *paraphasia, paragraphia*. (6) He cannot recognize familiar objects (*mind blindness*); not only is he unable to give the name of such articles as watch, ring, scissors, match, but he cannot indicate what they are used for. (7) A similar inability to recognize the meaning of familiar sounds is *mind deafness*. (8) Inability to recognize an object by its general characters—*e.g.*, its hardness, softness, shape, roughness, smoothness—is termed *apraxia*. The groups of defects (5) to (8) are due to lesions affecting not only the centres, but also or principally the association fibres between the different regions of the brain. It is rare to get a case of simple sensory or motor aphasia, as in most cases the lesion causes a sufficient mixing of symptoms to render the diagnosis difficult.

APPENDICES

APPENDIX I

PAINFUL THROAT AFFECTIONS

Symptoms.	Acute Pharyngitis.	Chronic Pharyngitis.	Secondary Syphilis.	Follicular Tonsillitis.	Phlegmonous Tonsillitis.	Diphtheria.	Scarlet Fever.
Pain ..	Smarting or burning	Slight	Very slight	Considerable	Severe	Moderate	Considerable
Fever ..	Moderate	Little or none	Slight	Considerable	High fever	Moderate	Considerable
Seat of the lesion and its nature	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, 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
Other prominent symptoms	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

APPENDIX II

MICRO-ORGANISMS IN THE URINE

A CATHETER specimen of the urine is to be obtained with complete aseptic precautions. Centrifugalize, or allow it to sediment. Ammoniacal urine may be heated on a water-bath with dilute potassium hydrate solution before centrifugalizing; urates are to be dissolved by warming. Smear a cover-glass with the sediment, and allow it to dry in the air at ordinary temperature. Fix by passing it three times rapidly through a Bunsen flame.

1. *Methylene Blue.*—Add 3 or 4 drops of a 5 per cent. solution of methylene blue in alcohol to a watchglass of water. Immerse the cover-glass in this dilute solution for two or three minutes, wash in water, dry with filter-paper, and mount in Canada balsam. A useful stain for all urinary bacteria, and chiefly for the gonococcus.

2. *Gram's Method.*—Add a few drops of aniline oil to about half a test-tubeful of water, shake thoroughly for about a minute, then filter the emulsion through a filter-paper previously wet with water; to a watchglassful of this aniline water add 3 or 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, transfer it for two minutes to Gram's iodine solution (iodine, 1 gramme; potassium iodide, 2 grammes; water, 300 c.c.). Wash in 95 per cent. alcohol till no more colour comes away; wash in water. Counter-stain for about half a minute with basic fuchsin (3 or 4 drops of a 10 per cent. alcoholic solution of basic fuchsin in a watchglassful of water). Wash in water, dry with filter-paper, and mount in Canada balsam. By this method the streptococcus, staphylococcus, and tubercle bacillus (Gram-positive), are stained dark blue or black (the smegma bacillus is not stained), while the gonococcus, *Bacillus typhosus*, and *B. coli communis* (Gram-negative) are stained red.

3. *Ziehl-Neelsen Method.*—Pour a few drops of carbol fuchsin solution (fuchsin, 1 gramme; alcohol, 10 grammes; 5 per cent. aqueous solution of carbolic acid to 100 c.c.) on the cover-slip smear. Hold it by a forceps over a Bunsen flame, and keep it heated for two minutes. Wash in water; pour on it a few drops of a 20 per cent. solution of nitric acid, remaining for three to five seconds; wash in water. Drop on 60 per cent. alcohol till the red colour disappears; wash in water. Counter-stain with dilute methylene blue as in (1); wash, dry, and mount in Canada balsam. The tubercle bacillus is best stained by this method.

APPENDIX III

WIDAL'S REACTION

A FEW drops of blood from a prick of the patient's finger are received in a small glass capsule. The serum thus obtained is diluted by means of a diluting pipette with 0·8 per cent. sodium chloride solution. A series of dilutions are made on glass slides—viz., 1 in 5, 1 in 10, 1 in 25, and 1 in 50. Take an equal quantity of each of these dilutions and of a freshly prepared culture of typhoid bacilli, and make a series of hanging-drop preparations of the dilution of 1 in 10, 1 in 20, 1 in 50, and 1 in 100. If the serum is from a typhoid patient, the bacilli will be motionless and clumped in less than two hours, and the lower dilutions sooner. The reaction may be reported positive if there is distinct agglutination in a 1 in 50 dilution in an hour.

This reaction is clinically almost restricted to typhoid fever, but it can also be applied to cholera and Malta fever, and to some other bacterial processes.

APPENDIX IV

WASSERMANN'S REACTION

By bringing an *antigen* (foreign organic extract) into contact with the antibody present in the serum of a syphilitic patient, complement is fixed. This is demonstrated by the absence of hæmolysis on introducing sensitized corpuscles to the serum which has been so treated.

Method.—The organic extract, as recommended by Fleming, is prepared by bruising in a mortar 1 gramme of fresh heart muscle in 5 c.c. absolute alcohol; heat to 60° C. for an hour, and incubate for twenty-four hours; the clear fluid, diluted with saline solution, is the extract. Into a small glass tube (1 inch long by ¼ inch diameter) 4 parts of extract (measured by means of a Wright's pipette) are placed, and into another similar tube are placed 4 parts of saline solution; 1 part of the serum under examination is now added to each tube, and they are placed in the incubator for half an hour. A similar control experiment is made at the same time with non-syphilitic serum. Add 1 part of a 10 per cent. suspension of sheep's corpuscles in saline to each tube, and incubate for an hour and a half. The control tubes should both show hæmolysis. If both saline and extract tubes of the suspected case show hæmolysis, the reaction is negative; if the extract tube shows no hæmolysis, while the saline tube is hæmolyzed, the reaction is positive; if no hæmolysis occurs in either tube, the reaction is indeterminate.

A positive reaction is hardly ever found in health, and rarely in any affection except syphilis, in which disease it occurs in over 90 per cent. of the cases. In the tertiary and parasyphilitic affections it also occurs in the majority of cases.

APPENDIX V

FREE HYDROCHLORIC ACID—QUANTITATIVE ESTIMATION

It is chiefly in the examination of the stomach contents that this test is employed, and is then regarded as a measure of the quantity of the gastric juice present.

To 10 c.c. of the filtered stomach contents add a few drops of a 0·5 per cent. alcoholic solution of dimethylamidoazobenzol ; free hydrochloric acid causes the solution to turn red. From a burette run in gradually decinormal soda solution (4 grammes NaHO to 1 litre water) till the fluid is neutralized, as shown by the disappearance of the red colour and its replacement by a greenish yellow. Note the quantity of soda solution expended : 1 c.c. of decinormal soda solution neutralizes 0·00365 gramme of free hydrochloric acid.

APPENDIX VI

TOTAL ACIDITY OF STOMACH CONTENTS

The acidity of the stomach contents is due to the presence of free hydrochloric acid, salts of the mineral acids (chiefly acid phosphate), and organic acids. The estimation of the acidity of other fluids—*e.g.*, the urine—may also be made by the following test :

To 10 c.c. of unfiltered stomach contents add, with thorough shaking, about 100 c.c. water and a few drops of a 1 per cent. alcoholic solution of phenolphthaleïn. This causes the solution to be pink if alkaline, and colourless if acid. From a burette run in decinormal soda solution (see Appendix V.) ; as soon as the solution begins to turn pink, note the amount of soda solution expended. The measure of acidity may be taken to be the quantity of soda solution required to neutralize 10 c.c. of stomach contents. During normal digestion the acidity is between 40 and 60.

APPENDIX VII

DIAZO REACTION

Two solutions are to be prepared: (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. A couple of drachms of (2) with a few drops of (1) are shaken up in a test-tube; add an equal quantity of urine, and ammonia to alkalinize. A positive reaction shows a port-wine colour with red froth.

This reaction is almost always found in typhoid fever in the second and third week; it is, however, given in cases of active tuberculosis, and often in pneumonia, measles, scarlet, and other fevers.

APPENDIX VIII

RUSSO'S METHYLENE-BLUE REACTION

To 4 or 5 c.c. of filtered urine add 4 drops of a 1 in 1,000 watery solution of methylene blue. Shake the tube, and if the patient is suffering from typhoid fever an emerald-green colour appears; in negative cases there is scarcely any colour.

APPENDIX IX

ELECTRICAL EXAMINATION

It is almost exclusively in disease of the lower segment of the motor tract that examination by means of electricity is of use; the interrupted and the continuous currents are both to be used. (The examination by X rays is not here considered.)

The region to be examined is first submitted to the *interrupted* or *faradic* current, which stimulates the motor nerves. A large terminal is placed on an indifferent region—*e.g.*, between the shoulder-blades—and

a smaller one is used to demonstrate the contracting power of the various muscles, by placing it near the spot where the nerve enters the muscle in question (*motor point*), and also over the trunk of the nerve. Observe the minimum power of current which produces a muscular contraction, and note if there is any difference in contractility between the sides of the body.

The *continuous* or *galvanic* current is next to be employed. The management of the terminals is similar to that of the faradic current, but the muscle as well as the nerve responds to this stimulus. The negative electrode or *cathode* is first to be used, noting by means of the galvanometer, in milliampères, the smallest current which produces a current on closing and on opening the circuit. Also, the same observation is to be made with the positive electrode or *anode* in use. Here also the effects of the current on both sides of the body are to be compared.

Normally the muscle contracts most readily with cathodal closure. The anodal opening and closing contractions require a stronger current, and the cathodal opening contraction is the most difficult to obtain. This may be expressed by the formula :

$$\text{C.C.C.} > \text{A.C.C.} > \text{A.O.C.} > \text{C.O.C.}$$

In disease of the nervous system affecting the nutrition of the nerves and muscles, one finds alterations in the character of the contraction and in the order in which currents of different characters and strength give rise to muscular contraction (*qualitative changes*). Also one finds changes in the degree of activity of the muscular contractions (*quantitative changes*). A characteristic series of alterations of the electrical reactions is known as the *reaction of degeneration*. When complete, this change consists of loss of response to the faradic current, with increased response of the *muscle* to galvanic stimulation. The contraction, however, while more readily produced by the continuous current, is sluggish in its onset, and usually responds more easily to the anodal than to the cathodal closing current.

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

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	Laboured 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 overripe 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. *Diplococcus intracellularis* in fluid (obtained by lumbar puncture) in cases of epidemic cerebrospinal meningitis	Most severe in youthful patients. Headache, vomiting; drowsiness often precedes coma. Sugar in the urine	Almost exclusively in females. 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

APPENDIX XI

PARALYSIS ACCOMPANIED BY (A) SPASM AND (B) ATROPHY OF THE MUSCLES

A. *Spastic Paralysis* (supranuclear or upper neuron lesion).

I. Disease of the spinal cord :

1. Transverse interruption of the cord :

(1) Compression of the cord :

(*a*) Caries of the vertebræ (Pott's disease).

(*b*) Tumours of the meninges and of the vertebræ (carcinoma, sarcoma, syphilis).

(*c*) Pachymeningitis (especially hypertrophic form).

(*d*) Aneurism of the aorta or of its branches.

(*e*) Traumatism of vertebral column, and intrameningeal hæmorrhage.

(2) Myelitis, acute and chronic, transverse or diffuse.

(3) Hæmorrhage into the cord.

2. Sclerotic changes in the upper segment of the motor tract :

(1) Primary lateral sclerosis.

(2) Hereditary spastic paraplegia.

(3) Lateral sclerosis complicated with other lesions of the cord :

(*a*) Amyotrophic lateral sclerosis (Charcot's disease).

(*b*) Ataxic paraplegia.

(*c*) Primary combined sclerosis (Putnam and Dana).

(*d*) Multiple (disseminate, insular) sclerosis.

(*e*) Syringomyelia.

(*f*) Pellagra.

II. Disease of the brain :

1. Lesions of the circulation :

(1) Hæmorrhage.
(2) Embolism.
(3) Thrombosis.

2. Tumours.
3. Degenerative and inflammatory changes :

(1) Multiple sclerosis.
(2) General paralysis of the insane.
(3) Cerebellar heredo-ataxia.

III. Functional disturbance (hysteria, neurasthenia, " railway spine ").

B. *Flaccid Paralysis* (lower neuron lesion).

I. Disease of the brain and spinal cord:

1. Chronic degenerations :

(1) Progressive spinal muscular atrophy.
(2) Bulbar paralysis.
(3) Ophthalmoplegia.
(4) Friedreich's ataxia.

2. Acute inflammations :

(1) Infantile paralysis (acute anterior polio-encephalo-myelitis).
(2) Landry's paralysis.

3. Traumatism : injury to cervical or lumbar enlargement.

II. Disease of the peripheral nerves :

1. Lesions of the cranial motor nerves.
2. Lesions of the spinal motor nerves.

APPENDIX XII

TESTS FOR TUBERCULOSIS

1. *Calmette's Test.*—One drop of a 0·5 per cent. solution of tuberculin is placed at the inner side of the conjunctival sac. A positive reaction, occurring within six hours, consists in swelling and redness of the lachrymal tubercle, and congestion and œdema of the conjunctiva.

The test is positive in most cases of active or latent tuberculosis, and in a number of apparently healthy cases. In chronic cachectic tuberculous patients and in virulent and extensive lesions the reaction is usually negative.

2. *Von Pirquet's Test.*—The skin is scarified over a small area, and an ointment of 25 per cent. Koch's old tuberculin is rubbed in. A positive reaction is the appearance in a few hours of a deep red papule, which persists for some days.

It is positive in very similar conditions to those in which Calmette's test is positive.

3. *Moro's Test.*—A morsel about the size of a pea of a 50 per cent. ointment of Koch's old tuberculin is rubbed on the skin. After twelve or perhaps twenty-four hours a papular or pustular eruption appears, when the reaction is positive.

The reaction occurs under similar circumstances to those just mentioned.

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THE END

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